Health Care for Older People

Holistic Approach

Musculoskeletal Problems in Older Adults

Sri Lankan Association of Geriatric Medicine

2021

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The book is intended to strengthen the knowledge of the members of the multi-disciplinary team and care givers. It is may not be used for the diagnosis of the disease and treatment purposes.

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An attempt to develop and promote multidisciplinary mutual coordination and collaboration among the teams involved in care of older patients at various levels in the health and social services sector.

'Team work divides the task and multiplies the success'

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Contents

1. Editorial

Sri Lankan Association of Geriatric Medicine launches yet another bulletin in the series under the theme "Health Care for Older People- Holistic Approach. This 5th addition to the series is on Musculoskeletal Problems in Older Adults. As in the past, a view of educating different levels of health care professionals caring for older people in the country is anticipated.

Musculoskeletal complaints take considerable amount of presentation of older adults to primary health care facilities and more advances facilities as well. Furthermore, a vast number of pathologies including degenerative disease, trauma and inflammation are causative aetiologies for varied presentation. Differentiation of these conditions and arriving at a differential diagnosis needs to be done with good clinical reasoning and conscientious thought.

We are most thankful to all the authors for their kind contribution to make this bulletin a success. The time and dedication provided by the authors who are busy professionals in their own fields in indeed commendable and highly appreciated. We thank Prof. Sarath Lekamwasam (President SLAGM) for the support and advice given.

We hope that this bulletin will provide continued education to health care professionals caring for older people in the country.

Dr. Shehan Silva & Dr. Achala Balasuriya
Editors
March 2021

2. Physiology of Skeletal Muscle and Joints

Prof. Dinithi Fernando

INTRODUCTION

Bones, skeletal muscles and joints create a system of articulating levers that are used to manipulate objects and move the body from one place to another. A pair of antagonistic muscles allow motions of the limb across the joint. In the case of the elbow, flexion is by biceps, and extension is by triceps (Figure 2.1). However, a full range of motions in the arm uses more than forty muscles. Skeletal muscles are used, not only for locomotion, but also for maintaining erect posture, and expansion and contraction of the lungs. This chapter provides a brief overview of the physiology of skeletal muscle and the joints.

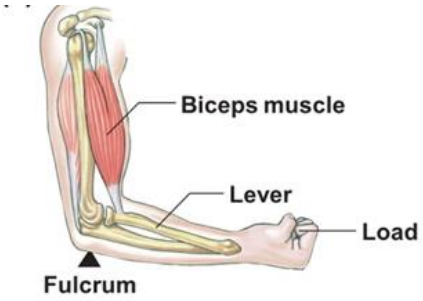

Figure 2.1 - A joint as a lever

FUNCTIONAL ANATOMY OF SKELETAL MUSCLE

Most skeletal muscles begin and end in tendons, and the muscle fibres are arranged in parallel between the tendinous ends. A muscle consists of many fasciculi. Several thousands of muscle fibres (muscle cells) make a single muscle fasciculus. A single muscle fibre, enclosed by a sarcolemma has many myofibrils. Myofibrils are composed of thin actin and thick myosin filaments. Sliding of these filaments are responsible for muscle contraction. Partial overlapping of actin and myosin filaments causes the myofibrils to have alternate light and dark bands,

giving rise to its striated appearance. The contractile unit of the muscle fibre is the sarcomere, which is the region between two z discs. Two adjacent z discs come close together during contraction of a muscle fibre. (Figure 2.2)

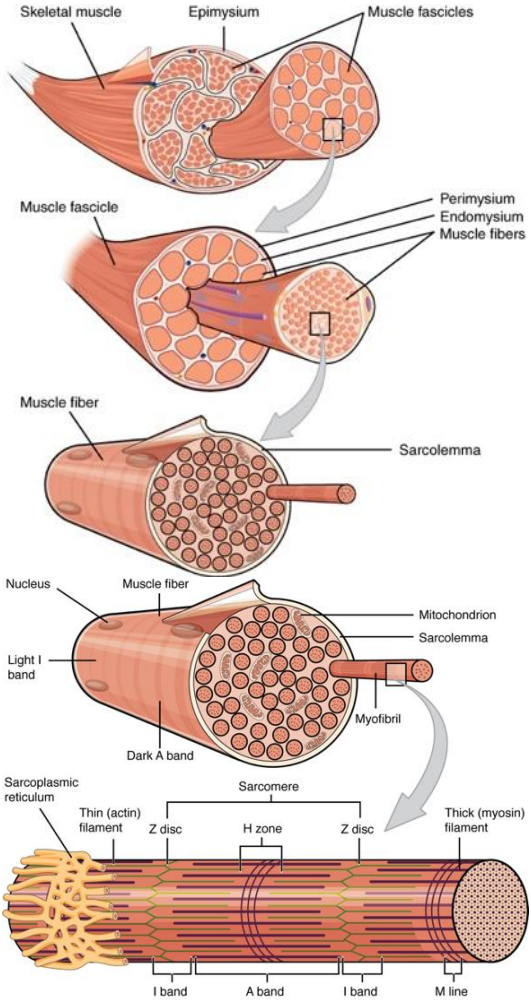

Figure 2.2 - Functional anatomy of muscles

THE SARCOTUBULAR SYSTEM AND THE CONTRACTILE MECHANISM OF SKELETAL MUSCLE

The myofibrils in a muscle fibre are surrounded by vesicles and tubules that form the sarcotubular system, which is made up of a T system and a sarcoplasmic reticulum (SR). The T system that is continuous with the sarcolemma allows rapid transmission of the action potential from the sarcolemma to the vicinity of the myofibrils. The voltage-gated dihydropyridine receptor (DHPR) in the T tubule triggers Ca2+ release from the SR via the ryanodine receptor (RyR). When the voltage change is sensed, there is an interaction between the DHPR and RyR, allowing Ca2+ release from the SR (Figure 2.3).

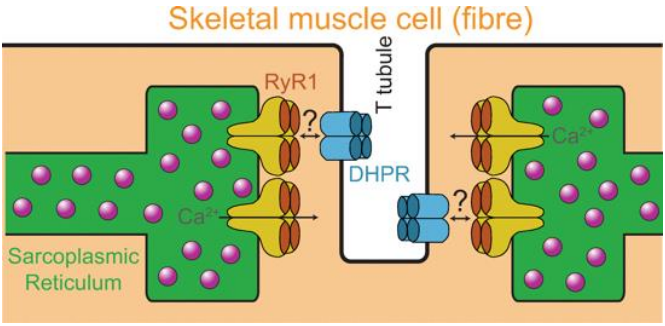

Figure 2.3 - Sarcotubular system of skeletal muscles

The contractile mechanism is initiated by a rise in the free calcium concentration in the sarcoplasm. As explained earlier, calcium is released from the sarcoplasmic reticulum when the action potential travels down the T tubules. Binding of Ca2+ to troponin C uncovers the myosin-binding sites on actin. Cross-linkages are formed between actin and myosin which allows the 'power stroke' or the 'walk along mechanism' producing sliding of thin filaments on thick filaments, leading to muscle contraction. At the end of a contraction, Ca2+ is pumped back actively into the sarcoplasmic reticulum. Ca2+ that is bound to troponin is released and the interaction between actin and myosin ceases (Figure 2.4).

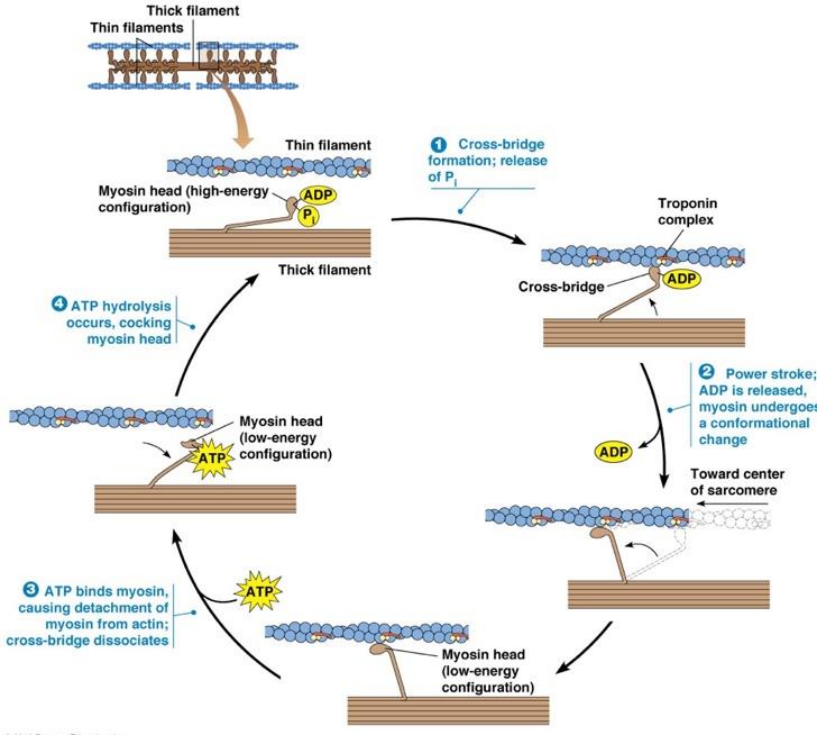

Figure 2.4 - Contractile mechanism of skeletal muscle

Energy for muscle contraction and muscle fatigue

Most of the energy required for muscle contraction is used for 'walk-along mechanism' by which the cross- bridges pull actin. Small amounts of energy are needed for pumping calcium ions back to the sarcoplasmic reticulum once contraction is over, and for Na+- K+ ATPase activity on sarcolemma. The ATP in the muscle fibre is split to ADP, transferring energy for the contractile machinery. ADP is re-phosphorylated immediately, maintaining a steady source it. There are three sources of energy used for re-phosphorylation. The first source phosphoryl creatinine contains high energy phosphate bonds is cleaved releasing energy for reconstitution of ATP. The second source is glycogen, which undergoes glycolysis, releasing energy. Glycolysis can

occur even in the absence of oxygen, allowing muscle contraction for several seconds, during anaerobic conditions. The third source is oxidative metabolism, where oxygen reacts with glucose and free fatty acids. More than 95% of energy for sustained and long-term muscle contraction comes from oxidative metabolism, the greater proportion of energy coming from fat metabolism.

Prolonged, strong contractions lead to muscle fatigue. It has been shown in athletes that muscle fatigue is directly proportionate to the rate of glycogen deplete. Lack of energy interrupts the contractile and metabolic processes of the muscles, diminishing the output of muscle work. In addition to energy depletion resulting from increased muscle work, lack of blood flow and interference to nerve signal transmission through the neuromuscular junction also cause muscle fatigue.

TYPES OF MUSCLE CONTRACTION

Functionally, there are two types of muscle contraction, isotonic and isometric. Isotonic contraction is further divided into concentric and eccentric types. In isometric contraction, the muscle fibres do not shorten and the afterload exceeds the total contractile force. During concentric isotonic contraction, the muscle fibres shorten and the afterload is overcome by the contractile force, whereas in eccentric isotonic contraction the muscle fibres lengthen. In practice, most contractions are a mix of all types (Figure 2.5).

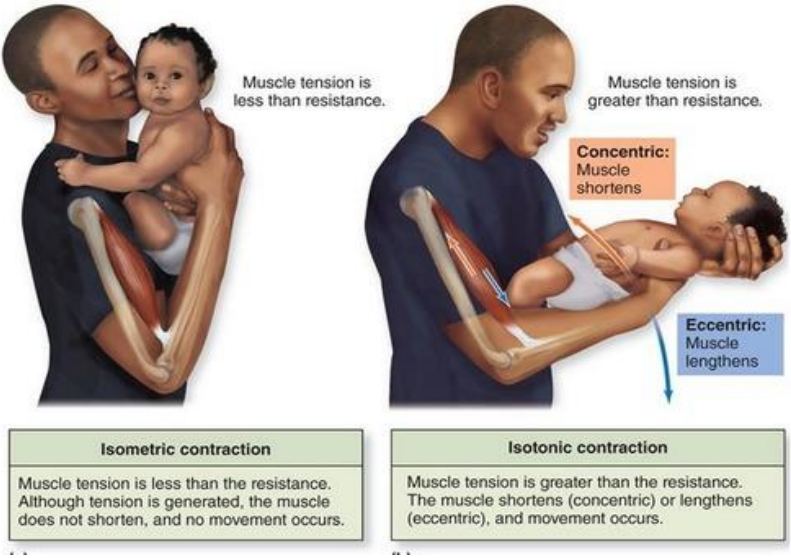

Figure 2.5 - Isometric and isotonic contractions

SKELETAL MUSCLE TONE

Muscle tone is the degree of tautness that is maintained in the healthy muscles, even when they are not actively contracting. Tone is a status of reflex and partial contraction that offers resistance to passive movements of a muscle. Stretch reflex (Figure 2.6) is the basis of this tonic activity of muscles. The gamma motor neurones that supply the intrafusal fibres of muscle spindles contract them, and when the intrafusal fibres shorten, the primary nerve endings on the intrafusal fibres are stimulated. These sensory fibres send signals to the spinal cord, and the anterior horn cells, via the alpha motor neurones, contract the extrafusal fibres of the muscle resulting in muscle tone.

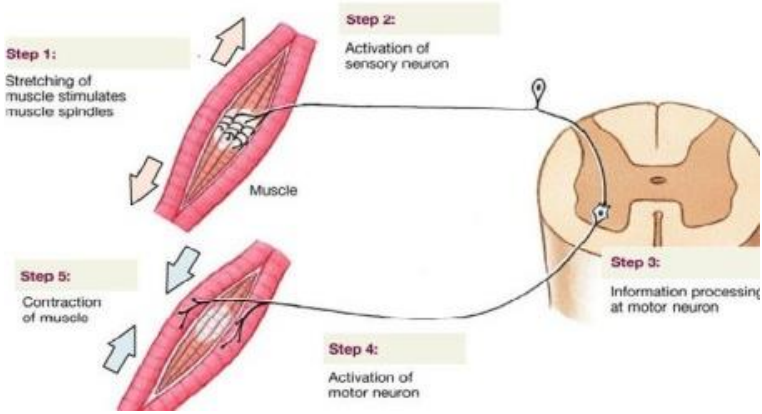

Figure 2.6 - Maintenance of skeletal muscle tone

MUSCLE REMODELLING: MUSCLE HYPERTROPHY AND ATROPHY

All muscles in the body undergo continuous remodelling to match the functions that they perform. Changes in fibre diameter, length, strength, fibre type and blood supply are seen during remodelling. The changes are rapid and can be observed as quickly as in few weeks.

Muscle hypertrophy refers to the increase in the total mass of a muscle. It is due to an increase in the number of actin and myosin filaments, causing enlargement of individual muscle fibres. Only a few strong contractions each day is adequate to cause significant hypertrophy that will be seen in 6-10 weeks. Muscle stretching causes hypertrophy as well by causing addition of new sarcomeres to the end of muscle fibres where they attach to tendons.

When a muscle remains idle for many weeks, the rate of degradation of contractile proteins exceeds the rate of their replacement, leading to atrophy. Muscle denervation is the most common cause of atrophy. Denervation atrophy is characterised by destruction of muscle fibre and replacement with fibrous and fatty tissue. The fibrous tissue has a tendency to contract and causes contractures. Stretching exercises during muscle atrophy is aimed at preventing debilitating and disfiguring contractures.

PHYSIOLOGY OF JOINTS

Skeletal muscles operate by applying tension to their points of insertion on the bone. The bones in turns, form different types of lever systems. Figure 2.7 shows the lever system activated by the biceps muscle. The study of different types of muscle and their lever systems, and their movements is called kinesiology. Kinesiology is studied in great depth by physiotherapists.

A joint is a place of articulation between two or many bones, irrespective of the degree of movement between those bones; for example, both elbow joint and skull sutures are joints in spite of the degree of movement in them being very different to each other. Almost all moments across a joint are caused by simultaneous activity of agonist muscle and relaxation of antagonist muscle. Reciprocal innervation leading to actions of agonists and antagonists are regulated by the higher centres, in the brain and the spinal cord.

Joints are classified according the structure (material that is in the joint) and function (type of movement that occurs at the joint). There are 3 types of joints based upon the structure: fibrous, cartilaginous and synovial. Joints are classified into synarthroses (immovable), amphiarthroses (slightly movable) and diarthroses (freely movable) according to the function. Skull sutures are a fibrous synarthrosis, while the shoulder and hip joints are synovial diarthroses. Vertebral joints and symphysis pubis are examples of fibrocartilaginous amphiarthroses.

Synovial joints are characterised by the presence of thin hyaline cartilage that lines the joint surface. The thin, spongy, cushion- like structure absorbs and distributes the weight placed on the bones and prevents damage to the bones of the articular surface. Synovial joints are covered by a bi-layered articular capsule; an outer tough fibrous tissue layer, and an inner synovial membrane layer. The joint cavity is occupied by a small volume of synovial fluid which is secreted by the cells of the synovial membrane. Synovial fluid acts as a lubricant that reduces friction between joint cartilage.

The table 2.1 and Figure 2.7 provide a classification of joints according to the main movements that take place in them.

Table 2.1 Functional classification of joints

Classification	**Movements**	**Example**
Hinge	Flexion-extension	Elbow
Pivot	Spin	Atlanto-axial
Ellipsoid	Flexion-extension Abduction -adduction	Radiocarpal, atlanto-occipital
Ball and socket	Triplanar	Shoulder, Hip
Plane	Slide +/- rotation	Intercarpal, intertarsal
Saddle	Biplanar	Sternoclavicular
Condyloid	Biplanar	Temporomandibular

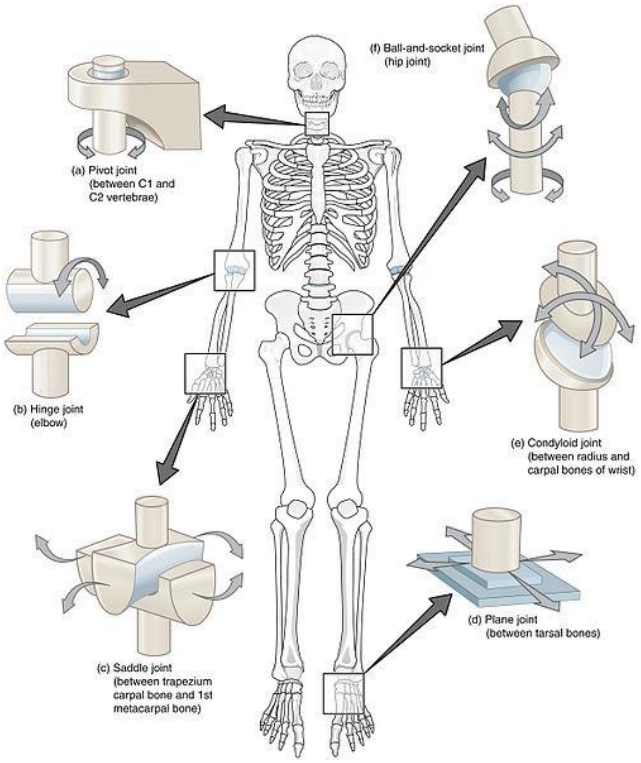

Figure 2.7 - Types of joints

CHANGES IN SKELETAL MUSCLES AND JOINTS WITH AGEING

Loss of skeletal muscle (sarcopenia) starts around 30 years and progresses throughout life. In old age, people become sedentary that leads to great degrees of muscle atrophy. During the process, the amount of muscle tissue and the number and size of muscle fibres gradually decrease. The quality of the muscle is impaired as the amount of fat and fibrous tissue increases. The oxidative capacity of the ageing skeletal muscle is diminished and the excitation -contraction coupling becomes impaired. As a consequence of sarcopaenia and other changes in the ageing muscle, there is a gradual loss of muscle bulk and muscle strength. This loss of muscle strength causes an increased stress upon the joints, particularly the knees, and may predispose to osteoarthritis

and postural sway leading to falls. However, the loss in muscle mass and strength can be partially prevented or at least significantly delayed by regular exercise. It has been shown that regular exercise that involves muscle training can restore muscle strength to maximum levels in the elderly.

Ageing joints are affected by changes in cartilage and in connective tissue, making them more prone to osteoarthritis. With cell senescence, the articular cartilage becomes thinner, and the proteoglycans in the cartilage are altered, which renders the joint less resilient and more susceptible to damage. Chondrocytes lose their ability to divide, and older chondrocytes become limited in their capacity to synthesise components of extracellular matrix such as collagen and ground substance. Mitochondrial dysfunction and increased production of reactive oxygen species (ROS) have been implicated in the pathogenesis. Additionally, joints become stiffer because the collagen networks within ligaments and tendons becomes more rigid and brittle, limiting the range of motion of joints. Lack of mechanical stimulation that is associated with increased amounts of bed rest and immobilisation prevalent in old age results in a thinner and softer cartilage, and studies have suggested that regular exercise training has the opposite effect on articular cartilage.

Further Reading

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3. Pathophysiology of Pain

Dr. Kumarangie Vithanage

Pain is a very common clinical presentation which has distinct features compared to other sensory modalities. Painful stimuli generally initiate potent withdrawal and avoidance responses. It warns that something is wrong, pre-empts other signals and has an inbuilt unpleasant affect. The International Association for the Study of Pain defines pain as *'an unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage'*.

TYPES OF PAIN

Pain has been classified into two major types: fast pain and slow pain. Fast pain is felt within 0.1 second following a painful stimulus, whereas slow pain begins only after 1 second or more and then increases slowly over many seconds or even minutes.

The character of fast pain could be acute, sharp, pricking or electric shock like pain. In contrast, slow pain could be described as a burning, aching, throbbing or a chronic pain which may lead to prolonged suffering. Slow pain can originate from skin as well as from any deep tissue or organ.

PAIN RECEPTORS

Pain receptors are free nerve endings placed widespread in the superficial layers of the skin as well as in certain internal tissues such as periosteum, joint surfaces, arterial walls, falx and tentorium in the cranial vault. Most other deep tissues are only sparsely supplied with free nerve endings. However, any extensive widespread tissue trauma can summate to cause slow chronic aching type of pain in most of these areas.

PAINFUL STIMULI

Pain can be elicited by multiple different stimuli. These can be classified as mechanical, thermal, and chemical pain stimuli. In general, fast pain

is elicited by mechanical and thermal types of stimuli, whereas slow pain can be elicited by all three types.

Some of the chemicals that excite chemical type of pain are bradykinin, serotonin, histamine, potassium ions, acids, acetylcholine, and proteolytic enzymes. In addition, prostaglandins and substance P enhance the sensitivity of pain endings but do not directly excite them. Chemical substances are especially important in stimulating slow suffering type of pain that occurs following tissue trauma. Bradykinin has been recognized as to stimulate free nerve endings during tissue trauma. The intensity of pain experienced correlates with the local increase in both potassium ion concentration and proteolytic enzymes, which directly attack the pain sensitive free nerve endings. Pain induced by tissue ischaemia is rapidly felt when the ischaemic tissue undergoes higher rate of metabolism.

PERCEPTION OF PAIN

Free nerve endings are receptors for all pain causing stimuli. There are 2 pathways that originate from these receptors corresponding to two types of pain viz. a fast-sharp pain pathway and a slow-chronic pain pathway.

The fast-sharp pain signals are elicited by either mechanical or thermal pain stimuli. They are transmitted in the peripheral nerves to the spinal cord by small type Aδ fibres at velocities between 6 and 30 ms^{-1}. Conversely, the slow-chronic type of pain is elicited mostly by chemical types of pain stimuli but sometimes by persisting mechanical or thermal stimuli. This slow-chronic pain is transmitted to the spinal cord by type C fibres at velocities between 0.5 - 2 ms^{-1}.

This dual innervation of pain afferents is the cause of 'double' pain sensation. There is first a sharp pain followed by a dull chronic aching pain. The fast pain afferents exert a protective function via removing the body away from the painful stimulus whereas slow pain carries the chronic suffering component of the pain.

PAIN PATHWAY

The first order neurons from nociceptors and thermoreceptors synapse on the neurons in the dorsal horn of the spinal cord. The axons of second order neurons which originate from the dorsal horn cross the midline and ascend in the ventrolateral spinal cord forming ventrolateral spinothalamic tract which synapse in the ventral posterolateral nucleus of the thalamus. Some of the fibres carrying nociceptive input from the dorsal horn synapse at the reticular formation forming spinoreticular pathway. These thereafter project to centrolateral nucleus of the thalamus. Third order neurons originating from the thalamus synapse at the somatosensory cortex and structures involving limbic system. (Figure 3.1)

Although the sensory cortex is the final destination of pain pathways, complete removal of somatic sensory areas of the cortex has still enabled pain perception. Thus, it is likely that pain perception occurs at subcortical structures. However, the cortex does play an important role in interpreting quality of pain.

Electrostimulation studies have demonstrated that electrical stimulation of subcortical areas as reticular areas of brain stem and intralaminar nuclei of thalamus where slow pain fibres terminate, has a strong arousal effect on the entire brain. This explains why it is virtually impossible to sleep when one is in severe pain.

Furthermore, neuroimaging techniques including PET and fMRI studies have demonstrated that in healthy humans' somatic pain activates the primary and secondary somatosensory cortex and cingulate gyrus contralateral to the stimulus. In addition, the amygdala, frontal lobe and insular cortex gets activated.

Therefore, these functional neuroimaging techniques have demonstrated two important components of pain pathways. Pathway to the primary somatosensory cortex remains responsible for the discriminative aspect of pain (pain localisation). In contrast, the pathway that includes synapses in the brainstem reticular formation and centrolateral thalamic nucleus projects to the frontal lobe, limbic

system, and insular cortex. This pathway mediates the motivational-affective component of pain.

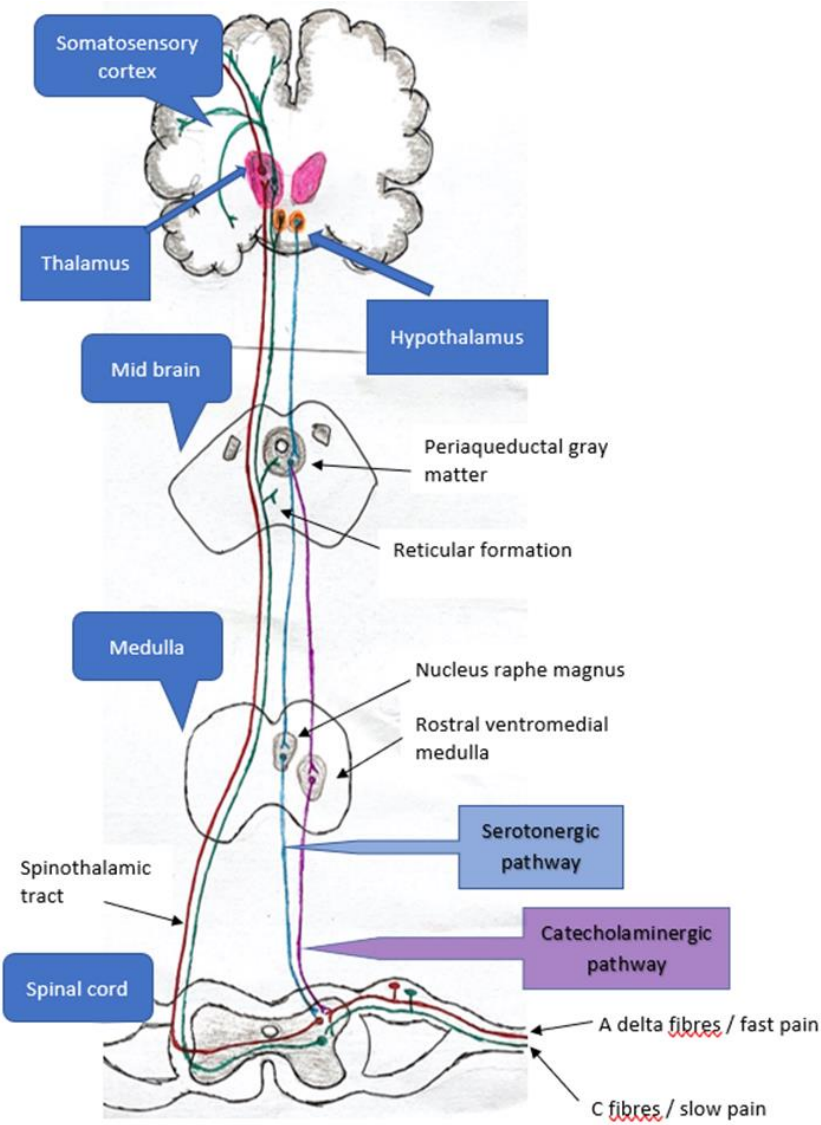

Figure 3.1 - Ascending and Descending Pain Pathways

MODULATION OF PAIN TRANSMISSION

Different individuals perceive pain to various degrees. Pain sensation as a sensory modality is unique as it suppresses or modifies its own sensory perception.

Pain transmission can be interrupted by actions within the dorsal horn of the spinal cord at the site of first order sensory afferent termination. Many persons have learned from practical experience that rubbing an injured site helps to alleviate the pain to a certain extent. The relief is supposed to be due to the simultaneous activation of innocuous cutaneous mechanoreceptors whose afferents emit collaterals that also terminate in the dorsal horn. The activity of these cutaneous mechanosensitive afferents may reduce the responsiveness of dorsal horn neurons to their input from nociceptive afferent terminals. This is described as the gate-control mechanism of pain modulation. It serves as the rationale behind the use of transcutaneous electrical nerve stimulation (TENS) for pain relief where electrodes are used to activate larger Aα and Aβ fibres near the injury.

ANALGESIC SYSTEMS

Opioids are commonly used pharmacological agents for their analgesic effects imparted on various places in the nervous system including in the spinal cord and dorsal root ganglia. Endogenous opioids (e.g. encephalin and dynorphin) are analgesic substances which are naturally present with in the body which mimic the action of pharmacological opioids. There are interneurons in the dorsal horn where nociceptive afferents terminate that contain endogenous opioids. Opioid receptors are located both on the distal terminals of first order pain fibres and on dendrites of dorsal horn neurons, allowing for both presynaptic and postsynaptic sites of actions for opioids. Activation of the postsynaptic opioid receptors hyperpolarizes the dorsal horn interneuron by causing an increase in K^+ conductance. Activation of the presynaptic opioid receptors leads to a decrease in Ca^{2+} influx, resulting in a decrease in release of glutamate and substance P which are the neurotransmitters released at the dorsal horn. Together, these actions reduce the duration of the excitatory postsynaptic potential in the dorsal horn neuron.

Activation of opioid receptors on dorsal root ganglia cell bodies also contributes to reduced transmission from nociceptive afferents.

The midbrain periaqueductal grey (PAG) is another site of action for morphine and endogenous opioid peptides. An injection of opioids into the PAG induces analgesia. The PAG is part of a descending pathway that modulates pain transmission by inhibiting primary afferent transmission in the dorsal horn. These PAG neurons project directly and activate two groups of neurons in the brainstem: serotonergic neurons in the nucleus raphe magnus and catecholaminergic neurons in the rostral ventromedial medulla. Neurons from these regions project to the dorsal horn of the spinal cord where they release serotonin and norepinephrine, respectively. The result is to inhibit the activity of dorsal horn neurons that receive input from nociceptive afferent fibres.

This inhibition at the level of the dorsal horn, at least in part, is thought to be due to the activation of the dorsal horn enkephalin-containing interneurons as described above.

The analgesic effect of electroacupuncture may involve the release of endogenous opioids and activation of this descending pain modulatory pathway. Electroacupuncture activates ascending sensory pathways that emit collaterals in the PAG and in the brainstem serotonergic and catecholaminergic regions. The analgesic effect of electroacupuncture is prevented by administration of naloxone, an opioid receptor antagonist.

STRESS INDUCED ANALGESIA

It is a well-known fact that at battlefields injuries cause minimal or no pain enabling the battlefront to carry on. However, the same injuries would start causing pain the moment the battle is over. This is a classic example of stress induced analgesia. Reduction in pain sensitivity occurs when faced with a stressful event due to release of norepinephrine, perhaps from brainstem catecholaminergic neurons, in the amygdala may contribute to this phenomenon. The amygdala is a part of the limbic system that is involved in mediating the motivational-affective responses to pain. The release of endogenous cannabinoids perhaps

may contribute to stress-induced analgesia. These chemicals can act on two types of G-protein-coupled receptors (CB_1 and CB_2). CB_1 receptors are found in many brain regions, and activation of these receptors accounts for the euphoric actions of cannabinoids. CB_2 receptors are expressed in activated microglia under pathologies that are associated with chronic neuropathic pain.

ABNORMAL PAIN SENSATION

Pain, though unpleasant can often be protective in identifying danger with regard to tissue injury. However, there are instances where pain signalling goes wrong. This results in abnormal pain which is described in numerous terms based on the abnormal pain sensation perceived and their respective circumstances giving rise to them.

Neuropathic pain refers to pain or any other abnormal sensation perceived as a result of a primary pathology involving the nervous system. Therefore, neuropathic pain could be due to a dysfunction of motor, sensory, autonomic system or a combination of any. Neuropathic pain could be spontaneously felt in the absence of an external noxious stimulus. Pain could be paroxysmal or continuous and is often described as burning, tingling or a shooting pain. There are several types of neuropathic pain.

- Allodynia: abnormal painful response to a normally innocuous stimulus.
- Hyperalgesia: severe exaggerated prolonged pain experiences to a minimally noxious stimulus.
- Hyperaesthesia: heightened sensitivity to a stimulus

Neuropathic pain can result from damage to neurons of either peripheral or central nervous system in. However traditionally the term 'neuropathic pain' has been referred to abnormal pain from peripheral nerves although pain originating from damage to central nervous system neurons has been referred to as 'central pain'.

When a peripheral nerve fibre is damaged it attempts to self-repair. However, it may not completely heal to its' original form but instead forms a swelling around the damaged site of the axon known as a

neuroma. Spontaneous firing and electrical activity occur around the neuroma which is thought to be due to altered distribution, expression and gating properties of sodium channels. Ectopic impulse generation is seen also at the level of the spinal cord and dorsal root ganglia. In addition to neuroma formation, peripheral pain receptors become increasingly sensitized at the site of tissue injury with a lower threshold potential for firing even in response to non-painful stimuli. This ectopic firing by the damaged nerves is influenced by physical stimuli as heat or cold, and by the presence of metabolic and chemical constituents existing during tissue injury.

Injury also includes Schwann cells and glia surrounding axons. These are non-neuronal cells providing support and nutrition to the nerves resulting in changes in axonal function. Furthermore, uninjured accents can spread into areas of injury (sprouting) which results in further exaggeration of abnormal pain sensation.

CENTRAL SENSITISATION

Changes occur in the dorsal horn of the spinal cord after nerve injury. Repetitive C fibre activation by noxious stimuli leads to a prolonged dorsal horn response. This phenomenon has been termed 'wind-up'. A net reduction in inhibition of pain transmission at the dorsal horn by the action of pain causing neurotransmitters and by excitotoxic death of inhibitory interneurons occurs. Simultaneously there is strengthening of excitatory synaptic connections. Afferent axons elicit ectopic activity resulting in exaggerated output onto spinothalamic tract. this process involves neurochemical changes mediated via N-methyl-D-aspartate (NMDA), neurokinins, and nitric oxide. The ultimate effect of all these changes is that the sensory threshold for pain perception is lowered with spread of the receptive field.

There is also structural rewiring and sprouting of C fibres into adjacent laminae at the dorsal horn triggered by loss of nerve growth factor. This explains the phenomenon of allodynia where non painful stimuli as touch causing pain due to disorganized C fibre terminations.

Following injury there is evidence of cortical remapping and reorganisation in both the primary somatosensory and motor cortices and in the subcortical areas. This is well recognized following limb amputation. Lack of afferent input from the amputated limb leads to less occupation of the corresponding area of the somatosensory cortex. As a result, the neighbouring cortical area (representing a different anatomical site) expands. This is referred to as *phantom limb pain*. These mechanisms not only develop phantom limb pain very soon after amputation, but also that the phantom limb can sometimes be mapped out by touching a very different site of their body (e.g. pain in phantom hand felt by touching side of face). Reduction in the intensity of the phantom limb pain by effective treatment has shown to reverse the cortical changes.

Further Reading

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4. A Synopsis of Inflammation and Therapeutics in Musculoskeletal Diseases

Dr. Shehan Silva

INFLAMMATION

Inflammation is initiated when leucocytes such as macrophages and mast cells encounter a pathogen, chemical or physical agent that causes injury to tissues. A cascade of events results in activation of cells by the release of cytokines: tumour necrosis factor-α (TNF-α), interleukins (IL-x) and other agents such as histamine and prostaglandins. IL-6 and TNF-α cause the endothelial cells to express cellular adhesion molecules which signals leucocytes such as neutrophils to attract, bind, translocate from circulation and migrate to the injured region. These cells thereafter phagocytose the agent of injury and destroy it. There is also collateral damage caused by neutrophils and macrophages to the surrounding tissue by the release of metalloproteinases and collagenases.

Pro-inflammatory eicosanoids and platelet activating factors are produced by mast cells, macrophages and endothelial cells. Phospholipid substrates in the cell membrane are converted to active chemicals by phospholipase A_2, cyclo-oxygenase (COX) and lipo-oxygenase. COX is composed of 2 isoforms. COX1 is found in most tissues and acts constitutively (constantly produced regardless of circumstance - *House Keeping Gene*). COX2 is inducible by IL-1β and TNF-α, and is found in macrophages and connective tissue cells (Figure 4.1). The active chemicals are as follows

a) Leukotrienes
   - activation and accumulation of leucocytes, vasoconstriction and increased vascular permeability.

b) Prostaglandins (PG)
   - vasodilation of microcirculation and pain signalling
   - prostaglandins protect the gastrointestinal mucosa and engage in renal homeostasis (by the action of PGI_2 and PGE_e)

c) Platelet activating factor and Thromboxane A2 – activation of coagulation and fibrinolytic cascades

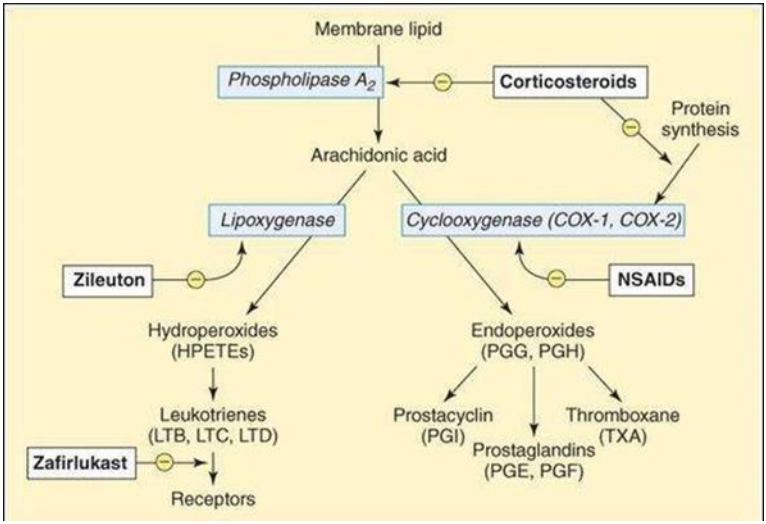

Figure 4.1 - Eicosanoid synthesis

DRUGS USED IN THE MANAGEMENT OF NOCICEPTIVE PAIN AND INFLAMMATION

a) Non-Steroidal Anti-Inflammatory Drugs (NSAIDs)

The common point of action in this group of drugs is by inhibition of synthesis of prostaglandins. Structurally heterogenous, these have varied modes of action such as inhibition of lipoxygenases, superoxide generation and scavenging, neutrophil aggregation and adhesion etc.

Approximately 60% of patient will respond to any NSAID while the remainder who do not respond to one may well respond to another. The analgesic effect commences with the first dose and a full effect is present within one week. The anti-inflammatory action is however delayed, occurring within 3 weeks. It is essential that patients are informed of this dual action of analgesia and anti-inflammation to ensure adequate time is given in trialling a drug. If there is poor response 1-3 weeks after commencement, switching of drugs ought to be considered.

NSAIDs are fully absorbed by the gastrointestinal tract. They are not subjected to first pass metabolism as they are highly protein bound.

Majority of them are weak acids and therefore localise preferentially in synovial tissue of inflamed joints.

Gastrointestinal irritation is caused by the inhibition of COX1 production of mucosal PG which is needed to maintain integrity of the mucosa in an acidic environment. Dyspepsia, upper gastrointestinal ulceration and haemorrhage, small intestinal ulceration and stricture formation are recognised manifestations. Gastrointestinal adverse effects present with increased risk in those above 65 years of age, past history of peptic ulcer disease, heavy smoking and alcohol abuse, presence of *Helicobacter pylori* infections and in treatment with low dose aspirin, steroids and anticoagulants. The risk of these side effects can be reduced by prescription of proton pump inhibitors (PPI), H_2 receptor blockers (less effective than PPIs) or misoprostol (a prostaglandin analogue that inhibits secretion of gastric acid in parietal cells). H_2 receptors are advocated in twice the usual dosage (famotidine 40 mg bd or ranitidine 300 mg bd)

NSAIDs reduce renal perfusion especially in those who depend on PG mediated vasodilation (congestive cardiac failure, chronic renal disease and cirrhosis). There is no major difference with COX-2 selective drugs and other NSAIDs regarding renal adverse effects. Furthermore, NSAIDs can cause fluid retention, oedema formation, hypertension and worsening dyspnoea. This is due to counteraction of the action of prostaglandins in its diuretic and natriuretic effects. Other adverse events in kidneys include papillary necrosis and interstitial nephritis.

Thromboxane is a PG produced by platelets by the COX-1 system which promotes platelet aggregation. In contrast, prostacyclin is a PG produced by the endothelium that inhibits platelet aggregation. COX-2 inhibitors therefore have no impact on platelet aggregation. However, they do inhibit COX-2-mediated prostacyclin synthesis in the vascular endothelium causing greatest risk with high doses in the long term. This risk is also present with NSAIDs such as diclofenac, aceclofenac and high dose ibuprofen (2.4 g daily). It is shown that low dose ibuprofen (<1.2 g daily) and naproxen have lower risks.

NSAIDs can also induce hypersensitivity, asthma exacerbations and rhinitis (cross sensitivity is present), haematological and hepatic damage.

Non-COX-2 selective drugs

a) Paracetamol (Acetaminophen)

This simple analgesic is useful to treat mild to moderate pain. It also has antipyretic features. The efficacy is comparable to aspirin in analgesic properties. However, paracetamol has weak anti-inflammatory actions. Its advantages include absence of gastrointestinal and cardiovascular side effects. Although paracetamol does not cause acute kidney injury, analgesic nephropathy is a well complication with over usage.

It is well absorbed by the gastrointestinal tract. The liver primarily inactivates it by conjugation. A small fraction is transformed to N-acetyl-p-benzoquinone imine (NAPQI) which when conjugation is saturated is oxidised by glutathione in level. When these pathways are overwhelmed, toxicity from paracetamol manifest. The maximum daily dose administered should be 60 mg/kg (4g for a normal adult). Paracetamol is preferred to NSAIDs in older persons requiring mild to moderate analgesia for non-inflammatory conditions.

b) Aspirin (Acetylsalicylic acid)

Aspirin acts in a unique manner compared to other NSAIDs by irreversibly binding and inhibiting COX. Therefore, its effects last until new COX is formed. Other than its anti-inflammatory effects, it has effects on respiration by stimulating the respiratory centre directly and by increasing CO2 production (increased peripheral O2 consumption) indirectly. Therefore, metabolic acidosis with respiratory alkalosis can manifest in overdose.

It is absorbed well by the upper gastrointestinal tract. Hydrolysis removes the acetyl moiety and is thereafter conjugated with glycine. This occurs at zero order kinetics at higher/ toxic doses which causes accumulation as salicylate. This is excreted via glomerular filtration and secretion to the proximal tubule. Other than the usual adverse effects of NSAIDs, patients could develop salicylism (tinnitus, auditory

disturbance, headache and confusion) and hypersensitivity type 1 (even leading to angioedema). Doses of 300 – 900 mg every 4-6 hours are indicated for analgesia.

c) Acetic acids

Drugs such as *indomethacin*, *diclofenac* and *aceclofenac* cause salt and water retention. Headache (similar to migraine) develops due to cerebral oedema. Furthermore, there can be vomiting, dizziness and ataxia. It is advised to start low and go slow to prevent this. Medication of this class are best avoided in gastroduodenal, renal or central nervous system diseases. However, *sulindac* seem to have less renal effects as renal prostaglandin synthesis are not affected.

d) Fenamic acid

Mefenamic acid can cause diarrhoea, epigastric discomfort, peptic ulcer disease and haemolytic anaemia. Older adults can develop non oliguric renal failure especially when dehydrated.

e) Propionic acids

Although the incidence adverse events are less upper gastrointestinal symptoms could occur with drugs such as *ibuprofen, naproxen, ketoprofen.*

f) Enolic acids

Piroxicam and *meloxicam* are examples of enolic acids

COX-2 selective drugs

These drugs inhibit COX-2, with 5 times greater potency than COX-1. They are associated with few gastrointestinal symptoms although other adverse effects may occur. Drugs such as *celecoxib*, *etoricoxib*, *meloxicam* and *etodolac* are members of this group. *Rofecoxib* was taken off the market as it was shown to increase the risk of myocardial infarction (VIGOR study). Patients with prothrombotic risk, coronary artery and cerebrovascular diseases should not receive these drugs.

b) Glucocorticoids

Glucocorticoids (GC) exert their effect by alteration of gene transcription. They readily diffuse through the cell membrane and bind

to cytosolic glucocorticoid receptors (GR) which in turn works in the nucleus. The GC-GR complex thereafter acts by

1) Binding to glucocorticoid receptor element targeting gene promotors to increase transcription of anti-inflammatory genes I-κB. This inhibits activation of Nuclear Factor (NF)-κB, and cytokines IL-4, IL-10, IL-13 and Transforming growth factor (TGF)β.
2) Interfering with binding of transcription factors activating protein (AP)-1 and NF-κB to their responsive elements. This decreases the transcription of pro-inflammatory mediators including IL-1β, TNFα, IL-2, metalloproteinases, COX-2 and nitric oxide synthase.
3) Increasing syntheses of polypeptide lipocortin-1 which inhibits phospholipase.

Furthermore, GCs induce apoptosis and inhibit proliferation and activation of leukocytes thus reducing lymphocytes, monocytes and eosinophils in circulation

Steroids are a group of drugs that are very useful for dramatic relief of symptoms. However, adverse effects limit the use of these agents. It is however justified in certain circumstances.

1) provision of interim relief of inflammation as DMARDs take time to act (bridging therapy)
2) single spaced mega doses (pulsed treatment) to suppress highly active inflammatory disease and to utilise as a respite in changing DMARDs. e.g. methylprednisolone 1g IV for 3 consecutive days.
3) High doses to effectively prevent inflammation especially that of vasculitis and rheumatoid lung. e.g. high dosed prednisolone 20-40 mg daily.
4) Failed therapeutic trial or development of adverse effects of DMARDs warrants a daily small dosage to control inflammation with minimal side effects. e.g. prednisolone 7.5 mg daily.
5) Synergistic effect with standard therapy to reduce destructive pathology.

Intra-articular injection of corticosteroids is considered in monoarticular or predominantly single joint involvement in polyarticular disease. However, too frequent administration may promote joint damage. Agents such as methylprednisolone and dexamethasone are employed. It is essential that local and intra-articular infections are excluded and that the drug is administered by aseptic precautions.

Table 4.1 - Effects and equivalence of commonly used glucocorticoids

Compound (tablet strength in mg)	Anti-inflammatory effect (glucocorticoid)	Na retention effect (mineralocorticoid)	Equivalent dosage (mg)
Hydrocortisone (20)	1	1	20
Prednisolone (5)	4	0.8	5
Methylprednisolone (4)	5	Minimal	4
Dexamethasone (0.5)	30	Minimal	0.75

Hydrocortisone is a naturally occurring steroid. It is available as an oral salt and soluble salt (acetate) which can be given intravenously. Furthermore, there is an acetate suspension that can be given intra-articular. *Prednisolone* is a biologically active agent with predominant anti-inflammatory agent with little mineralocorticoid effect. This is used as the conventional anti-inflammatory pharmacotherapy. *Methylprednisolone* is similar to prednisolone but is used intravenously for megadose pulse therapy.

Serious adverse effects follow prolonged administration. However, spread of infection could occur with 1-2 doses in the early days. Serious unwarranted effects of steroids occur in daily doses of hydrocortisone 50 mg. These include the following.

- Endocrine - Cushing syndrome, secondary diabetes mellitus, hypothalamic/pituitary/adrenal axis suppression

- Musculoskeletal – Proximal myopathy, tendon rupture, osteoporosis, avascular necrosis of bones
- Immune – Immunosuppression leading to activation of tuberculosis, severe varicella infections, relapse of herpes zoster, unwelcomed live vaccine infection, widespread candidiasis
- Gastrointestinal – peptic ulcer disease, acute pancreatitis
- Central nervous system - Depression, psychosis, euphoria, relapse of schizophrenia, epilepsy, raised intracranial pressure
- Ophthalmic – posterior subcapsular lens cataract, glaucoma, corneal/scleral thinning
- Miscellaneous – poor wound healing

c) Opioids

Opioids are useful in managing nociceptive pain as they have stronger analgesic effect compared to NSAIDs. Furthermore, their efficacy is more for visceral pain rather than that of somatic origin. Opioids are also used as a third line therapeutic agents for neuropathic pain as they have an efficacy comparable to tricyclic antidepressants and gabapentin at the expense of side effects. The therapeutic effect of opioids is augmented with the quick onset of action and accompanying euphoric effect.

Analgesia is generated via the neuroinhibitory μ receptors found at spinal and supraspinal levels. Opioid acts in similar fashion to endorphins and encephalins (body's own opioids) that bind to these receptors in blockage of neurotransmitter release by hyperpolarisation. The euphoric and analgesic effects are generated by μ_1 receptors in the brain (synergistic action). The μ_2 receptor deals with respiratory depression and reduced gut motility. The κ receptor is responsible for effects at the spinal cord. The role of the δ receptor is less clear in humans. (Figure 4.2)

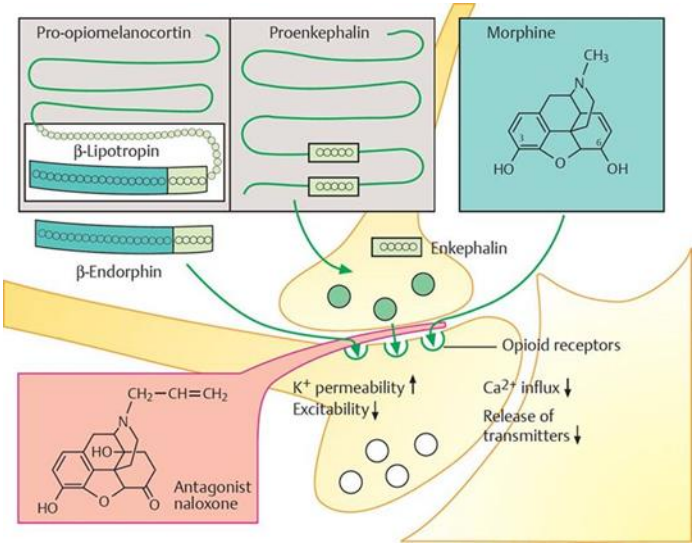

Figure 4.2 - Action of opioids

However, opioids have a higher risk of adverse effects in particularly in the older persons. There is rapid development of tolerance to the drugs which can also lead to risk of misuse and addiction. The effects on the central nervous system includes the following.

- Nausea and vomiting
- Confusion, clouding of mentation and cognitive impairment
- Mood changes
- Sedation
- Urinary retention
- Constipation (central and peripheral effects of reduced gastrointestinal motility)
- Respiratory depression
- Hypotension (central and histamine release causing vasodilation)
- Suppression of cough reflex

Table 4.2 - Analgesia of commonly used opioids and their equivalence

Moderate analgesia	**Strong analgesia**
Codeine 30 - 60 mg Co-codamol (codeine paracetamol 8/500, 15/500, 30/500) x2 Tramadol 50 -100 mg	Morphine 10 mg (PO) Morphine 5 mg (IV, IM, SC) Dihydrocodeine 100 mg (PO) Oxycodone 6.6 mg (PO) Tramadol 100 mg (PO) (maximum 400mg /d)
Equivalent analgesic dosage Morphine (PO) 10 mg Morphine (IV, IM, SC) 5 mg Dihydrocodeine (PO) 100 mg Oxycodone (PO) 6.6 mg Tramadol 100 mg	

In chronic pain management, 10-30 mg of immediate release morphine administered orally, 4 hourly with break through doses can be administered. The total daily dose needed to control pain is then calculated which can be converted to modified release morphine 12 hourly. Alternatives to oral administration include transdermal, per rectal and subcutaneous routes. Long-acting preparations such as modified T release oxycodone and transdermal fentanyl can be employed.

When prescribing opioids in the older adults it is essential that the prescriber is mindful of addressing risks of constipation, falls, cognitive impairment and urinary retention (especially in those with prostatism). These need to be managed non pharmacologically and pharmacologically with good counselling. Dosages need to be altered in hepatic impairment (precipitation of hepatic encephalopathy) and renal impairment (poor excretion of active metabolites). Fentanyl is appropriate in renal impairment as the metabolites are inactive. Tramadol is known to cause seizures and serotonin syndrome (when co-prescribed with SSRI, SNRI, TCS and herbal supplements such as ginseng, nutmeg and St. John's wort).

With frequently repeated therapeutic doses of morphine, the effectiveness gradually wanes off. A larger dose is required to reproduce the original response. This is known as tolerance. Furthermore, there is withdrawal (abstinence syndrome) when the drug is stopped or an antagonist is prescribed. These include agitation, hyperalgesia, hyperthermia, hypertension, diarrhoea and mydriasis. There is release of all pituitary and adrenal hormones. Affective symptoms such as dysphoria, anxiety and depression can develop. These phenomena are highly aversive and motivate the recipient to make effort to avoid withdrawal states.

Codeine is a low efficacious drug. Ten percent of it is converted to morphine. There is poor efficacy for severe pain (most actions are 10% that of morphine). Dependence occurs much less. *Pethidine* has less spasmodic activity on smooth muscles and is safer in asthmatics (less histamine release). *Tramadol*, other than its weak opioid analgesic effects inhibits neuronal noradrenaline uptake and enhance serotonin release. It is relatively rapidly absorbed from the gut. Twenty percent undergoes first pass metabolism but 30% is excreted unchanged in urine. It is almost effective as pethidine postoperatively and as morphine in chronic pain. Tramadol is less likely to constipate, depress respiration and cause dependence. *Meptazinol* is a high efficacy partial agonist with central cholinergic effect used for moderate intensity acute and chronic pain. It does not caused euphoria. Furthermore, there is no observed withdrawal symptoms. *Fentanyl* has higher efficacy than morphine but last shorter (up to 1 hour). It has fewer cardiovascular effects and has less tendency to release histamine. There is high lipid solubility causing entry to brain (peak analgesia in 5 min after IV injection). Fentanyl is available as self-adhesive patches, releasing the drug at 25 µg/hr for 72 hours

Nociceptive Pain Management

The WHO analgesic ladder is a useful protocol to consider which agent to use (Figure 4.3). To maintain the relief from agony, medication needs to be administered at the due time ('by the clock') rather than regular scheduled basis. The dosage frequency depends upon the nature of action (short vs. long action). The oral route is usually the preferred

route due to convenience ('by the mouth'). In practically difficult scenarios, the least invasive one is selected (e.g. sub lingual or sub cutaneous prior to intravenous). Intramuscular route should). Intramuscular mode should never be selected. Prompt administration should be in the order of non-opioids, as necessary mild opioids and ten strong opioids until the patient is free of pain ('by the ladder'). Selections should be appropriate to the severity. Severe pain may require commencement at the top rung of the ladder. When the pain is controlled, the patient should be maintained on the dose that was effective. The medication need not be tailed down unless the pathological process is resolved. Adjuvants can be initiated along main line medication. These include antidepressants (e.g. amitriptyline), anticonvulsants (e.g. gabapentin), glucocorticoids (e.g. dexamethasone) and anxiolytics (e.g. diazepam).

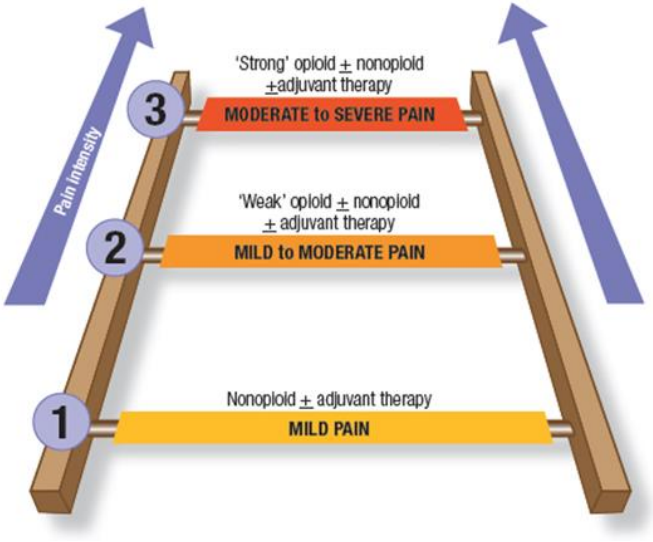

Figure 4.3 WHO Pain Ladder

Topical Analgesia

Topical NSAID analgesics such as diclofenac salts and ketoprofen have shown to demonstrate relief in a fraction of patients with osteoarthritis. There is hardly any strong evidence in other chronic inflammatory

conditions although it is shown to penetrate the interface and enter tissues. These preparations are accompanied with local cutaneous reaction leading into generalised hypersensitivity. Although absorption is less than oral agents there are reports of gastrointestinal and renal damage caused by topical agents. NSAIDs are available as suppositories as well.

Lidocaine is available as creams or patches and is used for localised neuropathic pain. Capsaicin depletes substance P from the primary afferent -neurons. Furthermore, it causes a burning sensation thus stimulating the gate concept of pain control. Localised neuropathic pain requires 0.075% strength while osteoarthritis can be managed by 0.025% cream.

Analgesia for older persons

Poor analgesia in older adults results in poor quality of life. Sleep may be affected. There is restriction is mobilisation reducing socialisation. This may all escalate to psychiatric conditions such as depression. There is no best of ideal analgesic for older adults. However, one or two drugs may be the best option for an individual. This is based on efficacy, safety, cost effectiveness and convenience of administration. Furthermore, the type and severity of pain, comorbidities, concurrent medication and social factors too will influence a good decision-making process. Patients and care givers should be offered appropriate advice and precautions. Frequent medication review and close monitoring also are essential components in a good prescriber.

DISEASE MODIFYING DRUGS

Disease modifying antirheumatic drugs are believed to restore and maintain a more normal immunity. They are used in inflammatory arthritides and in peripheral disease with spondyloarthropathy. DMARD action takes a longer period of time. Therefore, patients need NSAIDs and bridging therapy with steroids when commenced on these agents. These drugs need regular monitoring as they affect the immune system, haemopoiesis and other organs (kidney or liver). Patients on DMARDS are inoculated with live attenuated vaccines. Furthermore, they are contraindicated in pregnancy and lactation.

Methotrexate is used to treat chronic inflammatory diseases including rheumatoid arthritis and psoriatic arthritis. It is a folate analogue which inhibits folate dependent enzymes in purine biosynthesis. Thus, lymphocyte proliferation is affected. Furthermore, it inhibits 5-aminoimidazole-4-carboxamide ribonucleotide (AICAR) transformylase (another folate dependent enzyme). Intracellular levels of AICAR are therefore raised which thereafter inhibits adenosine deaminase that degrade adenosine. Adenosine has anti-inflammatory effects such as reducing TNF-α and IFN-γ.

Leflunomide affects pyrimidine synthesis by inhibiting the mitochondrial drug dihydro-orotate dehydrogenase through its active metabolite A77 1726. Azathioprine and mycophenolate mofetil (MMF) also inhibit purine biosynthesis thus affecting proliferating lymphocytes. Azathioprine is used in the management of rheumatoid arthritis. The enzyme thiopurine S-methyltransferase (TPMT) deals with activation and deactivation of steps in azathioprine.

Hydroxychloroquine is used in rheumatoid arthritis as an adjunct to other DMARDS. Although the action is not very well known, it can reduce production of cytokines such as TNF-α and IL-1β. Sulfasalazine is composed of mesalazine (5-amino-salicylic acid, 5-ASA) and sulphapyridine. These two compounds are released in the gut by colonic bacterial enzymes. Only a quarter of mesalazine is absorbed which reduces eicosanoid, IgM, IgG, IL-2 and TNF-α production. Furthermore, it reduces HLA-DR upregulation by IFN-γ and reduced production of free radicals. The sulphapyridine component has an action in rheumatoid arthritis to inhibit IL-2 induced T cell proliferation and inhibit macrophage Il-1 and IL-12 production.

DMARDs are prescribed as per differing regimes up to 3 agents. They may be given in sequence (trialled to see individual efficacy) with or without a washout period and or steroid cover. They can be also given in combination in the beginning. Patient and physician preference, the course of disease and response to therapy are determinants of the strategy.

Table 4.3 - Commonly used DMARDs				
Agent	**Prescription**	**Adverse Effects**	**Interaction**	**Contraindications**
Methotrexate	P.O 7.5 – 10 mg once weekly Increased to 25 mg as bone marrow and liver functions allow. Parenteral preparations available. Folic Acid 5 mg weekly prescribed on a different day	Bone marrow toxicity Hepatotoxicity Regular (monthly FBC and LFT) Pneumonitis (Baseline CXR needed) Mouth ulcers and nausea (Folic acid used)	Use with trimethoprim, cotrimoxazole or sulphonamides create a risk for megaloblastic anaemia and pancytopaenia. (Folinic acid is useful)	Moderate to severe renal impairment Liver disease Active infection Prenancy and Lactation *Men and women should be counselled on effective contraception while on and for 6 months after stopping*
Azathioprine	P.O. 25-50 mg initially rising over several weeks to 1.5-2.5 mg/kg daily. Erythrocyte TPMT activity assessed prior	Bone marrow toxicity Hepatotoxity Susceptibility to infection (Regular FBC and LFT) Nausea, alopecia, allergy Risk of malignancy (long-term)		Relatively safe in pregnancy
Mycophenolate mofetil		Myelosuppression Hepatoxicity Electrolyte imbalance Dyslipidaemia Increased risk of malignancy Pancreatitis		Pregnancy

Leflunomide		Diarrhoea Hepatitis Leucopaenia Alopecia Hypertension Allergy (Elimination can be enhanced with cholestyramine)		Liver disease Pregnancy (Keep a gap of 2 years after cessation for conception) Hypoproteinaemia Immunodeficiency Pregnancy and lactation
Hydroxychloroquine	200-400 mg daily	Very low side effect profile GI disturbance Rashes Blood dyscrasias Retinal toxicity (Bull's Eye Maculopathy) – Annual visual acuity checks advised		Caution in hepatic or renal disease, neurological disease, G6PD deficiency and porphyria Contraindicated in pregnancy and lactation
Sulfasalazine		Cytopenia Hepatitis Lupus like syndrome		

BIOLOGICS

Monoclonal antibodies and fusion proteins are used to selectively target specific components of the inflammatory response. They show greater efficacy and fewer side effects compared to conventional immunosuppressives.

1) TNF-α is targeted by most of these medication to counter act chronic inflammation. These include
   - *Infliximab* – chimeric monoclonal Ig1 Antibody (RA, psoriatic arthritis, ankylosing spondylitis)
   - *Adalimumab, Golimumab* – fully human monoclonal Ig1 antibody
   - *Etanercept* – fusion protein of 2 p75 TNF-α receptors coupled to Fc component of human IgG1
   - *Certolizumab pego*l - pegylated humanised Fab fragment against TNF-α

The main adverse effect of anti TNF modality is the increased risk of activation of intracellular organisms lying dormant (such as Mycobacterium tuberculosis). Meticulous attention should be taken to evaluate for the presence of latent infections which may even require chemoprophylaxis. Furthermore, formation of antinuclear antibodies and double stranded DNA may rapidly occur in those especially having RA. There are also reports of reactions related to infusion such as fever, pruritus, hypotension, chest pain and dyspnoea, and of development of demyelination (radiographically) and worsening heart failure. There is a theoretical risk of increasing neoplasms although lymphomas are seen to be increased.

2) B cell depletion is employed by biologics which hit on the CD20 molecules found on the surface of cells. *Rituximab* is the main drug in this class. This is a chimeric monoclonal IgG1 antibody.
3) Abatacept is a fusion protein with the domain CTLA4 linked to human IgG1. CTLA4 found binds to CD 80 and CD 86 of antigen presenting cells.

4) Il-1, a pro inflammatory cytokine is blocked by anakinra (a recombinant IL-1Ra, endogenous antagonist). However inflammatory arthritis did not show promising results.
5) IL-6 is found abundantly in the synovium of RA knee joint. Tocilizumab is a monoclonal antibody which has shown benefit in RA.
6) Other novel agents include ustekinumab (targets IL-12 and IL-23) and secukinumab (IL-17). These are licensed for use in Psoriatic arthritis in UK

DRUGS USED IN GOUT

a) Colchicine

Colchicine is a useful drug in the management of gout. In gout, phagocytosis of uric acid crystals by neutrophils triggers the inflammatory cascade. It is presumed that colchicine inhibits assembly of microtubules and spindle formation, interfering with cell division and phagocytosis. It is a drug that brings rapid relief to the patient. Such relief does not occur with other arthritides. This phenomenon assists in diagnosis (although the corollary does not occur).

The dosage in acute gouty flare is 1 mg by mouth followed by 0.5 mg 2-3 hourly (not to exceed 6 mg in total). The course should not be repeated within 3 days. Colchicine can cause severe abdominal pain with vomiting and diarrhoea (even blood stained), neutropaenia, agranulocytosis and aplastic anaemia. Muscle paralysis and alopecia are also reported.

b) Xanthine Oxidase Inhibitors

Uric acid is formed from water soluble catabolic products of purine nucleotides, xanthine and hypoxanthine. Allopurinol inhibits the enzyme xanthine oxidase which degrades both of these metabolites. Thus, they are avidly excreted by kidneys. *Allopurinol* is therefore used in gout and in hyperuricaemia states such as neoplastic disease, cytotoxic therapy and renal disease. The usual dosage is 300 mg daily which could be doubled up in some patients.

Allopurinol should not be initiated in an acute flare as the drug can precipitate or worsen such a state. Allopurinol can cause a rare but severe disease identified as allopurinol hypersensitivity syndrome: hepatitis, eosinophilia, desquamating erythematous rash. Steven Johnson syndrome and toxic epidermal necrosis are life-threatening complications. Furthermore, allopurinol interferes with metabolism of azathioprine (by-product 6-mercaptopurine is degraded by xanthine oxidase), giving rise to myelosuppression.

c) Uricases

These drugs convert uric acid to allantoin which is a readily soluble metabolite excreted via urine. Rasburicase is a recombinant drug which rapidly corrects hyperuricaemia. However, repeated administration limits its efficacy due to antibody formation. The pegylated formulation pegloticase has less immunogenicity and has a longer half-life.

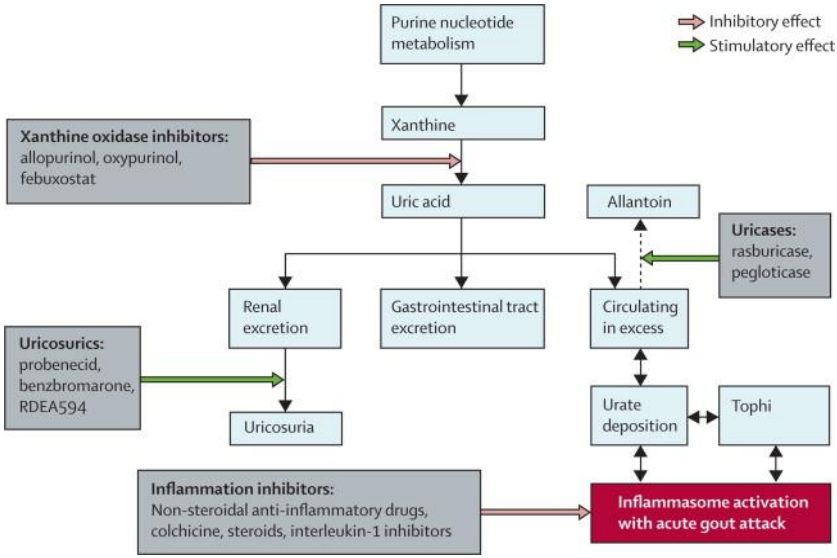

Figure 4.4 - Drugs used in gout & hyperuricaemia

d) Uricosurics

Renal excretion is enhanced by drugs such as sulfinpyrazone, probenecid and benzbromarone. Febuxostat is a new uricosuric drug which has similar or more effectivity as allopurinol.

DRUGS USED IN MANAGEMENT OF NEUROPATHIC PAIN

Amitriptyline, duloxetine, gabapentin or pregabalin are used in management of neuropathic pain (except trigeminal neuralgia). The evidence is limited for many of the drugs used in neuropathic pain. However, the positive results are seen in diabetic neuropathy, post herpetic neuralgia and post stroke and spinal injury. If the initial treatment is not effective or not tolerated, one of the other drugs may be trialled consecutively.

Older persons are at a greater risk of adverse effects. Therefore, the concept of start slows and go slow is essential with judicious monitoring of these effects. A minimum trial period of 6-8 weeks at maximum tolerated dose is necessary to assess the benefit.

Table 4.3 - Drugs used in neuropathic pain and their side effects

Tricyclic Anti-Depressants	Duloxetine	Gabapentin and pregabalin
Arrhythmia (QT prolongation) *Predominantly with amitriptyline* Anticholinergic (constipation, urinary retention, blurred vision) *Predominantly with Imipramine* Hyponatraemia (SIADH) Sedation (H2 receptor blockage) *Predominantly with imipramine, nortriptyline*	Nausea, anorexia, dyspepsia Sleep disturbances, abnormal dreams Anxiety Agitation Somnolence Dizziness Hypertension Constipation	Drowsiness Ataxia Dizziness Fatigue Cognitive disturbance Severe respiratory depression

Postural hypotension (α blockage) Decreased seizure threshold		

Tramadol should be only used if acute rescue therapy is required. Capsaicin cream is contemplated in localised neuropathic pain who wish to avoid/cannot tolerate oral treatments. The following are not advocated as first line agents.

Cannabis sativa extract
Capsaicin patches
Carbamazepine (except in trigeminal neuralgia)
Lacosamide
Lamotrigine
Levetiracetam
Morphine
Oxcarbazepine
Topiramate
Tramadol (for long-term use)
Venlafaxine
Sodium valproate

SKELETAL MUSCLE RELAXANTS

Skeletal muscles can adopt spasm in conditions such as fibromyalgia, mechanical low back and neck pain. Pharmacological agents are employed to reduce this phenomenon without impairing voluntary movements. Baclofen, diazepam and tizanidine act on the central nervous system. Baclofen is structurally related to γ-aminobutyric acid (GABA), Dantrolene acts directly on the muscle by preventing release of calcium from sarcoplasm. These drugs can cause sedation, confusion and muscular hypotonia. Chronic use of these drugs can lead to dependence and withdrawal symptoms. For skeletal muscle spasm a short-term course of 2-3 weeks is practiced.

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5. Pharmacological agents affecting musculoskeletal system

Prof. Nirmala Wijekoon

Disorders related to the musculoskeletal system are common in the elderly. Among such disorders are, those due to multiple aetiologies, medication-induced muscle damage, bone, and connective-tissue disorders. Increasing one's awareness of these disorder assists in identifying high risk patients, to use alternative medications wherever possible, to optimise the prescriptions in such a way to minimize risk to the patient, and by using other treatment alternatives.

MEDICATION-INDUCED MUSCLE PROBLEMS

Medication-induced muscle problems range from asymptomatic elevation of creatine kinase or mild myalgia, to myositis and to life-threatening rhabdomyolysis. Myalgia is presence of muscle symptoms without elevation of creatinine kinase (CPK), the enzyme that is produced by the muscles and elevated levels indicate muscle damage. Myositis is the presence of muscle symptoms with elevation of CPK. Rhabdomyolysis is a severe form of muscle damage associated with elevation of creatine kinase >10 times the upper limit of normal along with acute kidney injury. The term myopathy refers to the complete spectrum of muscle-related problems in general. The muscle symptoms that are observed in medication-induced myopathies include muscle pain, tenderness, stiffness, weakness and cramps.

A variety of mechanisms are responsible for medication-induced myopathy. Some medications directly affect muscle organelles such as mitochondria, lysosomes, and myofibrillar proteins and generate an immunologic or inflammatory reaction. Some medications alter the electrolyte or nutritional balance, which subsequently affects muscle function.

It is important for healthcare workers to recognize the medication-induced muscle problems early in their course as appropriate measures could be taken to prevent further irreversible damage. The medications

known to cause myopathy are listed in table 5.1. Different drugs produce different clinical manifestations.

Table 5.1 - Medications causing myopathy

Medication	Clinical manifestations
Amiodarone	Neuromyopathy - proximal and distal muscle weakness + distal sensory loss
Antiretroviral agents	Myalgia, myositis, rhabdomyolysis (rare)
Antipsychotics (risperidone, olanzapine, haloperidol)	Myositis, necrotizing myopathy
Glucocorticoids	Proximal myopathy – atrophy and weakness of proximal muscles
Diuretics (loop diuretics, thiazide diuretics and thiazide-like diuretics)	Hypokalaemic myopathy – muscle weakness, muscle cramps
Overuse of laxatives	
Fibrates	Myalgia, myositis, rhabdomyolysis (rare)
Quinolones	Acute severe myalgia, tendinitis, tendon rupture (risk is high in elderly)
Statins	Myalgia, myositis, rhabdomyolysis (rare)

Myopathy caused by lipid lowering drugs

Both statins and fibrates are well known to cause myopathy. The concomitant use of both these agents together, increases the risk. The stain-gemfibrozil combination is contraindicated as the risk is deemed very high. If the combination of a statin and a fibrate is clinically indicated fenofibrate should be chosen in preference. The risk factors for statin-associated muscle problems are listed in Table 2. Advanced age is a main risk factor. Drug interactions with other lipid lowering drugs and drugs which affect metabolism of statins is a frequently encountered risk factor. Drug interactions are more likely in older people as they tend to be on multiple medications. Simvastatin,

lovastatin, and atorvastatin are primarily metabolized through hepatic CYP3A4 isoenzyme and are most implicated in drug interactions. Rosuvastatin, fluvastatin and pravastatin are minimally affected by such interactions and becomes a better choice. The drugs which affect metabolism of statins are given in table 5.2.

When a patient develops statin-associated muscle symptoms, a careful evaluation and optimisation of the prescription is needed to avoid such drug interactions. In patients with rhabdomyolysis and severe myositis, the statin should be stopped immediately. The options available for managing milder disease include discontinuation and re-challenge, dose reduction, alternative dosing regimens (every-other-day, twice a week or once weekly with atorvastatin or rosuvastatin), switching to an alternative statin with lower risk of myopathy (e.g. rosuvastatin, fluvastatin, pravastatin) and combining low-dose statin with non-statin lipid lowering therapy (e.g. ezetimibe, a PCSK9 inhibitor).

Table 5.2 - Risk factors for statin-associated muscle problems

Patient factors	**Treatment factors**
• Advanced age • Female gender • Genetic factors • Asian ancestry • Untreated or undertreated hypothyroidism • Impaired renal or hepatic function • Low body mass index • Acute infection • Major surgery or trauma • Unaccustomed physical activity • Excess alcohol	• High dose • Concomitant use of a statin and a fibrate • Drug interactions affecting metabolism of statins

Table 5.3 - Drugs affecting statin metabolism

Drugs affecting statin metabolism
• Amiodarone • Antidepressants (fluoxetine, fluvoxamine, sertraline, tricyclic antidepressants) • Azole antifungals (itraconazole, ketoconazole, fluconazole) • Calcium Channel Blockers (verapamil, diltiazem) • Cyclosporine • Fibrates • HIV protease inhibitors • Macrolide antibiotics (erythromycin, clarithromycin) • Warfarin

Glucocorticoid- induced myopathy

Long term use of systemic glucocorticoids causes proximal muscle weakness and atrophy. The course is slow and it is often associated with other systemic adverse effects of long-term use of glucocorticoids. The risk is higher with higher doses and with fluorinated glucocorticoids such as dexamethasone, betamethasone and triamcinolone.

MEDICATION-INDUCED BONE PROBLEMS

Osteoporosis and fractures are common problems among older people. Many medications that are commonly prescribed have harmful effects on bone homeostasis, leading to decreased bone mineral density and increased risk of fractures.

Glucocorticoids

Long term use of systemic glucocorticoids is the third most common cause of osteoporosis. Postmenopausal women and older men are at the highest risk of glucocorticoid-induced osteoporosis. The other risk factors include low body mass, smoking, excess alcohol intake, hip fracture in parents, and intravenous pulse steroids. Risk of bone loss is greatest within the first 6 to 12 months of treatment and it is dose-dependent. The daily dose of the glucocorticoid predicts fracture risk more than the cumulative dose. Fracture risk is increased fivefold with prednisolone doses >7.5mg/day. Even 2.5 mg/day has been found to

be associated with an increased risk of spine fracture. The fracture risk returns to baseline 2 years following discontinuation of glucocorticoid therapy. Multiple mechanisms are responsible for corticosteroid-induced osteoporosis which include increased osteoblast apoptosis, increased bone resorption secondary to decreased levels of gonadotropins and calcium deficiency secondary to decreased gastrointestinal absorption and increased renal excretion.

In patients who are on long term glucocorticoids, precautions need to be taken to minimize the risk of osteoporosis and the elderly need special attention with regard to these precautions. The minimal effective dose for the minimum required duration is the golden rule. Prophylactic therapy with bisphosphonates is indicated for adults aged ≥40 years with high or moderate risk of fracture. Oral bisphosphonates, alendronate and risedronate are first-line options. Intravenous zoledronic acid or teriparatide are alternatives that can be used in patients intolerant of oral bisphosphonates. Optimizing calcium intake (1,000–1,200 mg/day) and vitamin D intake (600–800 IU/day) and life style modifications are important in all adults irrespective of age and fracture risk.

Antiepileptics

The anti-epileptics most commonly associated with osteoporosis include phenytoin, phenobarbital, carbamazepine, and primidone. One mechanism responsible for osteoporosis include rapid metabolism of vitamin D due to induction of hepatic cytochrome enzymes resulting in reduced absorption of calcium which leads to secondary hyperparathyroidism and increased bone turnover. Phenytoin seems to have direct inhibitory effects on osteoblasts too. Even though sodium valproate does not cause induction of cytochrome enzymes, it increases fracture risk secondary to hypophosphataemia. Newer antiepileptic drugs such as topiramate and lamotrigine have also been associated with increased fractures. Levetiracetam has not been associated with fractures. However, long term studies are lacking. Supplementation of calcium and vitamin D is important in reducing the osteoporotic fractures associated with the use of antiepileptics. The daily requirement

of vitamin D is higher than usual (2000-4000 units/day) in those who are on enzyme-inducing antiepileptics.

Other medications

Heparin - Long term, high dose therapy with unfractionated heparin causes osteoporosis. Heparin causes increased bone resorption by stimulating osteoclasts and suppressing osteoblast activity. Osteoporosis is not an adverse effect of low molecular weight heparin (LMWH).

Progestins - Medroxyprogesterone acetate (MPA) has been linked to osteoporosis recently. It is used in some hormone replacement products.

Proton pump inhibitors - Long term use of proton pump inhibitors is associated with a modest increase in osteoporotic fracture risk.

Aluminium-containing antacids- They increase the risk by binding calcium in the gastrointestinal tract and reducing calcium absorption.

Loop diuretics – They tend to decrease the bone mass by increasing renal calcium excretion.

Over supplementation of thyroxine – This is another cause for medication-induced osteoporosis. Regular monitoring of thyroid function tests is especially important in elderly as the thyroxine dose requirement may decrease with age.

Selective serotonin reuptake inhibitors (SSRI) and serotonin and norepinephrine reuptake inhibitors (SNRI) - These are widely used drug classes which enhance bone loss and increase fracture risk.

Methotrexate - It has increased the risk of osteoporosis when used in very high doses, such as in the treatment of malignancies.

Aromatase inhibitors (e.g. letrozole, anastrozole, and exemestane) - They are prescribed as adjuvant hormone therapy for oestrogen-receptor positive breast cancer in postmenopausal women. They inhibit the peripheral conversion of androgens to oestrogens. As a result, the oestrogen levels fall below what is normally found in postmenopausal women and it accelerates bone loss.

Androgen-deprivation therapy - It is used for metastatic prostate cancer. It establishes a hypogonadal state and increases bone loss.

Calcineurin inhibitors (cyclosporine and tacrolimus) – These immunosuppressants have been associated with bone loss and increased fracture.

Skeletal complications of long-term bisphosphonate therapy

Even though the bisphosphonates are effective in prevention and treatment of osteoporosis, prolonged treatment with bisphosphonates leads to other complications of the skeletal system.

Atypical femoral fractures have been reported rarely with long term bisphosphonate treatment. Because of this risk, the need to continue bisphosphonate treatment should be re-evaluated after 5 years of use. Patients should be advised to report any thigh, hip, or groin pain during treatment with a bisphosphonate.

Another skeletal complication of bisphosphonate use is osteonecrosis of the jaw. The risk is particularly high with intravenous bisphosphonate use for the treatment of cancer. Patients should maintain good oral hygiene, receive routine dental check-ups, and report any oral symptoms such as dental mobility, pain, swelling, non-healing ulcers or discharge.

Benign idiopathic osteonecrosis of the external auditory canal has been reported very rarely with bisphosphonate treatment, mainly in patients receiving long-term treatment (2 years or longer). Risk factors for developing osteonecrosis of the external auditory canal include steroid use, chemotherapy, infection, an ear operation, or cotton bud use. Patients should be advised to report any ear pain, discharge from the ear, or auditory symptoms.

MEDICATION-INDUCED ARTHRITIC AND CONNECTIVE TISSUE DISORDERS

There are many medications which are known to cause arthritic and connective tissue disorders as an adverse effect. These medications can

be categorized into different groups based on the underlying mechanisms. They are described in table 5.4.

Table 5.4 - Medications causing arthritic and connective tissue disorders

Mechanism	Medications
Arthralgia as an isolated drug side effect	bisphosphonates, calcitonin, quinolones, cefixime, macrolides, rifaximin, pyrazinamide, nitrofurantoin, antivirals, fluticasone, gabapentin, lamotrigine, vigabatrin, aminosalicylates (mesalazine, sulfasalazine), selective serotonin reuptake inhibitors (SSRIs), mirtazapine, olanzapine, rasagiline, selegiline, melatonin, carbimazole, propylthiouracil, conjugated oestrogens, mycofenolate mofetil, cytotoxic medications, granulocyte-colony stimulating factor, aromatase inhibitors, interferon, monoclonal antibodies, erythropoietin, desferrioxamine, retinoids, statins, ezetimibe, beta blockers, loop diuretics, amiloride, methyl dopa, verapamil, angiotensin converting enzyme inhibitors (ACEIs),
Precipitation of a transient flu-like syndrome including arthralgia	interferon, monoclonal antibodies, parenteral bisphosphonates, cytotoxic chemotherapy, intravenous iron
Serum sickness and similar drug reactions causing arthralgia /arthritis	beta-lactam antibiotics (penicillins, cephalosporins, carbapenems), ciprofloxacin, sulphonamides, streptomycin, metronidazole, streptokinase, hydralazine, halothane, iron dextran, carbimazole, propylthiouracil, carbamazepine, azathioprine, monoclonal antibodies
Medication-induced lupus	hydralazine, penicillamine, chlorpromazine, methyldopa, isoniazid, quinidine, minocycline, beta blockers
Medication-induced gout	low dose aspirin, diuretics, cytotoxic chemotherapy, pyrazinamide, ethambutol, cyclosporine, tacrolimus, fructose

Medication-induced hyperuricaemia and gout

Gout is a common inflammatory arthritis in adults. It is a complication of hyperuricaemia. Hyperuricaemia is a well-recognized adverse effect of treatment with several medications.

In humans, the end product of purine metabolism is uric acid. Uric acid is primarily eliminated by the kidney. Almost all uric acid is filtered from glomeruli and post-glomerular reabsorption and secretion regulate the amount of uric acid excretion. Uric acid reabsorption and secretion occurs in the proximal tubule, and under normal physiological circumstances approximately 90% is reabsorbed into blood.

Medications commonly raise serum uric acid level by increasing uric acid reabsorption and/or by decreasing uric acid secretion by the kidney. Some drugs are known to increase uric acid production. The medications causing hyperuricaemia and gout are described in table 5.5. Maintenance of adequate hydration and routine monitoring of uric acid level should be done when drugs known to induce hyperuricaemia are prescribed. They also need monitoring for early symptoms of gout. Allopurinol is indicated for prevention of cytotoxic chemotherapy induced hyperuricaemia and gout.

Table 5.5 - Medications causing hyperuricaemia

Medication	**Mechanism of hyperuricaemia**
Anti-tubercular drugs: pyrazinamide ethambutol	Increased reabsorption Decreased secretion Decreased fractional excretion
Aspirin (low dose aspirin)	Increased reabsorption Decreased secretion
Cytotoxic chemotherapy	Massive destruction of tumour cells and increased production of uric acid
Diuretics (loop diuretics, thiazide diuretics and thiazide-like diuretics	Increased reabsorption
Fructose	Increased nucleotide turnover and increased production of uric acid Increased reabsorption
Immunosuppressants: cyclosporine	Increased reabsorption

tacrolimus	Decreased glomerular filtration rate secondary to afferent arteriolar vasoconstriction Reduced excretion

Anti-tubercular drugs

More than 80% of uric acid clearance is reduced by therapeutic doses of pyrazinamide. It causes hyperuricaemia and precipitate acute attacks of gout. Ethambutol reduces renal clearance of uric acid to a lesser extent.

Aspirin

In low dosages (up to 300mg once daily), aspirin reduces uric acid excretion, whereas higher doses of aspirin are uricosuric.

Cytotoxic Chemotherapy

Hyperuricaemia caused by cytotoxic chemotherapy is the most serious type of medication-induced hyperuricaemia. It occurs as a part of the tumour lysis syndrome. Hyperuricaemia typically appears 48-72 hours after chemotherapy.

Diuretics

Gout is more common with loop diuretics than with thiazide diuretics. Hyperuricaemia may occur within a few days after the initiation of treatment. It is dose dependent. Uric acid level returns to the baseline a few months after stopping the diuretic.

Fructose

Fructose is a glucose substitute used in total parenteral nutrition. Intravenous fructose administration over several days is associated with hyperuricaemia.

In conclusion, the iatrogenic musculoskeletal disorders are an important problem among older people. Healthcare workers need to be aware of the medication-induced musculoskeletal disorders in order to prevent

them and for early detection and treatment which in turn helps to improve the quality of life in elderly.

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6. Evaluation of Arthritis in Older Adults

Dr. Duminda Munidasa, Dr. Kalum Deshapriya, Dr. Rasika Munasinghe

INTRODUCTION

The ability to be mobile is a key factor to maintain physical and mental health as well as independency in elderly. Joint pathology may lead to reduced mobility, increased risk of falls, low energy, dependency and depression in geriatric population. Chronic pain itself is strongly associated with psychological distress and fatigue. Treatment with medications for arthritis of older adults with multimorbidity, decreased organ functions and effect on their life (even due to a minor adverse effect) is a challenge. Since there is a worldwide increase of geriatric population, arthritis places an enormous burden on the individual and on society. Therefore, the importance of correctly diagnosing and managing joint pain in the elderly is paramount.

AETIOLOGY

There are many potential causes of joint pain in older patients. As in all age groups, different aetiological categories such as degeneration, trauma, neoplasms, infections, metabolic and autoimmune diseases exist. Specific symptoms and signs elicited in the history and examination can indicate the possible aetiology of the patient's arthritis (table 6.1).

TABLE 6.1 - Aetiological groups for causes of arthritis and cardinal features.

Aetiological groups	**Cardinal features elicited in history**	**Diseases in elderly**
Degenerative	Worse in evening and with activity Gel phenomenon *(Synovial fluid becomes thickened making movement difficult)*	Osteoarthritis Spondylosis
Metabolic	Acute onset Severe manifestations	Crystal arthritis

			(Gout, Pseudogout, Milwaukee)
Infections	Acute	Acute onset Severe manifestations Constitutional features	Septic arthritis Viral arthritis
	Chronic	Insidious onset	Tuberculosis
Autoimmune		Early morning stiffness Worse in morning and with rest	Rheumatoid arthritis Reactive arthritis Polymyalgia rheumatica Psoriatic arthritis
Neoplasms		Night pain	Primary synovial neoplasms Secondary deposits
Trauma		History of trauma	Fractures Intra articular tissue injuries

The pattern of joint involvement as of area, type and number of peripheral joints will also indicate the aetiology of the arthritis (table 6.2). Classical clinical presentation of inflammatory arthritis may not be apparent in elderly but elucidation of these features is important in the diagnosis. Symmetricity of joint involvement and the upper limb predominance may not be apparent in some patients.

TABLE 6.2 - Arthritic diseases and their typical presentations.

Differentiation of joint involvement	**Type of joints**	**Diseases in elderly**
Areas of joints	Axial	Spondylosis Secondary deposits
	Peripheral	All below
Number of peripheral joints	Monoarthritis	Osteoarthritis Haemarthrosis Crystal Induced arthritis Septic arthritis Reactive arthritis Intra articular injury

		Oligoarthritis	Reactive arthritis Psoriatic arthritis
		Polyarthritis	Rheumatoid arthritis Palindromic rheumatism Nodal osteoarthritis Paraneoplastic arthritis Psoriatic arthritis Non suppurative arthritis Atypical polyarticular gout Remitting seronegative symmetrical synovitis with pitting oedema (RS3PE)
Predominant Type of peripheral joints	I	Small joints	Nodular osteoarthritis Rheumatoid arthritis Crystal arthritis Psoriatic arthritis
		Large joints	Osteoarthritis Haemarthrosis Crystal arthritis Septic arthritis Reactive arthritis Intra articular injury
	II	Upper limb	Rheumatoid arthritis Nodular osteoarthritis
		Lower limb	Osteoarthritis Crystal arthritis Reactive arthritis

In older adults, the most likely aetiology for arthritis is osteoarthritis. However, differential diagnosis includes conditions such as septic arthritis, crystal arthritis and inflammatory arthritis, which lead to severe morbidity, if not diagnosed early and treated.

DIAGNOSIS

Arriving at a diagnosis should be done in a methodical manner.

History taking

History taking is the first step of the diagnosis. It is important to elicit the differentiating features of aetiological groups mentioned in table 1, when taking the history of the patient. Presentation of elderly onset rheumatoid arthritis will be different from young adults.

In general, the history taking should include the onset and duration of arthritis, chronology of progression, characteristics of the pain, aggravating or relieving factors, previous episodes, diet, alcohol consumption, and exercise. In addition, it should include any history of trauma, traveling, sexual exposure, past disease and drug histories, and family histories of arthritis, immunologic diseases or urinary stones. Each of these can have a bearing on the diagnosis and the management plan. Especially in the geriatric population, assessment of daily activities and social circumstances in the history will help in planning further management.

Physical examination

Physical examination will establish the presence of arthritis by eliciting at least one of swelling, tenderness or pain on range of movement in a painful joint. A joint need to be examined systematically in the order of inspection, movement and specific manoeuvres for the particular joint. A general and system physical examination looking for extra articular features of different arthritic illnesses will help in establishing a diagnosis.

Laboratory tests

Laboratory tests can be useful in evaluation of an older adult with arthritis, for confirmation of the suspected diagnosis, baseline values of body functions before initiation of treatment and follow-up monitoring. Requests for laboratory tests should be based on the differential diagnosis you have arrived after evaluating the patient through the history and examination. Unnecessary investigations will cause a physical, mental and financial burden to these patients. Definitive diagnosis of inflammatory arthritis occurring on a primary osteoarthritic joint as well as differentiation from primary osteoarthritis flare up from an inflammatory arthritis will be difficult by physical examination alone,

especially in patients with mono or oligo arthritis. They will benefit from laboratory tests including synovial fluid analysis.

It is also well established that the older adults have a higher incidence of false positive autoantibodies such as rheumatoid factor and antinuclear antibodies. Hence testing them as a routine procedure will result in inappropriate management.

Synovial fluid analysis

Synovial fluid analysis will be essential in the diagnosis of septic arthritis and crystal induced arthritis. In an elderly patient with acute onset painful joint, without a history of recent trauma it is essential to send the joint fluid for analysis including culture for organisms and crystal detection. An arthrocentesis can be performed on a swollen joint, as long as there are no contraindications, such as overlying infection or severe dermatitis. Synovial fluid will be helpful to differentiate aetiology of the joint swelling (table 6.3). The appearance, viscosity, protein, glucose, white and red cell counts, and PMN percentages under light microscopy differ among these aetiological groups.

TABLE 6.3 - Differentiating the aetiology of arthritis with joint fluid analysis

	Normal	**Degenerative**	**Inflammatory**	**Septic**
Appearance	clear	clear	opaque	opaque
Viscosity	high	high	Low	low
White cells/mm^3	<200 *(<500)*	200-10,000 *(500-5,000)*	5,000-75,000 *(5,000-50,000)*	>50,000 *(>50,000)*
Percentage of neutrophils	<25%	<50%	>50%	>75%

Imaging studies

Imaging studies of joints by plain x-ray, ultra sound scan, computed tomography and magnetic resonance imaging are helpful for

evaluation of arthritis in geriatric patients as in others. But as for laboratory investigations, these also need to be done after proper clinical evaluation. Imaging is indicated only if clinical diagnosis needs further confirmation, if helpful in definitive diagnosis in presence of a differential diagnosis, ongoing disease activity in spite of treatment or if surgical interventions are needed for treatment. Most elderly people will have osteoarthritic changes in their joints. However, this may not be the cause of arthritis. Hence interpretation of imaging studies in elderly need to done with care.

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7. Osteoarthritis

Dr. Aruna Caldera

INTRODUCTION

Osteoarthritis (OA) is the commonest form of arthritis which could range from being asymptomatic (with incidental findings on clinical or radiologic examination) to a progressive disabling disorder eventually culminating in joint failure. It is essentially a disease which starts in late middle age or elderly. The prevalence of OA varies according to the definition of OA, the specific joint(s) under study, and the characteristics of the study population. The WHO Scientific Group on Rheumatic Diseases estimates that 10% of the world's population who are 60 years or older have significant clinical problems that can be attributed to OA.

PATHOGENESIS

While previously characterized as a disease of progressive articular cartilage degradation, OA pathophysiology now is known to involve all of the tissues that form the synovial joint (subchondral and metaphyseal bone, synovium, ligaments, joint capsules, and the muscles acting across the joint). Subchondral bone remodelling, osteophyte formation, synovial inflammation, ligamentous laxity, and the weakening of periarticular muscles exemplify several joint structure alterations observed.

The trigger of OA is unclear; however, it may begin with tissue damage from mechanical injury, infiltration of inflammatory mediators from the synovium into the cartilage, or defects in cartilage metabolism/homeostasis. Chondrocytes attempt to repair cartilage damage/degradation by increasing the production of extracellular matrix macromolecules. As degeneration continues, catabolic mechanisms overpower the anabolic capabilities of chondrocytes and the homeostatic balance is tipped over resulting in progressed cartilage breakdown.

RISK FACTORS

Risk factors for OA include

- Age
- Obesity
- Previous trauma
- Genetics
- Reduced levels of sex hormones
- Muscle weakness
- Repetitive joint use
- Infection
- Crystal deposition
- Acromegaly
- Pre-existing inflammatory arthritis
- Heritable metabolic causes (e.g., alkaptonuria, haemochromatosis, Wilson disease)
- Haemoglobinopathies (e.g., sickle cell disease and thalassaemia)
- Neuropathic disorders leading to Charcot joint (e.g., syringomyelia, tabes dorsalis, and diabetes mellitus)
- Underlying morphologic risk factors (e.g., congenital hip dislocation and slipped femoral capital epiphysis)
- Disorders of bone (e.g., Paget disease and avascular necrosis)
- Previous surgical procedures (e.g., meniscectomy).

CLINICAL FEATURES

The common symptoms and signs of osteoarthritis includes joint pain and tenderness, limitation of range of movements, bony swelling, joint deformity and joint instability.

Pain is the most frequent symptom of OA with a usage related pain that settles down with rest. The pain is usually worse in late afternoon, early evening and as well as in early mornings. It progresses through various stages though not universal. Initially, the pain is a sharp pain which is brought on by mechanical insult, settling with joint rest. Later pain can become more constant, affecting activities of daily living. Ultimately there is progression to a constant dull/aching pain punctuated by

episodes of often unpredictable, intense, exhausting pain that results in severe limitations in function. Joint pains can be associated with morning stiffness, which usually lasts less than half an hour.

Joint line tenderness is also a common finding in OA. There is limitation of range of motion mainly resulting from marginal osteophytes and capsular thickening. Synovial hyperplasia and effusion may also play a role in some patients. Bony swelling reflects remodelling of the bone and cartilage on either side of the joint and marginal osteophytes formation. Joint deformity is a sign of joint damage (commonest example is a genu varum). Joint instability is present more as a result of feeling of apprehension and lack of confidence to weight-bear rather than literally giving way.

OA is categorised in to single or multi-joint OA and generalized OA.

a) ***Single or multiple-joint OA*** has a predilection for the knees, hips, finger interphalangeal joints, first carpometacarpal (CMC) joints, first metatarsophalangeal (MTP) joints, and facet joints of the lower cervical and lower lumbar spine. OA is less common in elbow, wrist, shoulder and ankle and other differentials should be considered first before making a diagnosis of OA of theme.

b) ***Generalized OA*** implies a polyarticular subset of OA typically involving the distal interphalangeal (DIP) joints, thumb bases (first CMC joints and trapezio-scaphoid joints), first MTP joints, lower cervical and lumbar facet joints, knees, and hips. The clinical marker for generalized OA is the presence of multiple Heberden nodes (bony swelling) of the DIP joints. Heberden nodes are often accompanied by less well-defined swelling of the proximal interphalangeal (PIP) joints referred to as Bouchard nodes. Generalized OA may occur in the absence of nodes, so called non-nodal generalized OA, which is more common in men (compared with nodal generalized OA, which is more common in women).

DIAGNOSIS

The diagnosis of OA is clinical though imaging has a role to play especially in advanced cases. Conventional radiography is the most

widely used imaging modality in OA. It allows for detection of characteristic features of OA including marginal osteophytes, joint space narrowing, subchondral sclerosis, and cysts. Clinicians should be mindful that x-ray changes may not correlate clinically in all patients, especially in early disease. Radiographic OA is a common incidental asymptomatic finding in older people. Radiographic examination may be used to support a diagnosis of OA but is not a routine test to consider explain clinical symptoms. Patients with a robust diagnosis of OA on clinical grounds may have normal plain radiographs, and vice versa.

Synovial fluid from OA joints is usually noninflammatory or mildly inflammatory with less than 2000 white blood cells/mm^3 predominantly mononuclear cells. Calcium pyrophosphate (CPP) crystals may be present in a portion of unselected OA patients. Most patients with OA and CPP deposition (CPPD) are older than 60 years, and common target sites are the knees, radiocarpal joints, second and third MCP joints, shoulder, and elbow joints. Some may present with acute episodes of crystal arthritis, whereas some may develop chronic crystal arthritis with a similar joint involvement pattern to rheumatoid arthritis.

Additional laboratory testing may include an erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP). Inflammatory markers are normal in OA and may be useful in excluding other diagnoses. Although rheumatoid factor is negative in patients with OA, a cohort of patients may have a marginally positive value which is age related and not due to joint disease related

VARIATIONS IN PRESENTATION ACCORDING TO THE JOINT/ JOINTS INVOLVED

Many of the characteristic clinical manifestations of OA are related to the involvement of particular joints.

In hand OA the symptoms are usually bilateral, and joint involvement is usually approximately symmetrical. Distal interphalangeal (DIP) joints, thumb bases, proximal interphalangeal (PIP) joints, and second and third metacarpophalangeal (MCP) joints are affected. Thumb-base OA

generally affects older postmenopausal women. There may be radial subluxation and adduction at the thumb base, giving it a swollen squared appearance. Nodal osteoarthritis characterised by Heberden's (DIP joints) and Bouchard's (PIP joints) (Figure 7.1).

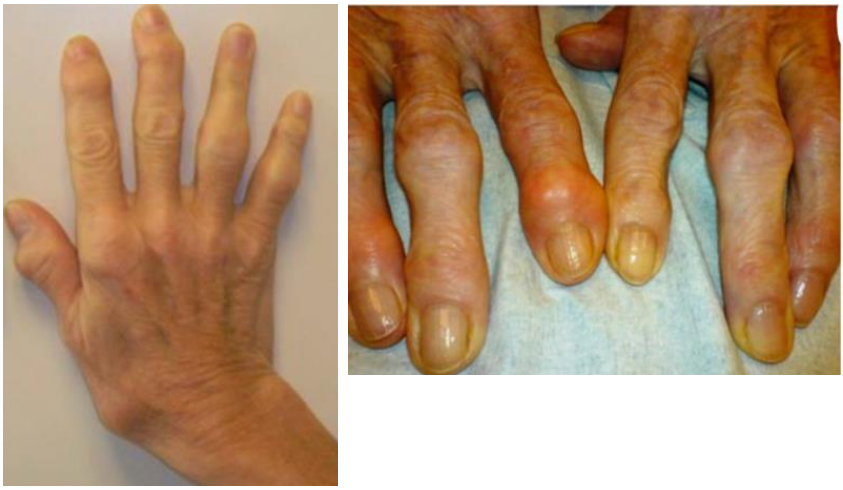

Figure 7.1 - a) Squared hand, b) Heberden and Bourchard joints

Erosive OA is an uncommon and particularly aggressive subset of hand OA. It has a synchronous polyarticular onset with marked joint inflammation. Erosive OA targets the interphalangeal joints (the DIP joints more frequently than PIP joints) and usually spares the thumb bases and MCP joints. These changes give rise to gull wing appearance. The alignment abnormalities of the thumb may cause a hitchiker's thumb (Figure 7.2).

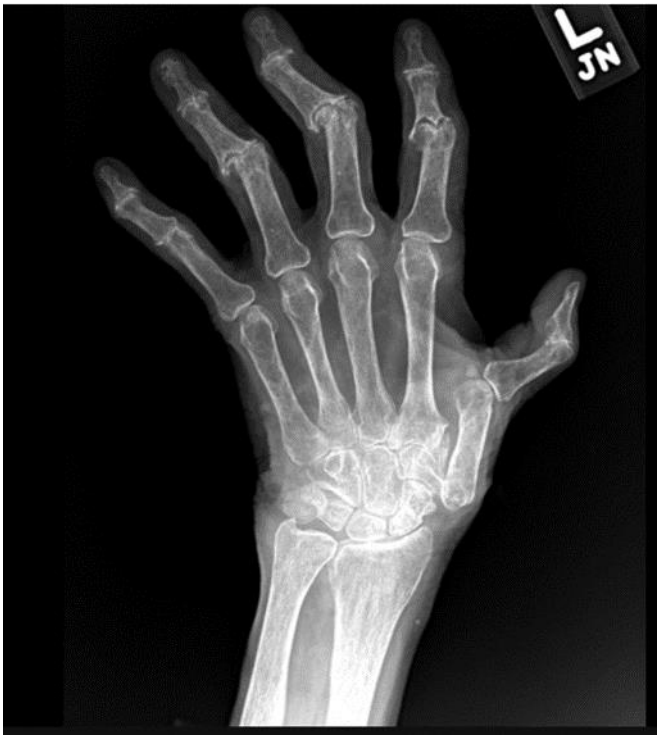

Figure 7.2 - Radiograph of erosive changes of hand in osteoarthritis

Knee OA is usually bilateral. The patellofemoral joint and/or the medial tibiofemoral joint are most affected, and isolated lateral tibiofemoral joint OA is relatively rare. Pain from knee OA is exacerbated by prolonged sitting, standing up from a low chair, and climbing stairs or inclines. Knee OA usually does not cause posterior knee pain unless there is a complicating popliteal (Baker's) cyst. Typical examination findings would be, joint tenderness, limited flexion, swelling, genu varum deformity and quadriceps and hip adductor weakness (Figure 7.3).

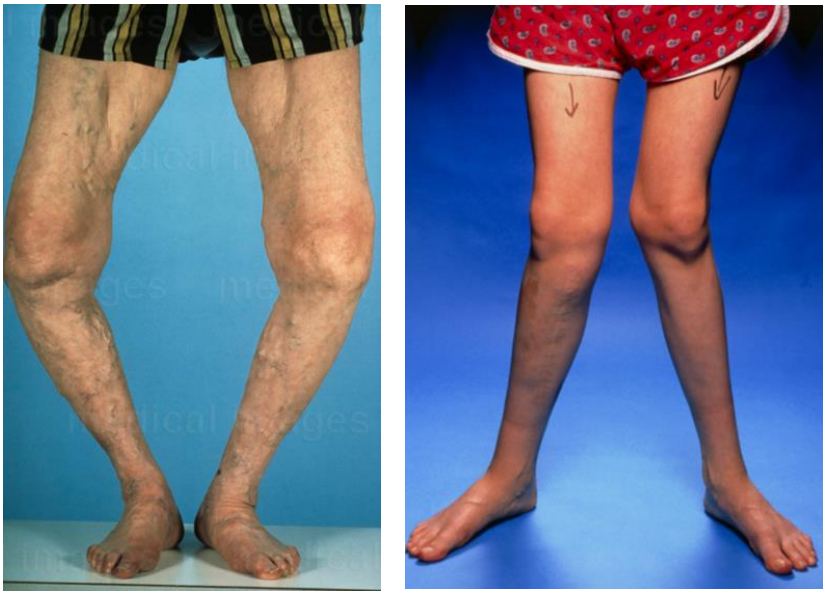

Figure 7.3 - a) Genu valgum, 3b) Genu varus

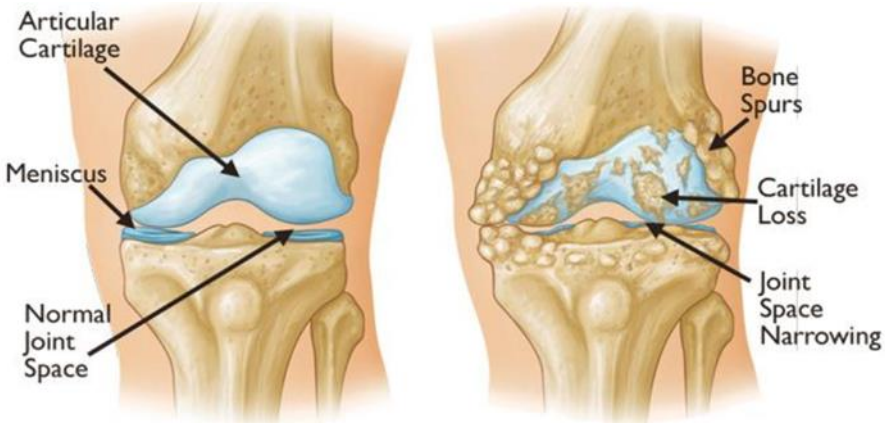

Figure 7.4 - Erosive changes of the knee in osteoarthritis

Hip OA presents with anterior groin pain, aching, stiffness, and restricted movement. The pain is exacerbated particularly by rising from a seated position and during the initial phases of ambulation. Unlike knee OA, hip OA is frequently unilateral. Internal rotation with the hip flexed is frequently the earliest and most affected movement. Wasting of thigh muscles, a positive Trendelenburg test and shortening of the affected extremity are findings of advanced disease.

Facet joint OA usually coexists with intervertebral disc degeneration. Lumbar facet joint OA leads to localized lumbar pain, which may radiate unilaterally or bilaterally to the buttocks, groin, and thighs. Symptoms are typically worse in the morning and during periods of activity, and are increased by stress, exercise, lumbar spine extension, rotatory motions, and when standing or sitting. Similarly, cervical facet joint OA may present with ipsilateral neck pain, which does not radiate beyond the shoulder and is aggravated by neck rotation or lateral flexion

The first metatarsophalangeal (MTP) joint OA is usually bilateral, and when symptomatic, leads to localized big-toe pain on standing and during ambulation. Bony enlargement of the first MTP joint is a common finding. Hallux valgus deformity (when the distal end of big toe points towards the midline of the foot), hallux rigidus (or restricted flexion, and extension at the first MTP joint), and cross-over toes are common deformities. Bony enlargement at the first MTP joint and hallux valgus frequently lead to the development of a complicating bursa with additional fibrous tissue reaction on the medial aspect of the first MTP joint (bunion). (Figure 7.5)

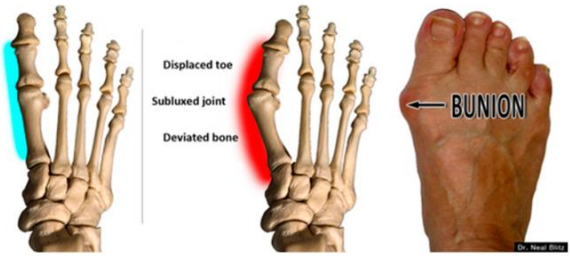

Figure 7.5 - Osteoarthritis changes of the foot

Glenohumeral OA predominates in older adults aged over 70 and less common. It causes anterior shoulder and upper arm usage related pain. Examination findings may include local glenohumeral anterior joint line tenderness, pain and restriction, mainly of external rotation and abduction (but eventually all other movements), coarse crepitus felt maximally over the anterior joint line and weakness and eventual wasting of all muscles acting over the joint (including the deltoid, and rotator cuff muscles).

Differential diagnosis for OA includes rheumatoid arthritis, psoriatic arthritis, crystalline arthritis, haemochromatosis and infectious arthritis.

Peripheral joint OA may be diagnosed confidently on clinical grounds alone if the following are present:

- Persistent usage-related joint pain in one or few joints
- Age more than 45 years
- Morning stiffness less than 30 minutes

Additional testing will be required in following situations

- Younger individuals with joint symptoms/signs of OA
- Presence of atypical symptoms and signs such as an unusual site of involvement, symptoms and signs of joint inflammation, marked rest and/or night pain, and rapidly progressive pain
- Presence of weight loss or constitutional symptoms

MANAGEMENT

A holistic assessment is made prior to setting up a management plan for a patient with OA. Patients should be fully informed about the disease and possible outcomes, specially emphasising on the fact that the adherence to management plan has a considerable say in outcome. Setting goals helps the informed patient identify current issues, set priorities, and focus on specific changes. Long-term goals should be broken up into short-term achievable steps (e.g. start walking for 10 minutes on three days of the week to ultimately be able to walk for 30 minutes on three days of the week in three months' time) and achievements should be positively reinforced.

Periodic clinical assessments should be performed regularly (ideally every three months) to assess the effects of treatment on symptoms, functionality, and status, as well as quantify objective changes in metrics related to interventions such as weight and muscle strength.

The clinicians could use various assessment tools in patients with OA such as Knee Injury and Osteoarthritis Outcome Score (KOOS), Hip Disability and Osteoarthritis Outcome Score (HOOS), the six-minute walk test, timed up-and-go test the Western Ontario and McMaster Universities (WOMAC) questionnaire, Australian/Canadian Hand OA Index (AUSCAN) pain subscale, AUSCAN function subscale, Functional Index for Hand Osteoarthritis (FIHOA) and physical tests measuring grip and pinch strength.

The goals of OA management are to minimize pain, optimize function, and beneficially modify the process of joint damage. The primary aim of clinicians should include targeting modifiable risk factors. Although there are no approved disease-modifying OA drugs, a wide selection of interventions is available to address pain and function. Due to the modest effects of the individual treatment options, a combination of therapeutic approaches is commonly used in practice and should prioritize therapies that are safer before considering drugs that can potentially cause harm.

Management should also be individualized and target modifiable factors contributing to pain, particularly presence of joint malalignment,

muscle weakness, overweight and obesity, and concurrent depression. The number of joints involved, presence of articular versus periarticular pain, and the degree of movement restriction and functional impairment should also guide the therapeutic plan.

Nonpharmacological interventions are the mainstay of OA management and should be tried first followed by or in concert with medications to relieve pain when necessary. These include weight management and exercise, application of braces and foot orthoses for patients suitable to these interventions, education, and use of assistive devices when required.

a) Exercise has effects of similar magnitude on pain and function compared with NSAIDs. A combination of aerobic and strengthening exercises is usually indicated to address the whole spectrum of disability associated with OA, but optimal prescription should be individualized.

b) Loss of at least 10% of body weight through a combination of diet and exercises has been associated with a 50% reduction in pain scores in overweight/obese patients with knee OA after 18 months.

c) Walking aids and knee braces for patients with malalignment (knee OA) may improve pain. This should be considered as adjunctive treatments. In addition, splints are particularly recommended for the treatment of OA at the base of the thumb.

Pharmacological agents are reserved for patients with symptomatic OA who have not responded adequately to initial nonpharmacologic measures or concomitantly with these interventions for those with more severe symptoms.

Pharmacological therapy should only be used during periods when symptoms are present, since none of the interventions have been shown to be disease-modifying. The main medications used in the pharmacologic management of OA include oral and topical NSAIDs, with topical capsaicin, and intraarticular glucocorticoids. In patients with one or a few joints affected, especially knee and/or hand OA, topical NSAIDs have similar efficacy compared with oral NSAIDs and have a

better safety profile. Oral NSAIDs are used in patients with inadequate symptom relief from topical NSAIDs. Lowest dose of the medication should be used. The use of NSAIDs in most patients is limited by the increased risk of serious gastrointestinal, cardiovascular, and renal complications.

Topical capsaicin is a treatment option when one or few joints are involved and other interventions are ineffective or contraindicated. However, its' use may be limited by common local side effects.

Intraarticular glucocorticoid injections are frequently used for patients who have responded poorly to oral and local NSAIDs though these injections are suspected to cause deleterious effects on the hyaline cartilage which may accelerate OA progression.

Paracetamol is not frequently used as a pain killer in these patients given the minimal or no effect on pain in patients with OA. Opioids can only be used for short-term use in patients with severe and disabling symptoms in whom other interventions have failed or are not appropriate.

The benefit of intraarticular hyaluronic acid is also controversial for knee and hip OA, and most evidence demonstrates only a small superiority over intraarticular placebo. Nutritional supplements such as glucosamine, chondroitin, vitamin D, and fish oil lack clear evidence of benefits and are not encouraged. But some supplements which gives symptomatic relief with no evidence of increased risk of side effects compared with placebo can be prescribed. Most of these supplements have demonstrated benefits only in a limited number of small trials and the quality of evidence was variable. The landmark GAIT trial (Glucosamine/Chondroitin Arthritis Intervention) demonstrated that combination of glucosamine and chondroitin sulphate may have some efficacy in patients with moderate-to-severe symptoms.

Surgical treatment is dominated by total joint replacement, which is highly effective in patients with advanced OA. Arthroscopic surgery for knee OA involving partial meniscectomy, debridement, or both has no clinically significant long-term benefits over conservative treatment or placebo surgery.

Platelet rich plasma (PRP) injections in patients with knee OA found a significant difference in pain scores up to 12 months in the PRP-treated groups in studies; but no studies have examined the structural effects of PRP in OA joints. Since there is lack of standardisation of the preparations of PRP amongst the trials, it is yet to be validated as a recommended treatment in OA.

Other alternative therapies that have been tried in the treatment of OA include acupuncture, traditional Chinese medicine, and transcutaneous nerve stimulation.

The placebo effect has a substantial implication in the treatment of patients with OA, often overcoming the actual effect sizes of individual treatments.

With this in mind and in line with the prerequisite of non-maleficence, it is likely that patients with OA pain would benefit from clinicians who are able to optimize and use the placebo effect in clinical practice in the favour of their patients.

SUMMARY

OA is the commonest form of arthritis among elderly population. It can affect many joints, knee being the commonest one. Management is multimodal though specific disease modifying agents are not available unlike for other forms of arthritis. OA can cause significant reduction in quality of life and simple measures like weight reduction is likely to have a more significant impact on the outcome than any forms of medications used in significant proportion of patients.

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8. Inflammatory arthritis

Dr. Sachithra Illangantilaka

Although degenerative arthritis predominates in the geriatric population, physiological and immune system dysregulations commonly seen in the older age groups predispose them to inflammatory arthritis than the younger population. These immune system variations in the two age groups, lead to differences between the older adults' disease and younger onset disease. Increased life expectancy, risk transition and improved quality of care also increases the number of patients living with chronic inflammatory rheumatic diseases.

Table 8.1 - Common causes for inflammatory arthritis in the older adults

Rheumatoid arthritis
Psoriatic arthritis
Polymyalgia rheumatica
Flare of osteoarthritis
Crystal arthritis (Gout, Pseudo gout)
Remitting seronegative symmetrical synovitis with pitting oedema (RS3PE)
Paraneoplastic syndrome
Systemic vasculitis
Hypertrophic osteoarthropathy
Septic arthritis
Reactive arthritis

Approximately one third of patients with Rheumatoid arthritis (RA) are above the age of 60 years which is a non-negligible amount. Compared to this, only 5% of the spondyloarthropathy (SpA) patients are more than 50 years of age. It should also be borne in mind that paraneoplastic arthritic manifestations are common in the seniors.

Management of these patients however becomes a challenge with the added complications of co-morbidities and polypharmacy. Yet it

remains an area clinician must be vigilant with the aging of the world population.

This article will deal with two of the commonest inflammatory arthritis, viz. RA and SpA.

RHEUMATOID ARTHRITIS

Introduction

RA is a chronic inflammatory arthritis characterized by destructive synovitis leading to joint erosion with systemic organ involvement. It can affect all joints but typically the joints of hand and feet. Although it affects all age groups, it remains the most prevalent type of inflammatory arthritis in the older adults, being 2% in this population.

Elderly onset Rheumatoid arthritis (EORA) is defined as development of RA in patients over 60 years. EORA has different characteristics from the Younger Onset Rheumatoid arthritis (YORA). It contributes to 10-33% of cases of RA. Furthermore, the pendulum shifts from a female male ratio of 4 to 1 to 1.5 to 1 in the older population.

Pathogenesis

The pathogenesis is believed to originate from both environment and genetic factors, though the exact mechanisms are yet to be elucidated. An external trigger such as infection, smoking or trauma triggers an auto immune reaction leading to the typical synovial hypertrophy and chronic joint inflammation and extra-articular manifestations. The initiated synovial hyperplasia and endothelial cell activation causes an uncontrolled inflammation resulting in cartilage and bone destruction.

It is found that cells such as CD 4 T cells, phagocytes, fibroblasts, osteoclasts and neutrophils and cytokines such as tumour necrosis factor alpha (TNF α), interleukin-1 (IL-1), IL-6, IL-8, transforming growth factor (TGF-β), fibroblast growth factor (FGF) and platelet derived growth factor (PDGF) play important roles in the propagation of the inflammatory process.

Clinical features

The onset of RA is usually insidious with associated fever, malaise, arthralgias before the joint swelling. Persistent symmetric polyarthritis of hands and feet is the hallmark with extra-articular features and constitutional features. There is morning stiffness of joints lasting more than 30 minutes. Furthermore, the pain is worse with rest but is relieved by activity. There is synovial swelling of joints (boggy as opposed to bony in osteoarthritis). The metacarpophalangeal, proximal interphalangeal, knee, metatarsophalangeal, shoulder, ankle, cervical spine, hip, elbow and temporo-mandibular joint are the commonly affected joints. Extra-articular involvement of skin, lungs, heart and eyes is also common. Females are more affected than males but the sex difference diminishes with age. There is significant effect of ADL and vocational activities.

The commonly affected hand joints are metacarpophalangeal joints, wrist joints and proximal interphalangeal joints. Common deformities are volar subluxation / ulnar deviation of the fingers, swan neck & boutonniere finger deformities. In the feet the metatarsophalangeal joints followed by the ankle and mid foot joints are affected. The MTP joints when inflamed are more prone to deformity especially due to heavy load bearing. Hallux valgus deformity develops in the great toe and phalanx subluxation at MTP joints gives rise to hammer toes. With these deformities weight-bearing surfaces/axis will change and result in callus formation. Affected joints will show features of inflammation such as swelling, pain, warmth and erythema with resulting reduction in range of motion. Interosseous muscle wasting could also be present early. Chronic inflammation in the affected joints and surrounding tissue could result in tendon, ligament and joint damage resulting in deformities and joint ankylosis.

Rheumatoid nodules commonly occur in the extensor surfaces or sites of frequent irritation. Olecranon process, proximal ulna, Achilles tendon, ischial tuberosities and occiput are the common sites while they could also be present on extensor finger surfaces, toe and heel pads.

Most older patients with RA have active synovitis, burnt out disease with deformities leading to limitation of movement together with extra-articular manifestations or both. The deformities could be due to untreated or under treated disease in the early stages.

Some also have evidence of corrective surgeries performed on these deformities.

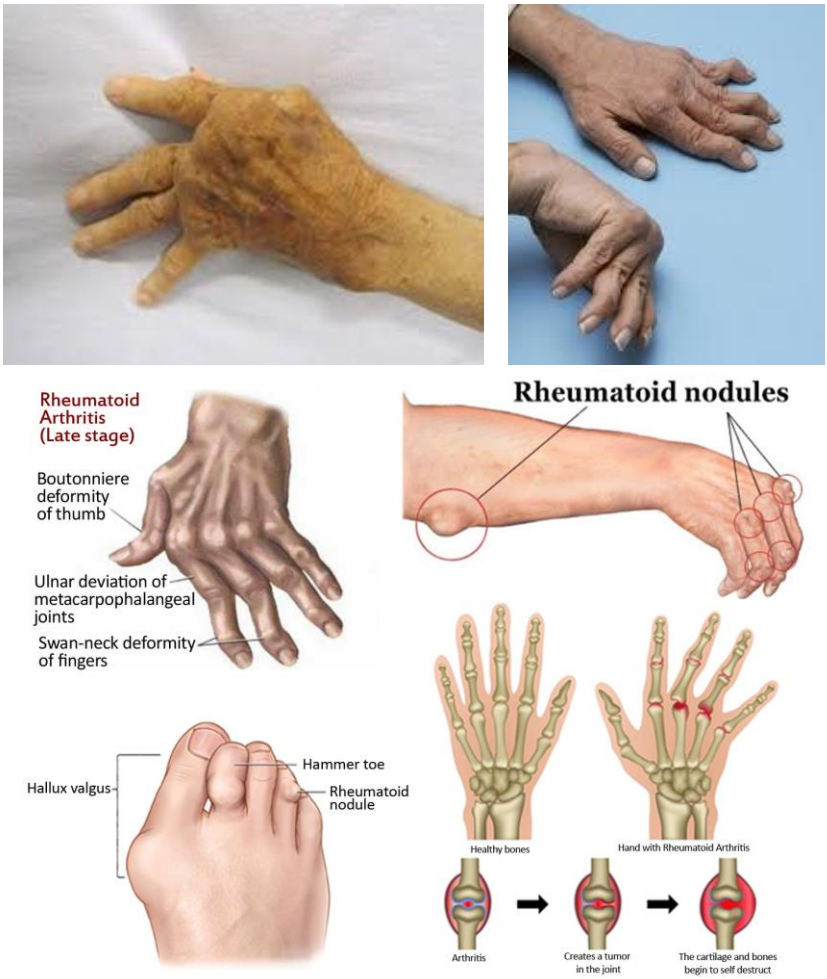

Figure 8.1 - Deformities in rheumatoid arthritis

Clinically, the manifestations of EORA can be heterogenous. There are three main patterns of the disease. Namely classical RA (70%) with positive RA (erosions and worse prognosis than YORA), the PMR-like form (25%) and the RS3PE like form. It is less likely to cause the typical hand deformities, rheumatoid factor positivity and extra articular manifestations than the EORA.

Table 8.2 - Differential diagnosis of RA in the older adults

Osteoarthritis
Polymyalgia rheumatica
Crystal arthritis (gout, pseudo gout, chronic pyrophosphate arthropathy)
Remitting seronegative symmetrical synovitis with pitting oedema (RS3PE)
Late onset Spondyloarthropathy
Connective tissue disease
Systemic vasculitis
Paraneoplastic arthritis
Hypertrophic osteoarthropathy
Sarcoidosis
Infectious arthritis (viral and bacterial infections)

Investigations

The diagnosis is made by a combination of clinical, laboratory and imaging features. No diagnostic test is pathognomonic. ESR, CRP, FBC should be performed.

Rheumatoid factor (RF) remains less useful in supporting the diagnosis with the increase of autoantibodies with age. Thus, anti-CCP antibodies may be more reliable, though it is not readily available. ESR and CRP increases can occur irrespective of joint involvement with increasing age attenuating their value in reaching a diagnosis. A FBC can show features of anaemia due to other factors related to ageing such as poor nutrition.

They are supplemented by plain radiography and also magnetic resonance imaging (MRI) and ultra-sonography (USS) of joints. Conventional radiography, though highly specific remains less sensitive for erosion detection as opposed to USS and MRI.

Treatment

Management of these patients is both non-pharmacological and non-pharmacological. The non-pharmacological therapies include heat and cold therapy, orthotics and splints, therapeutic exercise, occupational therapy, adaptive equipment, joint protection education and energy conservation education.

Pharmacological treatment includes analgesics such as paracetamol, NSAIDs, corticosteroids and non-biologic DMARDs (such as methotrexate, sulphasalazine, leflunamide and hydroxychloroquine) and biologic DMARDs such as TNF blockers (infliximab, etanercept, adalimumab, certolizumab, golimumab) and non-TNF blockers (rituximab, tocilizumab, tofacitinib).

Surgical procedures such as tendon realignment, reconstructive surgery and arthrodesis can be offered to patients with disabling deformities.

Due to the altered pharmacokinetics and pharmacodynamics of this age group, side effects should be closely monitored. Bone marrow aplasia is commoner with methotrexate. Out of the biological agents, TNFα blockers are found to be more efficacious than tocilizumab, etanercept, rituximab and tofacitinib.

Prognosis

The cardiovascular risk of RA itself augments the morbidity related to the existing cardiac disease in the older age group. It has also been found that the seronegative subset of EORA has a milder disease course than the classical RA while the seropositive EORA have a far aggressive disease and worse prognosis. Female gender and greater damage at presentation are related to poor outcomes. The EORA tend to have higher incidence of remission rate.

SPONDYLOARTHROPATHY (SPA)

Introduction

Spondyloarthropathy (SpA) is an umbrella term used to denote a collection of diseases with common characteristics such as axial and peripheral arthritis, enthesopathy and specific extra- articular

manifestations with HLA-B27 positivity. Ankylosing spondylitis, psoriatic arthritis, reactive arthritis, inflammatory bowel disease associated arthritis and undifferentiated spondyloarthropathy comes under this umbrella.

With increased life expectancy, these patients will survive with residual disease though the disease is inactive. They will have stiffness of the spine with stooped posture resulting in the characteristic question mark posture. Compared to RA, only 5% of the spondyloarthropathy patients are more than 50 years of age. It should be borne in mind that late onset /elderly onset of SpA (EOSpA) is underdiagnosed in favour of other inflammatory disorders which are common in older adults as clinical and radiological features are modified. The advent of new diagnostic tools such as MRI and new diagnostic criteria for axial spondylarthritis may help the clinicians to increase their diagnostic accuracy.

EOSpA is defined as diagnosis of the disease in the more than 50-year age group though most epidemiological studies evaluating treatment safety define older adults as more than 65 years. All spondyloarthropathy categories are diagnosed in seniors.

Pathogenesis

The pathogenesis is a result of complex interplay of genetic risk factors and environmental triggers leading to activation of auto immunity and inflammation. HLA-B27 remains the most important genetic factor while non-MHC genes of pathways of IL -17/23 have also being implicated in the pathogenesis. Abnormal intestinal microbiota and infections plays an important role in the development of the joint disease as well as mechanical stress which leads to micro damage, inflammation, and repair. At molecular level, the TNFα and IL-23/17 and at cellular level IL-23R +, CD3+/CD4-/CD8-T cells have been identified to play a role in the pathogenesis.

Clinical features

The prototype of spondyloarthropathy is ankylosing spondylitis which is characterized by inflammatory back ache with an onset usually before the age of 40 years with constitutional and extra-articular manifestations such as uveitis, inflammatory bowel disease, psoriasis,

abnormalities in the conduction system of the heart, valvular regurgitation, apical lung fibrosis and IgA nephropathy. Dactylitis and enthesopathy are other juxta-articular features of this condition.

The axial skeleton, namely vertebrae with associated enthesis and ligaments and the sacroiliac joints are affected in the axial spondyloarthropathy resulting in thoracic kyphosis, a positive Schober test, loss of chest expansion and loss of lumbar lordosis. There may be tenderness over the sacroiliac joints with direct and indirect compression. Shoulder joints, hip joints, costovertebral joints, costo-sternal joints, manubriosternal joints are some of the other joints involved in this condition. Other peripheral joints may be involved asymmetrically.

Features of SpA associated with psoriasis is termed psoriatic arthropathy, SpA associated with Crohn's disease or ulcerative colitis is termed inflammatory bowel disease associated spondyloarthropathy and SpA associated with preceding GI or GU infections is termed reactive arthritis. Patients are categorized under undifferentiated spondyloarthropathy, if they have features of spondyloarthropathy which do not fulfil criteria for any specific spondyloarthropathy.

The geriatric patients with long standing SpA will have the progressed stooped posture or evidence of corrective spinal surgery. There are deviations from the typical clinical presentations of SpA in the geriatric patient population. The older adults show more frequent constitutional symptoms, cervical spine involvement and peripheral arthritis of both upper and lower limb joints and mixed disease i.e. both axial and peripheral SpA. Out of the subsets, the undifferentiated SpA predominates, and the senior age group is more likely to have a shorter disease duration before diagnosis compared to their younger counterparts. (Figure 7.2)

Figure 8.2 - Progression of spondylarthritis

Table 8.3 - ASAS (Assessment of Spondylarthritis International Society) criteria for axial spondyloarthropathy

Back pain > 3 months and age > 45 years
For diagnosis: Sacroiliitis by MRI or radiography + 1 feature OR HLA B 27 + 2 other features are needed
Features of axial SpA
Inflammatory back pain
Arthritis
Enthesitis
Uveitis
Dactylitis
Psoriasis
Inflammatory bowel disease
Good response to NSAIDs
Family history of SpA
HLA B27
Elevated CRP

These diagnostic criteria can be used to diagnose the EOSpA as well, with the clinical co-relation proven in studies despite of not being validated for the > 45-year age group.

The clinical spectrum is of EOSpA is as broad as the young onset Spondyloarthropathy (YOSpA). Late onset ankylosing spondylitis patients have axial disease which predominates in the cervical spine while peripheral arthritis predominates in the lower limbs. Enthesitis, dactylitis and uveitis may occur in varying combinations. EOSpA patients can have distal inflammatory swelling with pitting oedema in up to one fifth of the patients. The undifferentiated variety is found to have more cervical and dorsal pain, anterior chest wall involvement, peripheral arthritis, aseptic osteitis, and systemic symptoms than patients with early onset SpA. The outset of the disease can mimic PMR or reflex sympathetic dystrophy. The psoriatic arthritis variant of EOSpA tends to be more severe and aggressive with significantly higher number of active joints, foot erosions. The lower limbs were found to be involved than upper limbs in an asymmetrical manner with associated inflammatory pitting oedema. The axial variety shows unilateral sacroiliitis and silent axial disease commonly. Reactive arthritis is not common in the geriatric population.

Table 8.4 - Diagnostic criteria for ankylosing spondylitis

New York criteria	Rome criteria
• Low back pain with inflammatory characteristics • Limitation of lumbar spine motion in sagittal and frontal planes • Decreased chest expansion • Bilateral sacroiliitis grade 2or higher • Unilateral sacroiliitis grade 3 or higher	• Low back pain and stiffness for >3 months that is not relieved by rest • Pain and stiffness in the thoracic region • Limited motion in the lumbar spine • Limited chest expansion • History of uveitis
Definite ankylosing spondylitis when fourth or fifth criterion mentioned presents with any clinical criteria	*Diagnosis of ankylosing spondylitis when any clinical criteria present with bilateral sacroiliitis*

Table 8.5 - CASPAR (Classification Criteria for Psoriatic Arthritis) criteria for the diagnosis of psoriatic arthritis (PsA)

Established inflammatory articular disease – 1 point
Current psoriasis -2 points, history of psoriasis (personal /family) – 1 point
Psoriatic nail history – 1 point
Juxta-articular new bone formation- 1 point
Negative rheumatoid factor – 1 point
To classify as PsA there should be 3 or more points.

The CASPAR criteria for psoriatic arthritis have similar sensitivity in the late onset psoriatic arthritis as in the early onset form.

Table 8.6 - Diagnostic criteria for Reactive arthritis

Typical peripheral arthritis: Predominantly lower limb, asymmetrical oligoarthritis plus
Evidence of preceding infection: a) If there is a clear history of diarrhoea or urethritis within the preceding four weeks, laboratory confirmation is recommended, but not essential b) If there is no clear infection, laboratory confirmation is essential
Exclusion criteria: Patients with other known causes of arthritis

Table 8.7 - Diagnostic criteria for Undifferentiated Spondyloarthropathy (6 or more points)

Inclusion criteria	Exclusion criteria
Inflammatory back pain-1 point	Diagnosis of specific spondyloarthropathy
unilateral buttock pain-1 point	Sacroiliitis on radiograph=grade 2
Alternating buttock pain-2 points	Precipitating genitourinary/ gastrointestinal infection
Enthesitis-2 points	Psoriasis
Peripheral arthritis-2 points	Keratoderma blennorrhagica
Dactylitis-2 points	Inflammatory bowel disease
Acute anterior uveitis-2 points	Positive rheumatoid factor
HLA-B27 positivity/family history of spondyloarthropathy-2 points	Positive ANA>1:80
Good response to non-steroidal anti-inflammatory drugs- 2 points	

The prevalence of extra-articular manifestations is the same in all varieties.

Table 8.8 - Differential diagnosis of axial SpA in the older adults

Osteoarthritis
Disc arthrosis
Diffuse idiopathic skeletal hyperostosis (DISH)

Table 8.9 - Differential diagnosis of peripheral SpA in the older adults

Remitting symmetrical seronegative synovitis with pitting oedema (RS3PE)
Polymyalgia rheumatica
Chondrocalcinosis
Amyloid arthropathy
Systemic lupus erythematosus
Mixed connective tissue disease
Sarcoidosis
Sjogren syndrome
Systemic sclerosis
Dermatomyositis
Polyarteritis nodosa
Paraneoplastic syndrome
Reflex sympathetic dystrophy

Investigations

Diagnosis is made by the afore mentioned clinical features and radiological features of the conventional X rays and the MRI. The latter is especially useful in the diagnosis of the early disease. ESR and CRP may be elevated in 75% of the patients and only co-relate with disease activity in some of the patients. HLA-B27 is positive in 92% of the Caucasians while the value is lower in other ethnicities.

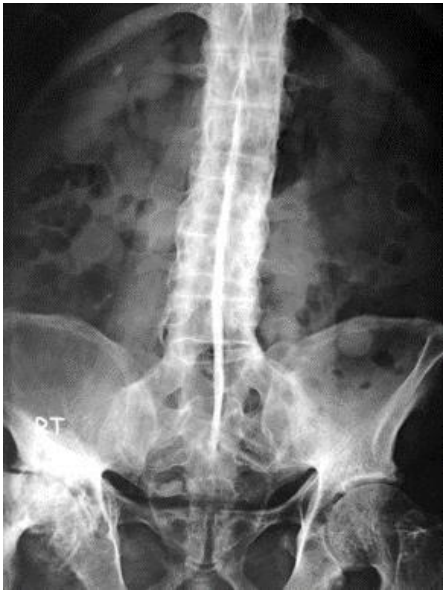

Figure 8.3 - Typical radiological changes of syndesmophytes and calcification of spinal ligaments leading to a fused spine

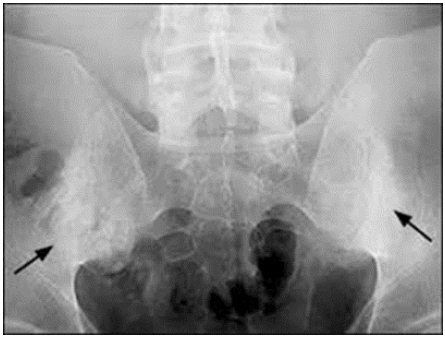

Figure 8.4 - Sacroilliac joint sclerosis and fusion in ankylosing spondylitis

The standard radiography is less helpful in the diagnosis due to age related skeletal changes such as osteoporosis and osteoarthritis. Thus, this clinical entity is under diagnosed in favour of other inflammatory disorders that are more frequent in the older adults. Although MRI is an important diagnostic tool with a specificity of 88%-98.5%, it is found that similar MRI changes were found in 20% of healthy individuals without inflammatory disease. Therefore, diagnosis requires a combination of clinical co- relation, HLA positivity and other

radiological findings. Ultrasound scan can be useful in the evaluation of the synovial and tenosynovial modifications and enthesial involvement.

Treatment

There is no definite disease modifying treatment for axial SpA. NSAIDs and biologics such as TNF α blockers and interleukin inhibitors can be used. Peripheral SpA patients can be treated with sulphasalazine, methotrexate and leflunomide. In addition, non-pharmacological therapies such as cessation of smoking and physical therapy is beneficial. The extra-articular manifestations such as skin, eye and bowel symptoms are managed in liaison with the relevant speciality.

The same principles of the management of spondyloarthropathy in the young applies to the older persons with certain adjustments in choice of drug class. Non-steroidal anti-inflammatory agents (NSAIDs) remains the first line therapy however the response is reduced. Side effects such as gastric ulcers and renal damage should be closely monitored due to low physiological reserve. Sulphasalazine remains the preferred DMARD in peripheral EOSpA and agranulocytosis with sulphasalazine is commoner in the older adults. Out of the biologics the TNF α blockers are found to be effective in the seniors. But there is lack of robust data for the latter. As there is a higher rate of associated infection, a rigorous evaluation should be done prior to initiating therapy.

Prognosis

Peripheral joint involvement, young age of onset, elevated ESR and poor response to NSAIDs are indicators of poor prognosis. The unilateral and intermittent nature of the disease at the onset could progress to more severe and constant joint stiffness which is symmetrical. With progression of the disease the inflammation of the spine leads to fusion, thoracic kyphosis and erosions causing disability. Age does not seem to affect the prognosis of EOSpA, but the co-morbidities and altered physiology in the older adult age group hinders the clinician's aggressive approach in managing these patients.

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9. Crystal Induced Arthritis

Dr. Deneshika Suriyaarachchi

Crystal induced arthritides disease are more prevalent among the geriatric population and they can present to medical attention with a variety of typical and atypical rheumatological involvement. Diagnosis of both of these common forms of arthritides need to be tailored to the patient while considering other medical problems that may remain as challenges to the clinician.

GOUT AND HYPERURICEMIA

Introduction

Gout is a clinical syndrome characterized by deposition of monosodium urate crystals (MSU) in extracellular fluid as a result of saturation of uric acid. The clinical spectrum of gout may include:

- Recurrent flares of inflammatory arthritis (gout flare)
- Chronic arthropathy
- Accumulation of urate crystals in the form of tophaceous deposits
- Uric acid nephrolithiasis
- Chronic nephropathy

There are many pharmacological agents that can be used to suppress gouty inflammation and reversal of hyperuricaemia to prevent unpleasant effects of acute inflammatory and chronic destructive manifestations of gout. However, this disorder still remains a challenge due to multitude of causes such as rising of the ageing population, polypharmacy, multimorbidity and lifestyle changes.

Gout is recognized as the most common form of inflammatory arthritis in men. Several epidemiological studies from different countries suggest that the incidence and prevalence of gout is increasing. The prevalence is likely to exceed 3 % of adults worldwide. Gout is more common in middle aged and in older persons (fourth or fifth decades in men and in sixth or seventh decades in women). Studies have suggested an average period of asymptomatic hyperuricaemia of at

least 10 years or more in both sexes prior to the clinical expression of gout.

Pathogenesis

Seventy percent of uric acid is formed from metabolism of cells as a degradation of purines in nucleic acid. Furthermore, a purine rich diet contributes to the uric acid pool in serum and tissues. Seventy percent of the uric acid pool content is excreted via the kidney while 30% is eliminated in the gut. Table 9.1 demonstrates the risk factors for this disease entity

Table 9.1 - Risk factors for hyperuricaemia and gout

Overproduction	Underexcretion	Mixed
Purine rich diet (meat, seafood) Increased cell turnover Tumour lysis syndrome Sickle cell anaemia Myeloproliferative disorders Psoriasis Lesch-Nyhan syndrome *HGPRT deficiency von Gierke disease *Hypoxanthine-Guanine Phosphoribosyl Transferase	Renal insufficiency Defects in urate renal transported system Drugs/ Toxins Thiazide diuretics Ethanol Cyclosporine A Pyrazinamide Lead nephropathy Low dose aspirin	Ethanol Increased lactic acid Fructose containing beverages e.g. cola

Uric acid becomes insoluble in serum and tends to deposit as crystals in tissues when serum concentrations are greater than 6.8 mg/dL at physiological pH and temperature (clinical hyperuricaemia). Crystal deposition in the synovium initiates an inflammatory reaction due to phagocytosis of the same by neutrophils. There is activation of complements cascading to a release of cytokines giving rise to a rapid inflammatory reaction (Figure 9.1).

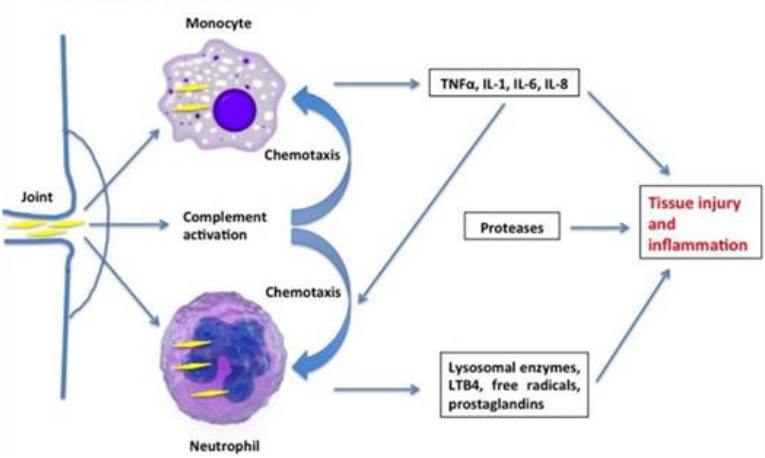

Figure 9.1 - Inflammatory cascade in gout

Risk factors for chronic hyperuricemia should be searched for in every person with gout. These include:

- Chronic kidney disease (CKD)
- Overweight and obesity
- Medications (e.g. diuretics, low-dose aspirin, cyclosporine and tacrolimus)
- Consumption of excess alcohol (particularly beer and spirits)
- Non-diet soda
- Meat and shellfish

Clinical Manifestations

There are four classic clinical stages in the natural history of progressive urate crystal deposition disease. Asymptomatic hyperuricaemia is usually the first stage that often starts years before symptoms appear due to the afore mentioned causes. This is followed by gouty flares, inter-critical gout, and chronic gouty arthritis and tophaceous gout (Figure 9.2)

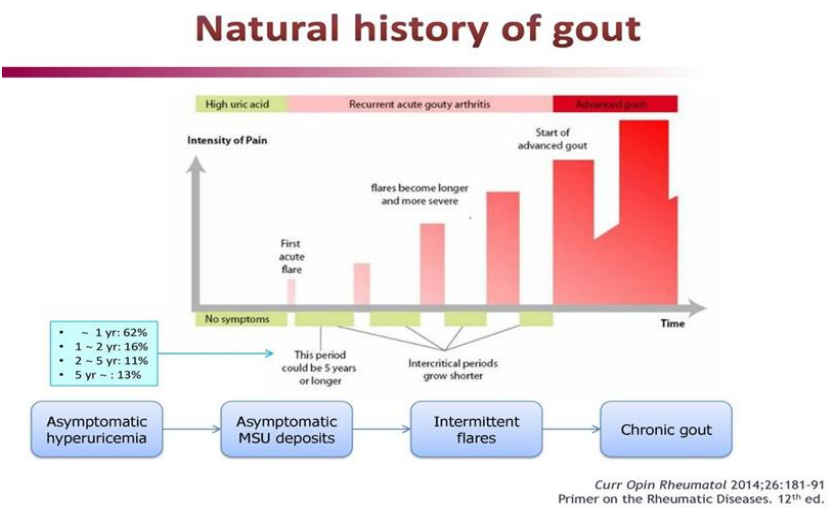

Figure 9.2 - The natural history of gout

a) Gouty flares

Acute gouty flares are typically monoarticular and intensely inflammatory, occurring in the lower extremities. However, some patients with more long-standing disease may experience polyarticular flares. The typical features demonstrate severe pain, erythaema, warmth, swelling and disability of the joint. The maximal severity of a flare is usually achieved within 12 to 24 hours. Complete resolution of the earliest flares almost always occurs within a few days to several weeks even in untreated patients. Gout flares are more common at night time and in early morning.

The most commonly affected sites are the base of the great toe (first metatarsophalangeal (MTP) joint - podagra) or the knee (figure 3). Inflammatory features can extend beyond the joint territory giving an impression of dactylitis, cellulitis or tenosynovitis. Recurrent gout flares can involve ankle, wrist, fingers, shoulders, hips and bursas. Gout in the spine is much less common but can affect lumbar region and sacroiliac joints giving rise to diagnostic confusion. In patients with hyperuricaemia, gout may arise secondary to a myeloproliferative,

lymphoproliferative disorder or in organ transplant recipients on tacrolimus or cyclosporine, with polyarticular flares as the initial presentation. (Figure 9.3)

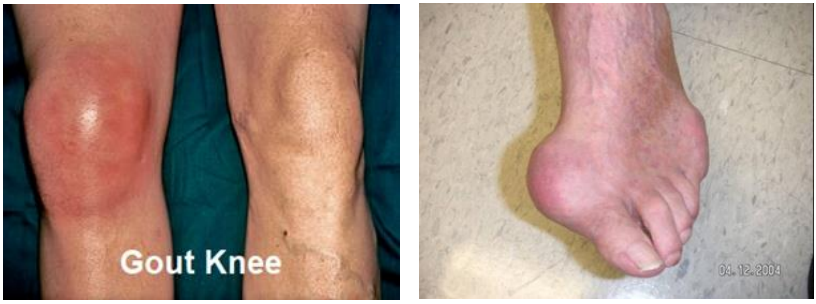

Figure 9.3 - Gout knee and Podagra of foot

b) Inter-critical gout and recurrent gout flares

The period between two gout flares is known as inter-critical stage. In the early course of disease, it is entirely asymptomatic, even after a severe and incapacitating flare. This characteristic is uncommon in other arthritic disorders therefore alerting clinicians to revisit diagnosis in such instances. The intervals between gout flares can be variable. Studies suggest that most untreated patients with gout will experience a second episode within two years.

c) Tophaceous gout

The deposition of solid urate along with chronic granulomatous inflammatory process is characteristic of tophaceous gout. There are destructive changes to the surrounding connective tissue. Tophi are often visible or palpable and can be present on pinnae or in the soft tissues such as articular bone and cartilage, tendons, ligaments, entheses or bursae. Tophi are typically not painful nor tender. However, they may discolour the skin revealing a yellow or white hue (Figure 9.4).

The chronic inflammatory process may extend beyond the confines of a single joint, producing a generalized enlargement of a digit due to the presence of tophi or the inflammation itself. The clinical appearance may be similar to dactylitis seen in other disorders such as psoriatic arthritis, other spondyloarthritides and sarcoidosis.

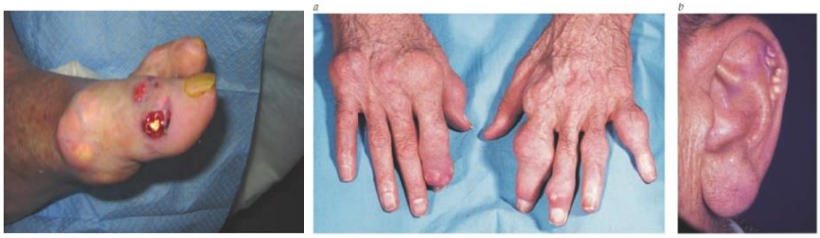

Figure 9.4 - Tophi in big toe (with ulceration), hands and pinna

Differential diagnosis of gout includes the following:

- Septic arthritis
- Stress fractures
- Calcium pyrophosphate crystal deposition disease (CPPD)
- Cellulitis
- Reactive arthritis
- Palindromic rheumatism

When synovial fluid analysis is not feasible, a clinical diagnosis of gout is made and supported by the following suggestive features (although they are not specific for gout):

- Monoarticular involvement of a foot (especially the first MTP) or ankle joint.
- Previous similar acute arthritic episodes,
- Rapid onset of severe pain and swelling (at its' worst in less than 24 hours) erythema
- Male sex and associated cardiovascular diseases
- Hyperuricaemia.

Tophaceous gout may mimic the clinical presentation of rheumatoid arthritis and tophi may be mistaken for rheumatoid nodules. Other differential diagnoses for tophaceous gout include dactylitis and osteomyelitis.

Laboratory Findings

Neutrophilic leucocytosis or elevation of the ESR and CRP that are commonly seen in gout flares have less diagnostic value.

Visualisation of synovial fluid (joints or bursae) under polarized light microscopy for the presence of MSU crystals is the gold standard diagnostic method during a flare. Material aspirated during inter-critical period from previously affected joints and tophaceous deposits can also be used. The crystals are negatively birefringent and needle in shape which appear yellow and blue at perpendicular planes (figure 9.5). The synovial fluid is inflammatory in type, with white blood cell counts ranging 10,000 - 100,000 showing neutrophil predominance. Synovial fluid will however have normal glucose levels.

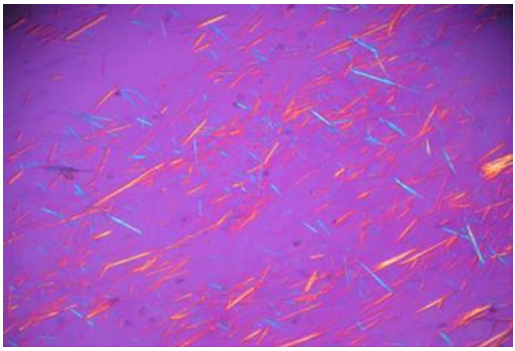

Figure 9.5 - Negatively birefringent needle shaped urate crystals in synovial fluid

Serum urate levels can be difficult to interpret during a gout flare as it may be high, normal, or low. In patients suspected of gout based upon clinical features, an elevated serum urate can lend support to the diagnosis. However, this is neither diagnostic nor required to establish the diagnosis. The most accurate time for assessment of serum urate is at least post two weeks' resolution of a flare. Diagnosis should not be made in the presence of hyperuricaemia alone as it is not a surrogate marker for the diagnosis.

Imaging

Imaging findings vary at different stages of gout. Plain radiographic changes take several years to develop, therefore being helpful in supporting a diagnosis of gout in the later stages. In the early phase,

radiography is often normal but soft tissue swelling may be apparent. Subcortical bone cysts may be suggestive of tophi or erosions. Para-articular erosions with 'overhanging edges' of bone are characteristic of gout. Later in the disease, radiographs may demonstrate tophi near joints, tissue swelling, para-articular erosions, periosteal new bone formation and joint deformity (figure 9.6). Many features can mimic RA.

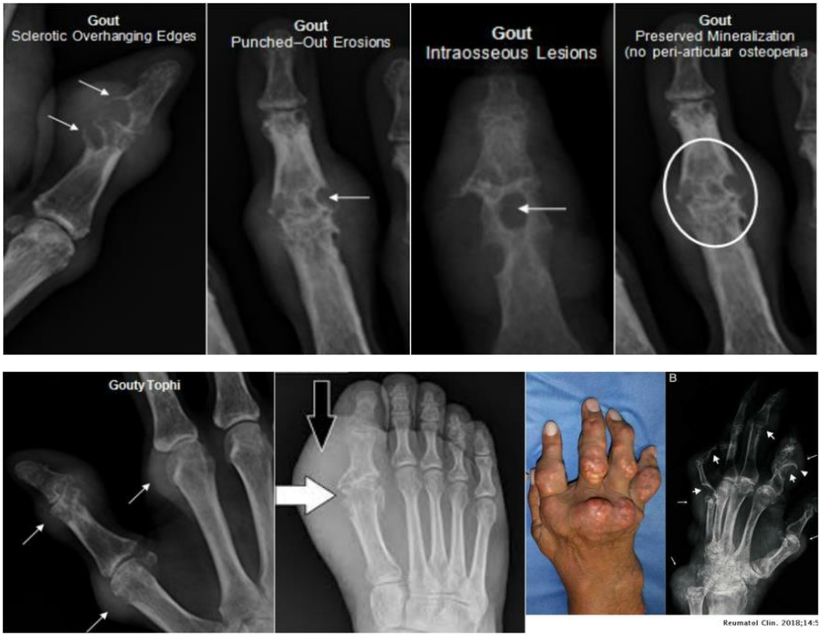

Figure 9.6 - Radiographic imaging of gout

Findings on ultrasound examination can strongly and independently support the diagnosis of gout and may be useful in the early detection and monitoring of therapy. Important diagnostic features include double contour sign or tophaceous-appearing deposits in joints or tendons, which are represented by an ovoid stippled signal (hyperechoic cloudy area). MRI can demonstrate subcortical bone cysts and overhanging edges of bone associated with bone erosions due to tophi occurring with more chronic disease. The characteristics of tophi on MRI include relatively homogeneous intermediate to low signal intensity on T1-weighted images. Dual-energy computed tomography

(DECT) examination can specifically identify urate deposits in articular and periarticular locations and can distinguish urate from calcium deposition.

Management

All patients should be informed about the disease process, effective treatment modalities available and the principles of managing acute attacks with the necessity of lifelong lowering of serum uric acid (SUA) below a target level. Advice should be offered on lifestyle modifications such as weight loss, cessation of smoking, and avoidance of alcohol, sugar-sweetened drinks, heavy meals and excessive intake of meat and seafood. Consumption of low-fat dairy products and regular exercise should be encouraged.

An integral part of the management of gout is the screening for associated comorbidities such as renal impairment, coronary heart disease, heart failure, stroke, peripheral arterial disease, obesity, hyperlipidaemia, hypertension and diabetes.

Acute flares of gout should be treated as early as possible. Patients should be educated to identify and self-medicate at the first warning symptoms. Rest, elevation and ice packs can partly alleviate symptoms. The choice of drugs should be based on the presence of comorbidities that may contraindicate, the patient's previous experience with treatments, time of initiation after flare onset, the number and type of joints involved.

The recommended first-line options for an acute flare are colchicine, NSAIDs, oral corticosteroids or articular aspiration with or/without injection of corticosteroids. Colchicine and NSAIDs should be avoided in patients with severe renal impairment. Colchicine should not be given to patients receiving cyclosporin or clarithromycin. Urate lowering therapy (ULT) is ineffective in acute flares. Patients who are on ULT already should continue it in the same dosage. NSAIDs reduce the pain, swelling and the duration of gout attacks. All NSAIDs including COX-2 selective have similar efficacy to treat gout. Patients with frequent flares and contraindications to the afore mentioned medication or having refractory disease should be considered on treatment with IL-1

inhibitors such as anakinra or canakinumab. Active infection is a contraindication to the use of IL-1 inhibitors.

Prophylaxis against flares is recommended during the first 6 months of ULT. The recommended drug of choice is colchicine with dose adjustment for renal functions. If it is not tolerated or is contraindicated, NSAIDs at a low dosage should be considered (if not contraindicated).

ULT should be considered with every patient with a definite diagnosis of gout from the first presentation. Indications includes recurrent flares (≥2/year) and presence of tophi, urate arthropathy and renal calculi. Early initiation of ULT is recommended close to the time of first diagnosis in patients presenting at a younger age or with comorbidities. SUA level should be monitored and maintained to <6mg/dL lifelong. Patients severe degree disease may require a target of <5mg/dL.

Allopurinol is recommended as the first-line ULT for patients with normal renal functions, initiated at a low dose and increasing gradually to reach the uricaemic target. If the SUA target cannot be reached by an appropriate dose, allopurinol should be combined with or switched to febuxostat or a uricosuric. These drugs are also prescribed if allopurinol cannot be tolerated or life threatening cutaneous adverse effects like toxic epidermal necrolysis and Stevens-Johnson syndrome develops. Allopurinol needs to be directed with extreme caution in the older adults and patients with renal dysfunction. In patients with renal impairment, the allopurinol maximum dosage should be adjusted to creatinine clearance. If the SUA target cannot be achieved at this dose, the patient should be switched to febuxostat or given benzbromarone with or without allopurinol, except in patients with eGFR <30 ml/min. Febuxostat is more effective in patients with chronic kidney disease than dose adjusted allopurinol.

Pegloticase or rasburicase is indicated in crystal-proven severe debilitating chronic tophaceous gout and poor quality of life, in whom the SUA target cannot be reached with any other available drug at the maximal dosage.

Substitution or cessation of a diuretic (if permissible) should be done if in the presence of a loop or thiazide diuretics -a flare develops.

CALCIUM PYROPHOSPHATE CRYSTAL DEPOSITION DISEASE

Introduction

Precipitation of crystals of calcium pyrophosphate dihydrate (CPP) in connective tissues may be asymptomatic or may be associated with the spectrum of calcium pyrophosphate crystal deposition (CPPD) disease resulting inflammatory arthritis and degenerative chronic arthropathies with radiographic cartilage calcification. Traditionally much terminology was used to describe CPPD that include pseudogout, chondrocalcinosis and pyrophosphate arthropathy.

CPPD disease is a disease of older adults. The average age at diagnosis of CPPD disease is 72 years with no major sex predominance. Radiographic surveys demonstrate an age-related increase in the prevalence of articular cartilage calcification.

CPP crystal formation is initiated in cartilage. Although the exact mechanism is not known, it is speculated to be associated with excessive CPP production by chondrocytes, augmented pyrophosphate metabolism and increased level of calcium.

Most cases of CPPD disease are idiopathic. The following conditions are however associated in young patients:

- Joint trauma
- Familial chondrocalcinosis
- Metabolic /endocrine disorders
  e.g. haemochromatosis, hyperparathyroidism, hypomagnesemia
- Use of bisphosphonates

Clinical Manifestations

Most individuals with CPPD are asymptomatic in absence of any joint involvement although radiographic evidence may be present. The peculiar feature of CPPD is that it can mimic any type of inflammatory or non-inflammatory arthritides. The spectrum of this disease entity is as follows

- Asymptomatic CPPD
- Acute CPP crystal arthritis (pseudogout)

- Chronic CPP crystal inflammatory arthritis (pseudo rheumatoid arthritis)
- Osteoarthritis with CPPD, with or without superimposed acute attacks
- Severe joint degeneration
- Spinal involvement

Acute CPP crystal arthritis is characterized by acute or sub-acute attacks of self-limiting inflammatory arthritis involving one or several joints of extremities. It resembles the acute gouty flares (the knee is mostly affected). Initial episodes of acute CPP crystal arthritis may persist longer before remitting. Trauma, parathyroidectomy and treatment with bisphosphonates or granulocyte-macrophage colony-stimulating factor (GM-CSF) has also been reported to precipitate acute attacks of acute CPPD.

Chronic CPP crystal inflammatory arthritis is a non-erosive, inflammatory arthritis with demonstrable crystals in joint fluid. This resembles rheumatoid arthritis in several aspects: presence of significant morning stiffness, fatigue, synovial thickening, localized oedema, and restricted joint movement in a symmetric pattern.

Osteoarthritis with CPPD is the most prevalent form of symptomatic CPPD disease. Many patients with symptomatic CPP crystal deposition disease show progressive (usually multiple) joint degeneration. Clinical features include asymmetric bony enlargement, tenderness, effusions, crepitus, and restricted joint motion. CPP crystal deposition is associated with severe joint degeneration which closely resembles neuropathic arthropathy with preserved neurological function.

CPP crystal deposition in the spine has been associated with a number of clinical manifestations which can resemble spinal changes of ankylosing spondylitis or diffuse idiopathic skeletal hyperostosis (DISH). Crystal deposition over the ligaments may lead to spinal cord compression syndromes.

Imaging

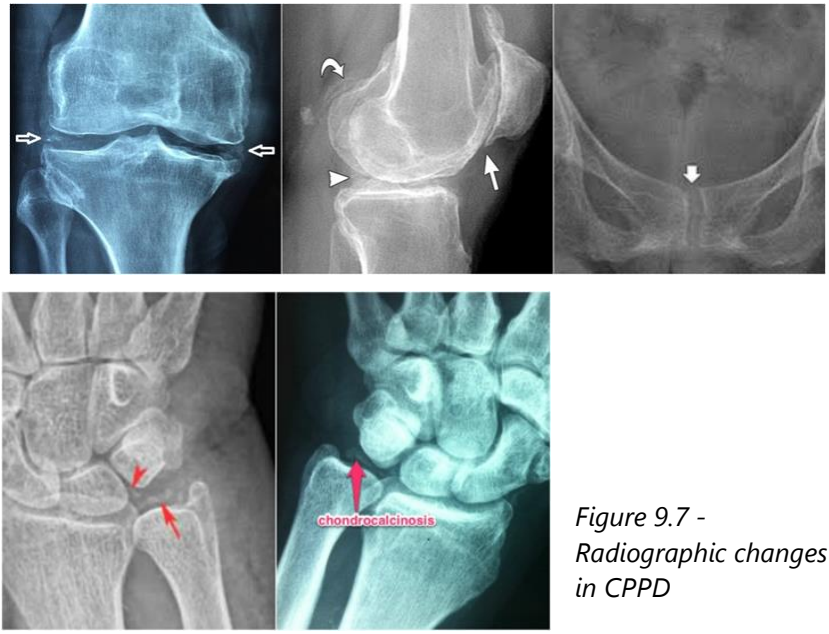

Figure 9.7 - Radiographic changes in CPPD

Calcification of cartilages (chondrocalcinosis) is the characteristic finding of CPPD in plain film radiography. CPPD typically appears as linear radiodensities in articular cartilage mainly in fibrocartilage or hyaline cartilage often seen in the menisci of the knee bilaterally and other large joints. Degenerative changes are also often associated with CPPD and characteristic features of osteoarthritis, including subchondral cysts, osteophyte formation and bone and cartilage fragmentation are recognized (figure 9.7).

Ultrasonography is a promising modality for clinical use in the diagnosis of CPPD disease and tracking the efficacy of treatment of CPPD. In CPPD, ultrasonographic appearance resembles the double contour sign (DCS) initially described in gout. Other features include small hyperechoic rounded amorphous shaped regions, nodular hyperechoic deposits in bursae and hyperechoic lines of calcification running parallel to tendon fibres.

Diagnosis

The diagnosis of CPPD is largely based upon the demonstration of CPP crystals in tissue or synovial fluid, and/or radiographic evidence of the disease. A diagnosis of CPPD disease should be suspected in an elderly individual with acute or sub-acute attacks of arthritis mainly of the knees, arthritis similar in character to rheumatoid or osteoarthritis, with radiographic changes. Patients suspected of CPPD disease should undergo arthrocentesis and synovial fluid analysis and conventional radiography. Radiographs of asymptomatic joints is sometimes also helpful.

Arthrocentesis of an affected joint with synovial fluid analysis should have presence of positively birefringent rhomboidal CPP crystals under polarized light microscopy (figure 9.8). Total synovial fluid leukocyte concentration in an acute attack is typically 15,000 to 30,000 per mm^3, with neutrophilic predominance.

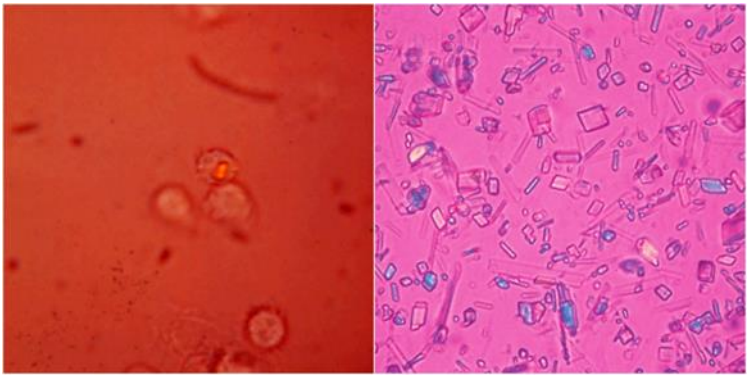

Figure 9.8 – Calcium pyrophosphate crystals in synovial fluid

Screening for associated metabolic conditions is particularly helpful when CPPD is diagnosed at a young age. Familial forms of CPPD disease may also occur early in life.

Management

Optimal treatment of CPPD requires both non-pharmacological and pharmacological modalities which should be tailored according to clinical features, comorbidities and the presence of a predisposing

metabolic disorder. The optimal and safe treatment for acute CPPD comprises of application of ice or cool packs, temporary rest, joint aspiration and intra-articular injection of long-acting glucocorticoids. Oral NSAIDs and low-dose oral colchicine are effective systemic treatments for acute CPP crystal arthritis which should be used with caution in elderly.

Other alternatives include a short tapering course of oral or parenteral glucocorticoids or ACTH. Prophylaxis against frequent recurrent acute CPP crystal arthritis can be achieved with low-dose oral colchicine or low-dose oral NSAIDs. The management objectives and treatment options for patients with OA and CPPD are the same as those for OA without CPPD. Pharmacological options in order of preference for chronic CPPD are oral NSAIDs or colchicine, low-dose corticosteroid, methotrexate and hydroxychloroquine. Associated condition should be treated if detected.

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10. Neck Pain

Dr. Prasanna Cooray

Although not as a common as lower back pain, neck pain still remains as a leading cause of disability. The musculoskeletal structures of the neck have the unenviable task of providing protection to the spinal cord whilst ensuring that a remarkable amount of movement is maintained.

AETIOLOGY

Neck pain can be categorized as causes related to

1. Musculoskeletal structures of the neck
2. Non-musculoskeletal structures of the neck

Musculoskeletal structure related causes

Pain arising from the joints due to wear and tear is primarily seen in this group (e.g. spondylosis, osteoarthritis). Pain in the cervical joints could also be due to inflammatory arthritis such as rheumatoid arthritis or axial spondylarthritides. The principal joints affected are the inter-vertebral and facet/zygapophyseal joints (Figure 10.1).

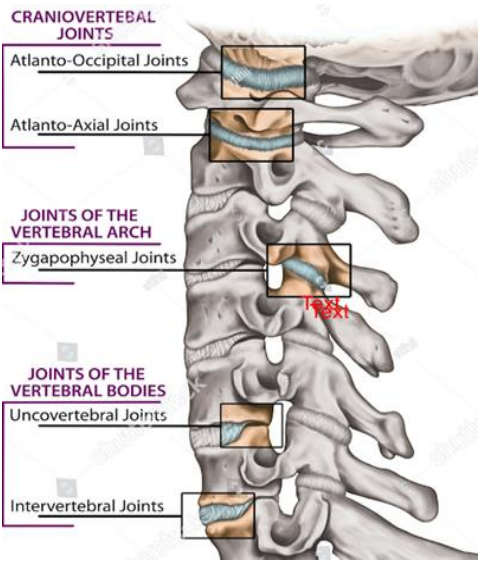

Figure 10.1 – Joints of the cervical spine

Nerve root entrapment is caused by an acute cervical disc prolapse or imparted pressure on the root from spondylotic osteophytes narrowing the root canal. Cervical spondylosis occurs in older persons with posterolateral osteophytes compressing the nerve root.

Whiplash injury results from acceleration deceleration forces. The common presentation is usually in a road traffic collision when a passenger wearing a seat belt in car is collided by another vehicle from the back. There is low possibility of bony injury if there is no midline cervical tenderness, no focal neurological deficit, no intoxication, no other painful distracting injury or if there is normal alertness.

Non-musculoskeletal related causes

Neck pain can be generated from sources outside the musculoskeletal structures of the cervical spine due to the phenomenon of referred pain. These are listed in Table 10.1.

Table 10.1 – Non-musculoskeletal related causes of neck pain

STRUCTURE	CONDITION
Pharynx/Larynx	Pharyngitis/laryngitis, laryngeal carcinoma
Trachea	Tracheitis
Lymph nodes	Lymphadenitis
Carotid arteries	Carotidynia, dissection, inflammation
Aorta	Aortic dissection
Cardiac	Ischaemia, pericarditis
Diaphragm	Irritation by infection

HISTORY AND EXAMINATION

The characteristics of pain related to the musculoskeletal structures of the neck are the following.

- Dull, deep and aching nature
- Pain made worse by activity and exertion but relieved by rest
- Pain that is usually centred around the posterior aspect of the neck (It is very unusual that it presents anterior to the sternocleidomastoid)

It is noteworthy that radiation of pain from the upper cervical region causes headache especially in the occiput. These are caused by pathologies above C3 vertebrae. Lower cervical pain radiates to the shoulder girdle, chest wall and inter-scapular region.

A sinister cause of neck pain may be pointed by the following symptomatology

- Unremitting pain (as in a malignancy or an infection)
- Electrical shock like or shooting pain (due to involvement of neural structures)
- Pain associated with early morning stiffness (as in Rheumatoid arthritis and polymyalgia rheumatica)
- Red flag symptoms of fever, weight loss and a past history of malignancy (Red flag signs)

The concept of radicular pain should be explored in history taking. It is usually a sharp and shock like phenomenon but however it can present as dull and aching type as well. Radicular pain from the root lesions of C2 & C3 may affect the head while root lesions involving C4 may cause pain across the top of the shoulders. These segments are seldom involved in benign spondylosis and should be taken as a harbinger of a space occupying lesion. Cervical Spondylosis usually involves the C6, C7 and C8 nerve roots which causes referred pain to the forearm and hand.

Symptoms such as imbalance and incontinence should alert the clinician to cervical cord compression.

A general examination with in consideration on structures that could be causing a referred pain should be carried out. Cervical lymph nodes should always be assessed. While the yield of a thorough general examination is low *vis a vis* a sinister cause, it should be weighed for the potentially disastrous consequences of missing a diagnosis that requires urgent treatment.

Once a diagnosis of musculoskeletal neck pain is made the following should be examined

1) Local tenderness – The response to pressure on the occipital protuberance can be used as a reference to elicit tenderness along the spinous processes, articular pillars and the trapezius muscle. However, generally the anatomic and aetiological correlation between areas of tenderness and actual pathology is poor.

2) Movements of the neck – Although traditionally done, it is of limited diagnostic value as movements of the cervical spine extend across a number of multiple joints with significant variation among different individuals. At best limitation of movement implies mechanical pain in the complex articular structures present in the cervical region

3) Signs related to nerve root compression - When the complaint is suggestive of radicular pain, it is reinforced with hard clinical signs. This is an indication for nerve conduction studies and/or MRI of the cervical spine.

Table 10.2 - Motor findings in nerve root involvement

Nerve Root	Muscle weakness/ Movement affected	Associated Tendon Jerk
C5	Shoulder abduction & Elbow flexion	Biceps
C6	Wrist extension & pronation	Supinator
C7	Elbow & wrist extension	Triceps
C8	Elbow & Finger extension	Finger
T1	Finger abduction, Thumb adduction and opposition	

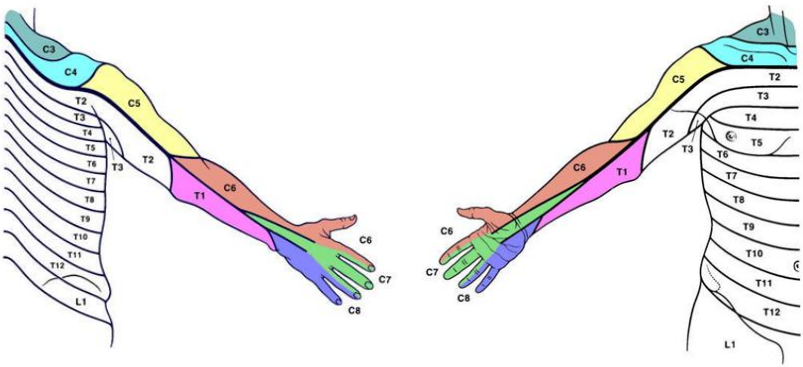

Figure 10.2 – Dermatomes of the upper limb

4) Signs related to possible cervical cord compression – These include extensor plantar response with exaggerated reflexes. Furthermore, a sensory level would confirm this manifestation. Cervical cord compression requires urgent referral to a neurosurgeon.

INVESTIGATIONS

In the absence of worrisome clinical history and findings, investigations are largely un-informative. An ESR (raised in inflammation and malignancy), full blood count (In infection), alkaline phosphatase and

calcium levels (raised in Paget Disease of bone and metastasis) are useful in the work up.

The cervical spine demonstrates radiographic evidence of wear and tear in older adults which may not be related or is disproportionate to the signs and symptoms observed. Lateral Flexion and extension view plain radiography can be used to look for atlantoaxial subluxation. X-rays are also of value if a recent trauma to the cervical spine has occurred and a fracture is suspected. While MRI has largely superseded the use of CT, bony pathology can be better identified through CT scans especially in whiplash injury. MRI in most cases would help establish a definitive diagnosis in particular for soft tissue injury.

Other useful investigations in an appropriate clinical context would be nerve conduction studies (nerve root lesions) and isotope bone scans (to differentiate inflammatory from degenerative pathology).

MANAGEMENT

Exercise

Exercise aims to increase range of motion and strengthen the musculature of the neck. Although there is reasonable evidence that exercise-based therapy has a role in providing relief especially in those with chronic or recurrent neck pain, no definitive trials have been done on the type of exercise that would show most beneficial. However, evidence is available to demonstrate that dysfunction of the deep neck flexors is responsible for pain in patients with long standing neck pain. It is therefore rational to target these muscles in exercise regimes.

Physical therapy & posture

Other physical therapy includes transcutaneous electrical nerve stimulation (TENS), infrared therapy, shortwave therapy, ultrasound therapy and laser therapy. All of these modalities seem to show some effectiveness in pain score reduction albeit in the short term. Manipulation or manual therapy also demonstrate short term benefits. The recommendations involving maintenance of correct posture while sitting and use of appropriately contoured pillows while sleeping has shown to be a useful adjunct.

Control of pain

Pain relief is a vital and the most important component of management. Most clinicians prefer a combination of paracetamol, NSAIDs, opioid analgesics (codeine and oxycodone) and neuropathic pain relief with agents such as pregabalin. Most radicular pain tends to spontaneously resolve over days to weeks and the above treatment modalities are sufficient for managing the symptoms in the interim. Surgery for nerve root lesions seem to offer more rapid relief when compared to conservative measures. However, such relief is not sustained long term unless a definitive underlying cause for the root lesion such as a tumour or infection is addressed. Indications for surgery for radicular pain would be progressive or unremitting symptoms which does not respond to conservative management.

Cervical collars are now frowned upon as they tend to delay recovery and at times promote muscle spasm.

Further reading

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11. Back Pain

Dr. Kusala Narangoda

Lower Back pain (LBP) is a ubiquitous medical problem, particularly among the older adults. Studies have suggested that the prevalence of musculoskeletal pain in this population ranges from 65 to 85%, with 36 to 70% of them suffering back pain. While most causes of LBP are non-specific and self-limiting, older adults are prone to develop certain LBP pathologies, given their age-related physical and psychosocial changes. An older adult with a very first episode of low back always warrants careful evaluation. Degenerative changes in the lower back are a normal feature of ageing: henceforth back pain should not be attributed to these changes every time, as this may conceal an underlying sinister pathology. Advance age itself is/can be a red flag of an underlying systemic pathology.

AETIOLOGY

The majority (>85 percent) of patients with back pain seen in primary care will have nonspecific low back pain i.e. patients have back pain in the absence of specific underlying conditions that can be reliably identified. Most of them improve within a few weeks.

LBP can be categorised into three main categories:

1) Mechanical lower back pain
2) Non-mechanical lower back pain
3) Visceral pain

Compared to younger generation, older adults are more likely to develop serious LBP pathologies such as osteoporotic vertebral fractures, metastatic bone malignancies, spinal infection, lumbar spinal stenosis and visceral disease with secondary back pain.

Poor general wellbeing is a known predictor of back pain. The older population is at even greater risk s health status tends to decline with age. Another overlooked but important associated condition is depression, which is significantly associated with the occurrence of

disabling back pain in those aged more than 70 years of age. Untreated or under-treated older adults with LBP may end up with sleep disturbances, withdrawal from social and recreational activities, psychological distress, impeded cognition, malnutrition, rapid deterioration of functional ability, falls, and ultimately depression. Hence it can ultimately become a vicious cycle.

Table 11.1 - Causes of low back pain

Mechanical lower back pain	**Non-mechanical lower back pain**	**Visceral pain**
1. Lumbar strain 2. Degenerative disease-Discs (spondylosis) Facet joints (osteoarthritis) 3. Spondylolisthesis 4. 4. Herniated disc 5. Spinal stenosis 6. Osteoporosis and fracture Congenital disease -Severe kyphosis -Severe scoliosis -Possible type II or type IV transitional vertebra 7. Possible spondylolysis 8. Possible facet joint asymmetry	1. Neoplasia -Multiple myeloma -Metastatic carcinoma -Lymphoma and leukaemia -Spinal cord tumours -Retroperitoneal tumours 2. Infection -Osteomyelitis -Septic discitis -Para spinous abscess -Epidural abscess ▪ Inflammatory arthritis (often HLA-B27-associated) Active or burnt-out disease -Ankylosing spondylitis -Psoriatic spondylitis -Reactive arthritis -Inflammatory bowel disease 3. Scheuermann disease (osteochondrosis) 4. Paget disease	1. Pelvic organs -Prostatitis -Endometriosis -Chronic pelvic inflammatory disease 2. Renal disease -Nephrolithiasis -Pyelonephritis -Perinephric abscess 3. Aortic aneurysm 4. Gastrointestinal disease -Pancreatitis -Cholecystitis -Perforating ulcer 5. Fat herniation of lumbar space

SERIOUS SYSTEMIC PATHOLOGY

Among patients who present with back pain to primary care settings, less than 1% percent will have a serious systemic aetiology such as cauda equina syndrome, metastatic cancer or spinal infection. These represent potentially treatable conditions.

Spinal cord or cauda equina compression

Cauda equina compression is a syndrome which has many causes e.g., herniation of the intervertebral disc (22.7 %), ankylosing spondylitis (15.9 %), lumbar puncture (15.9%), trauma (7.6%), malignant tumour (7.2%), benign tumour (5.7%), and infection (5.3%). Patients with compressive lesions in the lumbosacral spine below the level of the conus medullaris may present with signs and symptoms of cauda equina syndrome. Symptoms are due to compression of multiple nerve roots of the cauda equina and may include pain radiating into one or both legs (radicular pain), paraesthesia and sensory loss in the distribution of one or more nerve roots (including saddle anaesthesia) and patchy and asymmetrical weakness and loss of reflexes. Bilateral lower extremity pain is the classic presentation that raises concern for cauda equina syndrome. Bowel and bladder dysfunction may occur with compression of the lower sacral nerve roots.

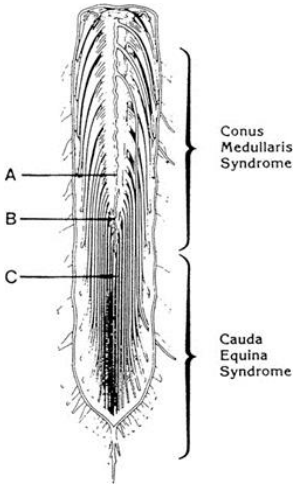

Figure 11.1 - Anatomy of conus medullaris syndrome and cauda equina syndrome

Table 11.2 - Comparison of conus medullaris syndrome and cauda equina syndrome

	Conus Medullaris Syndrome	**Cauda Equina Syndrome**
Level	L1-L2	L2- Sacrum
Spinal Level	Sacral cord segment and roots	Lumbosacral nerve roots
Presentation	Sudden and bilateral	Gradual and unilateral
Low back pain	More	Less
Motor strength	Symmetrical Less marked Hyper-reflexic distal paresis Fasciculation	Assymetrical More marked Areflexic paraplegia Atrophy more common
Reflexes	Ankle jerks affected	Both knee and ankle jerks affects
Sensory	Localised numbness to perianal area Symmetrical and bilateral	Localised numbness at saddle area Assymetrical and unilateral
Sphincters	Early urinary and faecal incontinence	Late presentation
Impotence	Frequent	Less frequent

Neoplastic epidural spinal cord compression (ESCC) is a complication of cancer that can cause pain, mechanical instability of the spine, and potentially irreversible loss of neurologic function. Early recognition and diagnosis of ESCC facilitates definitive and palliative therapies, which aim to maximise function, treat pain, and prevent further neurologic complications. Majority of patients with ESCC have pain as the initial symptom prior to the onset of motor or bladder dysfunction. Approximately 60 to 70 percent of cases occur in the thoracic spine while 20 to 30 percent are seen in the lumbosacral spine and 10 percent in the cervical spine. Affected patients usually notice a severe local back pain, at the level of the lesion, which progressively increases in intensity. The pain is often worse at night.

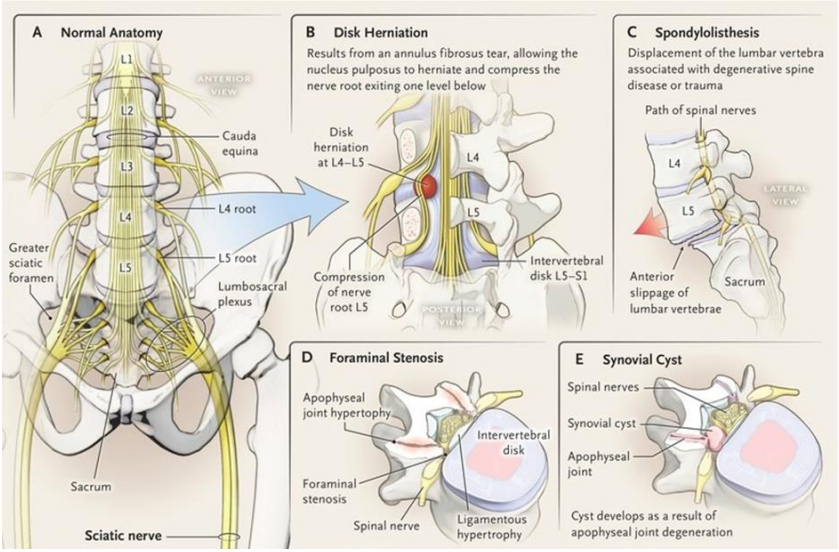

Figure 11.2 - Compressive pathologies of the lower back

Malignancy

The incidence of malignancy increase with age. Bone is one of the most common sites of metastasis. A history of cancer (excluding non-melanomatous skin cancer) is the strongest risk factor for back pain from bone metastasis. Metastatic disease from breast, prostate, lung, thyroid, and kidney cancers account for 80 percent of skeletal metastases from solid cancers. Approximately 60 percent of patients with multiple myeloma have skeletal lytic lesions present at diagnosis. Typical symptomatology of spinal tumours is that of a progressive, unremitting, localised, or radiating pain, aggravated by movement. Furthermore, it is worse at night and cannot be eased by rest. In addition, patients may experience constitutional symptoms such as weight loss and anorexia.

Infection

Vertebral osteomyelitis is a life-threatening infection of vertebral bones that has increased incidence with ageing. Four aetiopathogenic mechanisms have been identified

1) Haematogenous spread of pathogenic bacteria from distant infection sites Reactivation of Mycobacterium tuberculosis in the bone, secondary to deterioration of the host immunity.
2) Aerobic gram-negative bacilli in older men with urinary tract infection that reach the lumbar spine through Batson's plexus and causing vertebral osteomyelitis.
3) Iatrogenic infection following spinal surgeries or injections.

Similarly, older people are more prone to develop pyogenic spondylodiscitis, which involves the infection of disc and adjacent vertebral bones. Clinical presentation of this is comparable to vertebral osteomyelitis

Visceral disease

Non spinal or visceral diseases can present as low back due to the phenomenon of referred pain. These include dissecting abdominal aortic aneurysm, cholelithiasis , nephrolithiasis, prostatitis, urinary tract infection, and pelvic inflammatory disease.

Mechanical low back pain

This is mainly due to progressive degeneration of the spine. Facet joint osteoarthritis in senior citizens may present as localized LBP with or without posterior thigh pain during walking. The pain may be aggravated during truncal extension, ipsilateral lateral flexion, and/or rotation.

Lumbar degenerative spondylolisthesis (defined as forward or backward slippage of a cephalic vertebra over a caudal one) is common among women aged 60 years or older. This is usually associated with hypertrophy of facets. The presence of degenerative spondylolisthesis alongside facet hypertrophy and thickening of ligamentum flavum may result in pain, spinal stenosis, and neurological deficits in older adults. Ambulation induced pain localised to the calf and distal lower extremity resolving with sitting or leaning forward (pseudo or neurogenic claudication) is a hallmark of lumbar spinal stenosis. Although spinal degenerative changes may induce LBP, not all anomalies on lumbar

imaging are related to LBP because abnormal imaging findings are common among asymptomatic older adults.

Osteoporotic vertebral fracture

Given the hormonal changes following menopause, women are more susceptible to osteoporotic fractures and related LBP. Vertebral fracture incidence increases markedly with age in both men and women. The incidence in women aged 50–54 is 3.6/1,000 person years, which rises to 29.3/1,000 person years in aged 75–79. As compared to patients with non-specific LBP, patients with vertebral fractures experience more disability. Unfortunately, significant proportion of patients are undiagnosed because many seniors assume bone and joint pain as part of the aging process. Older age itself, corticosteroid use, and trauma (e.g. falls), nutritional deficiencies are risk factors for vertebral fractures. The commonest site for osteoporotic vertebral fracture is the thoracolumbar region. These fractures can lead to complications such as radiculopathy, kyphosis and spinal stenosis.

HISTORY AND EXAMINATION

The clinical evaluation of low back pain includes a history and physical examination to evaluate for signs or symptoms that indicate need for immediate imaging and further evaluation. Therefore, inquiring about symptoms which would suggest an underlying significant aetiology is paramount in the assessment. These symptoms are otherwise known as 'Red flag symptoms'. Advanced age (>50 years) itself is a red flag.

Red flag symptoms

- Constitutional symptoms (anorexia, significant weight loss)
- History of malignancy
- Fever
- Recent spinal procedure
- Progressive neurological deficit e.g. bladder and bowel dysfunction. saddle anaesthesia
- Chronic steroid use
- IV drug use
- Recent trauma and recurrent falls

Further detailed analysis of the pain is needed in order to come to a diagnosis i.e. duration, nature, site, severity, progression, aggravating and relieving factors. Pain can be categorised as acute (<4 weeks), Subacute (weeks to 3 months) or Chronic (>3 months).

The clinician should also inquire about associated other medical conditions particularly depression and low mood. Evidence now strongly suggests that psychosocial and emotional factors are better predictors of low back pain outcomes than either physical examination findings or severity and duration of pain. Psychosocial factors that may predict poorer low back pain outcomes include depression, passive coping strategies, higher disability levels or somatisation.

The physical examination is to identify features that suggest the need of further evaluation. rather than to make a primary diagnosis. The physical examination should include the following components.

- Inspection of the spine – for deformity, scars, impending or active herpes zoster infection
- Palpation of spine – Palpation of the back is usually performed to assess vertebral or soft tissue tenderness. Vertebral tenderness is a sensitive, but not a specific finding of spinal infection. This may also be seen in patients with vertebral metastases and osteoporotic compression fracture.
- Detailed neurological examination to identify any deficits. (saddle anaesthesia and loss of anal sphincter tone with diminished or absent reflexes suggest cauda equina syndrome).
- Relevant general examination and other system examination including breasts and thyroid gland.

Table 11.3 - Motor findings in nerve root involvement

Nerve Root	Muscle weakness/ Movement affected	Associated Tendon Jerk
L2	Hip Flexor	None
L3	Quadriceps	Patellar
L4	Anterior tibialis	Patellar
L5	Extensor hallucis longus	None
S1	Gastrocnemius/ soleus/ peroneals	Achilles

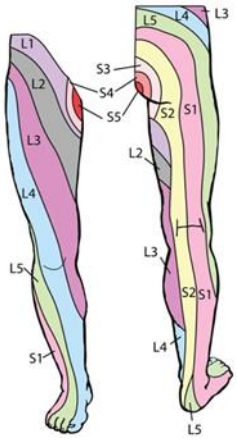

Figure 11.3 - Dermatomes of the lower limb

It may not be possible to define a precise cause of low back symptoms for most patients even after a thorough history and examination.

INVESTIGATIONS

Patients with worrisome or 'red flag' findings on history or physical examination require aggressive evaluation and timely neuroimaging studies of the lumbosacral spine. Laboratory tests may also be indicated e.g. FBC, ESR, CRP, Blood culture, calcium and phosphate levels, serum protein electrophoresis, prostate specific antigen, and alkaline phosphatase etc.

Clinicians should not feel the need to routinely obtain imaging or other diagnostic tests in patients with nonspecific low back pain without any alarming signs or symptoms. Nevertheless, the threshold to order additional investigations needs to be low, as we are dealing with a sub group of patients who are known to have a higher incidence of malignancy and infections.

The main imaging modalities to evaluate back pain are plain radiographs, spinal MRI and CT. The American College of Physicians best practice advice states the following

- Plain radiographs are recommended for suspected malignancy and osteoporotic fracture.

- MRI is recommended in patients with acute low back pain who have risk factors for spinal infection or signs of the cauda equina syndrome.
- MRI is recommended for patients with radiculopathy or spinal stenosis who are candidates for spinal surgery or epidural steroid injection.
- MRI is generally preferred over CT scan for most cases of low back pain. CT scan may help visualize bony abnormalities and is used when patients have a magnetic implant that is not suitable for MRI

The physicians however need to be pragmatic in choosing the imaging modality, considering the available resources in the hospitals.

MANAGEMENT

Management of back pain in older person is always individualised and multi-disciplinary. It depends on the patient's disease condition, severity and type of pain (nociceptive or neuropathic) and most importantly patient factors such as other co-morbidities, other medication and the psychological stability. Older adults are often untreated or under-treated for pain. Barriers to effective pain management include challenges in proper assessment of pain, underreporting by patients and atypical manifestations of pain. It is hence imperative to actively inquire about the pain and to maintain a validated pain scale/score as a continuous assessment in the management.

Pharmacologic Management of Pain

Even though adverse drug reactions in the geriatric population are a significant risk, pharmacologic intervention for pain management is the principal treatment modality for pain. Along with considering age-associated changes of pharmacokinetics and pharmacodynamics, physicians must consider the likelihood of drug-drug and drug-disease interactions. Most mild or moderate pain responds well to paracetamol given around-the-clock. This agent is well tolerated in older patients provided that both renal and hepatic functions are normal. If paracetamol cannot control pain, non-steroidal anti-inflammatory

drugs (NSAIDs) (e.g., COX-2 therapy) may be used as adjunct therapy. This needs to be combined with a proton pump inhibitor to prevent gastric complications.

Despite its efficacy, NSAIDs should be used with caution in the geriatric population due to the high risk of serious side effects. The commonest and most serious adverse effect is GI bleeding due to peptic ulcers. The risk is higher amongst seniors who are already on low dose aspirin or anticoagulants for cardiovascular conditions. It is well described that, longer the duration of NSAID usage, higher the risk of GI bleeding. Out of all the NSAIDs, Indomethacin is known to have the most adverse side effects. NSAIDS best be avoided in older adults with heart failure and the use. of this class of medications in seniors with renal insufficiency, platelet dysfunction, hypovolemic state, hyponatremia, hepatic impairment, warrants close monitoring.

Opioid analgesics are considered for managing severe pain or failed response to other treatments in the older person. Based on compelling evidence, long-term opioids should not routinely be used for chronic pain. If opioids are necessary, they should be used at the lowest effective dose, with ongoing reassessment of risks and benefits. According to the 2019 American Geriatrics Society Beers Criteria, the physicians need to be cautious when prescribing Tramadol since it is known to exacerbate or cause SIADH or hyponatremia. Therefore, monitoring sodium level closely when starting or changing dosages is necessary.

Antiepileptic drugs such as carbamazepine, gabapentin, and pregabalin are mainly used for neuropathic pain. Nevertheless, these should not be combined with opioids as it can cause severe sedation increasing the risk of falls. Muscle relaxants such as baclofen, cyclobenzaprine, and methocarbamol may not be tolerated by older adults due to side effects, including sedation, dizziness, anticholinergic effects, and weakness. Therefore, they should be avoided for individuals age 65 and older.

Non-Pharmacological treatment

The patient and carers need to be educated regarding the disease to remove obstacles that affect implementation and delivery of effective pain management. Nonpharmacological modalities are essential for comprehensive management as it helps the patient in coping better with pain along with improvement in daily functions. These modalities including physical therapy, psycho-behavioural therapies, and social work consultation, which should not be overlooked.

The physical therapy should be always individualised. Physiotherapists need to do a detailed physical assessment of the condition including the posture, balance and risk of falls. He/she needs to concentrate on flexibility and range of motion, strength and muscle endurance, balance, coordination, and agility. Early mobilisation is a cornerstone of physical therapy which not only enhances the recovery process but also prevent complications of long-term immobility in the older adults.

Assessment of risk of falls is important in those having lower back pain situation. Meta-analyses of prospective studies have shown that any bodily pain or joint pain in any location is associated with 40%–71% greater odds of any fall. Home and Environmental approaches aimed at improving individual safety at home, outdoors, and in community and public places can reduce the risk of falls in them. This may include assessment for and the provision of an assistive device, material adaptations (e.g. clearing pathways, fastening carpets, non-slip strips on step edges), behavioural adaptations (e.g. avoiding ladder use) or structural modifications (e.g. installing a skylight to improve visibility)

The final goal of the non-pharmacological treatment is to improve the functional activity, and performance-specific activity. This needs to go parallel with the adequate pain management as persistent pain will hinder patients' participation in regular physical activity.

SUMMARY

Back pain is a common medical problem amongst the geriatric population. Even though most of the cases are due to nonspecific back pain, this particular sub group needs careful clinical evaluation

considering high prevalence of malignancy and infection. Henceforth, special attention is needed for the red flag symptoms. Any senior with a suspected underlying sinister pathology needs urgent further investigations including appropriate imaging. Management is multi-disciplinary and individualised. Careful consideration is needed when selecting the appropriate pain medication. Ancillary therapeutic services are needed to go parallel with pharmacological management to achieve a better outcome.

Further reading

American Geriatrics Society 2019 Updated AGS Beers Criteria; for Potentially Inappropriate Medication Use in Older Adults.

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12. Pain in shoulder region

Dr. Duminda Munidasa, Dr. Rasika Munasinghe

Pain in the shoulder region is an important disability in the geriatric population. Pain in this region may arise due to articular disorders (shoulder, acromioclavicular or sternoclavicular), peri-articular disorders, cervical disease or referred pain from thoracic and sub-diaphragmatic organs as well as from diaphragm. The prevalence of shoulder disorders in patients older than 70 years is around 21% with female predominance. Twenty-five percent and 17% of female and male older persons respectively are affected. Approximately 70% of shoulder associated problems correspond to rotator cuff diseases. Other conditions in the decreasing order of frequency are frozen shoulder, acromioclavicular joint osteoarthritis, rheumatoid disease and Milwaukee shoulder. Cervical spondylosis is a common differential diagnosis of the above. It is important to note that, a definitive diagnosis can be commonly reached through a systematic clinical evaluation by history and examination findings. Examination should include inspection, palpation, range of movement and specific structure related clinical tests. This will spare these elderly persons going through multiple investigations as well as inappropriate treatment.

SHOULDER REGION PAIN EVALUATION

In older patients as in others who present with shoulder pain, history remains a key element in diagnosis. The characteristics of shoulder pain aid to point to the pathology and anatomy. (Table 12.1)

Table 12.1 - Characteristics of the pain

Symptom	***Possible cause***
Radiation up to elbow	Pathology in the shoulder
Radiation below elbow	Pathology in the neck
Pain on lying down / sleeping on shoulder	Adhesive capsulitis or supraspinatus tendinitis of the same side
Pain on bringing the arm down from full abduction	Bicipital tendinitis
Pain on throwing an object	Infraspinatus tendinitis

Pain on top of the shoulder in touching opposite shoulder	Acromio-clavicular joint disease

The presence of constitutional symptoms may indicate neoplasms with secondary deposits in shoulder region, inflammatory arthritis or infections. A past history of injury or prolonged use of crutches may lead to secondary osteoarthritis. A systemic review of symptoms related to cardiac, respiratory or sub-phrenic organs like gallbladder diseases which may give rise to radiating pain in shoulder region also need to be elicited.

Examination of shoulders is best done by standing behind the patient, who should be seated on a chair without arm rests.

Inspection of the shoulder may point to some important disease entities (Table 12.2)

Table 12.2 - Abnormalities seen in shoulder region inspection

Area to inspect	Abnormality	Possible Pathology
Contour of shoulders	Squaring (Figure 12.1a)	Shoulder dislocation
Supraspinatus, infraspinatus and deltoid muscles	Wasting	Long standing shoulder pathology
Delto-pectoral groove	Obliteration or swelling (Figure 12.1b)	Large shoulder joint effusion
Sternoclavicular and acromioclavicular joints	Swelling	Arthritis of these joints
Arm with elbow semi flexed	Popeye sign (Figure 12.1c)	Rupture of long head of biceps

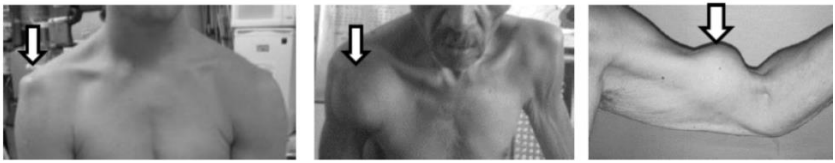

Figure 12.1 a) Squaring b) Obliterated delto-pectoral groove c) Popeye sign

Eliciting tenderness of specific structures around the shoulder joint can be very helpful in diagnosing the cause of shoulder region pain. This will not be possible if there is diffused tenderness in this region. A sound knowledge of surface anatomy to locate these specific structures is an important factor in correctly elicit these signs. The trapezius muscle should be palpated for tenderness before proceeding with the shoulder disorder. While sternoclavicular and acromioclavicular joints can be easily palpated on either side of the clavicle, the anterior margin of the glenohumeral joint is palpated 1 cm below and 1 cm lateral to the coracoid process. The midpoint between the acromioclavicular joint and the upper end of the anterior axillary fold demarcates the locate the palpable area of long head of the bicipital tendon. Tenderness indicates possible inflammation of these structures.

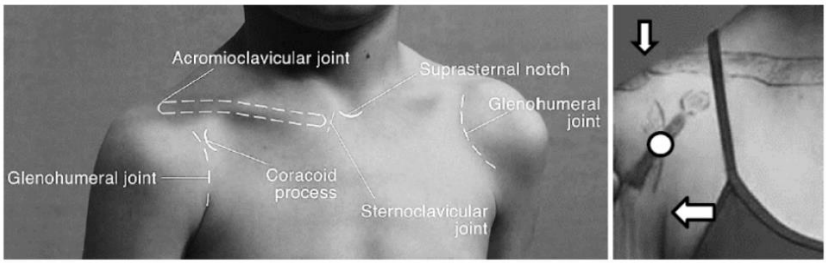

Figure 12.2 - Surface anatomy of the shoulder joint

The range of motion is examined actively (by asking the patient to do full range of movements) and passively (the examiner does the movements of the patient's joints). If both active and passive movements are restricted, it is likely to be due to a joint disease. Restriction of active movement that can be overcome by the examiner to full rage or normal range actively in spite of the pain, indicates a peri-

articular disorder. Examination for the range of the neck movements (flexion-extension, lateral flexion and rotation) is important, looking for reduction of the rage and/or increase shoulder region pain, pointing towards cervical disorder as the cause of pain.

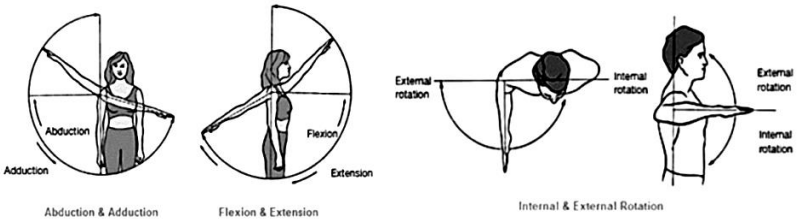

Figure 12.3 - Range of movements of shoulder

When placing one's hand over patient's shoulder on performing passive movements will detect crepitation in the joint indicating the presence of osteoarthritis. A painful arc will be noticed by the patient while abducting the shoulder between 70 to 120 degrees likely in the presence of supraspinatus tendinitis or sub-acromion bursitis.

SPECIFIC TESTS FOR PARA-ARTICULAR STRUCTURES

a) Supraspinatus

- ***Neer's test***

The examiner needs to stabilize the patient's scapula with one hand and passively flex the arm to 1800 while it is internally rotated. If the patient reports pain in this position, then the result of the test is considered to be positive.

- ***Empty can test***

The patient can be seated or standing for this test. The patient's arm should be elevated to 90 degrees in the scapular plane, with the elbow extended, full internal rotation, and pronation of the forearm. This results in a thumbs-down position, as if the patient were pouring liquid out of a can. While examiner is applying a downwardly directed force

to the arm, the patient tries to resist this motion. This test is considered positive if the patient experiences pain or weakness with resistance.

Neer's test		Empty can test
64 – 68 %	Sens	71 %
30 – 61 %	Spec	49 %

b) Subscapularis & Infraspinatus

If a patient reports pain while doing internal rotation and external rotation against resistance of the examiner's hand, while stabilizing the elbow joint against his trunk, it indicates a disorder in the infraspinatus or subscapularis muscles respectively.

c) Bicipital tendon

- ***Speed's test.***

To perform this, the examiner places the patient's arm in shoulder flexion, external rotation, full elbow extension, and forearm supination; manual resistance is then applied by the examiner in a downward direction. The test is considered to be positive if pain in the bicipital tendon or bicipital groove is reproduced.

- ***Yergason's test***

The patient should be seated or standing, with the humerus in a neutral position and the elbow in 90 degrees of flexion. The patient is asked to externally rotate and supinate their arm against the manual resistance of the examiner. Yergason's Test is considered positive if the pain is reproduced in the bicipital groove during the test.

Presence of tenderness while palpating the long head of the biceps is more sensitive for bicipital tendinitis, Speed's test is more specific. Interpreting results of these two test in conjunction would help in diagnosis of bicipital tendinitis.

d) Acromioclavicular joint

- ***Cross-over adduction test***

This test is performed by the motion of forward flexion to 90° with horizontal adduction of the arm across the chest. Reproducible pain over the joint suggests AC joint involvement

INVESTIGATIONS

Since most common disorders of the shoulder can be diagnosed clinically, investigations can be limited to patients who are poorly responding to the initial specific treatment or for those with atypical features. In such an older person ESR, FBS and plain radiography of shoulder, cervical spine and chest should be done initially. These will be helpful in detecting, shoulder pathology, cervical spondylosis, neoplasms in relevant areas as well as diabetes a common associate of shoulder disorders. In an older person ultra sound scan and MRI of the shoulder may not be of great value in further management of shoulder disorders. are done.

CONCLUSION

Shoulder region pain and shoulder disorders are common causes of distress and disability in the elderly population. Correct diagnosis and treatment will relive these patients. History and examination if properly used, can lead to early diagnosis without use of extensive investigations.

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13. Pain in hip joint and pelvic region

Dr. Gunendrika Kasturiratne

Pain in the hip region is a common musculoskeletal complaint of the older adult. Although the patient may point to the hip, the origin of pain may not be in the hip joint itself. It may arise intra or extra-articular to the hip, the lumbar spine, sacroiliac region or as referred pain from the knee. The commonest cause of intra-articular hip pain in the elderly is hip joint osteoarthritis which can present as bilateral or unilateral. Hip joint pain secondary to inflammatory arthritis is not uncommon.

In the older adults the aetiology of acute, subacute or chronic hip pain needs to be appreciated. Furthermore, it can also be categorised as anterior, lateral and posterior hip pain. (Table 13.1)

Table 13.1 - Categorisation of Hip Joint Associated Pain

	Anterior	Posterior	Lateral
Acute	Septic Arthritis Fracture Avascular necrosis	Sacral insufficiency fracture	Fracture of greater trochanter
Sub-acute	Inflammatory Arthritis Iliopsoas bursitis Enthesitis of anterior superior iliac spine	Piriformis syndrome Iliogluteal bursitis Sacroiliitis Radicular pain	Trochanteric bursitis Enthesitis of iliac crest muscle insertions
Chronic	Osteoarthritis Inflammatory Arthritis Paget disease		

CLINICAL APPROACH

A detailed history guides to differential diagnoses prior to commencing the examination. The history along with the presence of typical rheumatoid arthritis deformities, posture of ankylosing spondylitis or

psoriatic rash will further aid diagnosis. Fractures need to be excluded if there is a history suggestive of osteoporosis in a recent history of minimal trauma. (Table 13.2)

A patient with underlying sepsis may present very ill and the hip joint movements will be severely restricted. Examination of the range of movements of the hip joint should include flexion, extension, adduction, abduction and rotations. (Figure 13.1) Usually, the whole range of both active and passive movements are affected in true hip joint arthritis. Enthesitis or bursitis will restrict some active movements only, and passive movements are usually allowed. When it is not genuine hip joint pathology, localized tenderness can be elicited at possible sites of enthesitis or bursitis.

Table 13.2 - Relationship of site of pain and pathology of conditions related to hip

Pathology	**Site of pain**
Arthritis of hip	Groin, buttock, front of thigh to knee
Trochanteric bursitis Gluteus medius tendinopathy	Lateral thigh
Meralgia paraesthetica	Anterolateral thigh
Referred pain from back Sacroiliitis	Buttock
Facet joint pain	Buttock and posterior thigh
Fracture neck of femur Avascular necrosis	Groin and buttock
Polymyalgia rheumatica	Lumbar spine, buttock and thigh

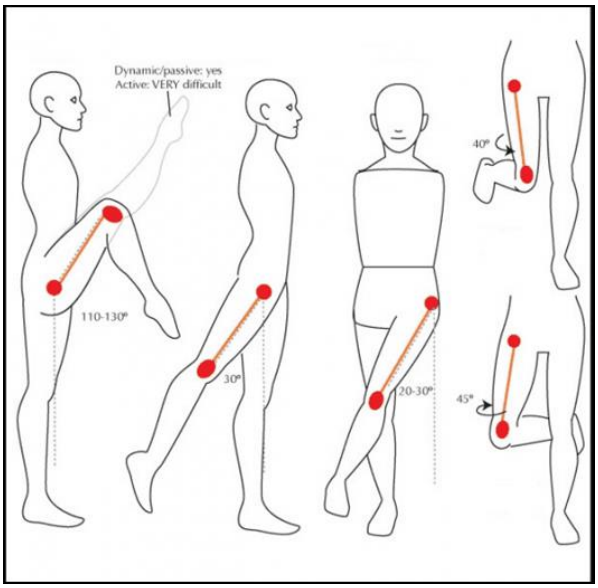
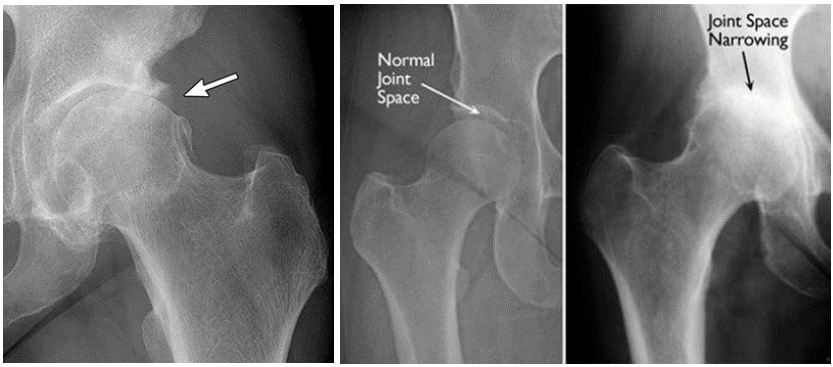


Figure 13.1 – Examination of hip joint movement

OSTEOARTHRITIS OF HIP

Hip pain due to osteoarthritis is common in older adults (affecting 5 – 10 % of the population). Patients will complain of pain in the anterior hip or in the groin which is made worse with prolonged standing or walking. When it is chronic, patients may experience disturbing night pain. Some patients may complain of early morning worsening, lasting for 5 – 10 minutes. Tenderness may or may not be present over the anterior hip. The hip movements will be restricted due to pain.

A plain radiograph could be diagnostic. The superior compartment of the hip joint will be narrowed in the anteroposterior (AP) radiography with sub cortical sclerotic changes. Osteophytes may be present as well. (Figure 13.2)



(a) *(b)*

Figure 13.2 – Radiographs of (a) Osteoarthritis and (b) Rheumatoid arthritis of hip joint

Management of hip joint osteoarthritis includes pain relief, physiotherapy and maintenance of mobility. Hip joint replacement surgery will improve quality of life.

INFLAMMATORY ARTHRITIS

Rheumatoid arthritis of the hip joint is seen in late stages of the disease. The hip is a commonly involved peripheral joint in spondyloarthritis with the onset occurring at an early age below 40 years.

Unilateral or bilateral involvement is seen in rheumatoid arthritis although it is unilateral in spondyloarthritis. The pattern of involvement of the other joints may aid the diagnosis of the type of inflammatory arthritis unless there are atypical new cases presenting late as monoarthritis. Hip pain due to inflammatory arthritis will be worse in the morning associated with early morning stiffness that typically eases with activity. In the older adults these typical features may be absent since they may experience hip pain due to chronic secondary osteoarthritis.

Inflammatory markers will be high in both conditions and rheumatoid factor may be positive in rheumatoid arthritis. Radiography is likely to

be diagnostic as most cases may have a chronic course since the disease onset should have been in younger ages.

In a plain radiograph, there will be concentric narrowing of the joint space in inflammatory arthritis affecting the hip. Cortical erosions are present in rheumatoid hip but may be difficult to visualize in the background of sub cortical sclerotic changes.

Gouty arthritis may involve hip joint in polyarticular forms. Episodic nature, involvement of big toe, the presence of gouty tophi may suggest the diagnosis of gout. Secondary causes of hyperuricaemia may be identified in the medical history. Elevated inflammatory markers and high serum urate can be detected in blood. Since the disease is likely to be at a chronic stage, typical punched out erosions may be evident in x-rays of the hip or other joints. Patients with inflammatory arthritis needs to be referred for management by a rheumatologist.

SEPTIC ARTHRITIS

Hip joint pain when presenting as a monoarthritis could be due to septic arthritis in a susceptible individual. A compromised hip joint due to chronic inflammatory arthritis or a prosthetic hip joint are at a higher risk of developing septic arthritis. Other risk factors include an age above 80 years, long term use of steroids or being on immune suppressant medications, diabetics and having concurrent skin infections.

Septic arthritis should be suspected in an older adult who is very ill and disabled, presenting with anterior hip joint pain and anterior hip tenderness. The patient will find it very difficult to bear weight. Early imaging with ultrasonography and a guided aspiration will be very helpful to get a joint fluid sample for bacteriological studies. Chronic infections like tuberculosis also can involve the hip joint.

FRACTURES

Pain from an undetected impacted or nondisplaced fracture could be a reason for hip pain. A shortened and externally rotated leg is suggestive of hip fracture. Pathological fractures are possible in the older adults

due to primary malignancies or secondary malignant deposits. Any person with a hip fracture following minimal force should be evaluated for osteoporosis. Osteonecrosis due to long term bisphosphonate use is a rare cause of pathological fracture. Sacral stress fractures can occur in osteoporotic females and will give rise to posterior hip pain. Furthermore, a patient with persisting pain in the hip (especially after a fall) despite exclusion of a hip fracture should be considered in having a pelvic fracture which could not be seen in a plain radiography. CT scanning may be valuable in such occasions.

AVASCULAR NECROSIS OF THE HIP

In avascular necrosis of the hip, cellular bone death and resultant collapse of the femoral head occurs due to ischaemia. The commonest cause in the older adult are traumatic fractures of the neck of femur. Non traumatic causes ranges from idiopathy, usage of steroids, smoking, alcohol, HIV infection and sickle cell disease. Systemic lupus erythematosus SLE is also a known cause predisposing to avascular necrosis of hip, although the incidence of active disease in older age group is uncommon.

Non traumatic avascular necrosis is usually of insidious onset with early radiography appearing normal. Later radiography may show well demarcated areas of increased bone density at the upper pole of the femoral head. MRI is the most sensitive and specific imaging modality in detecting this disease with evidence of bone marrow oedema. Early diagnosis and intervention can circumvent the necessity for joint replacement although most patients present late.

PAGET DISEASE

Paget disease is a slowly progressive chronic bone disorder with abnormal rapid bone destruction and reformation giving rise to bone pain, deformities and fractures. The rapidly formed bone is dense but fragile. The disease usually affects localized areas of bone including the hip region. When the skull or long bones are involved, bone deformities may be obviously present. Hearing loss can occur when the skull is involved. The onset is in later life with slight preponderance in male

gender. The initial stages can be symptom free with the diagnosis an incidental occurrence. It is less common in Asian descendants. A hip radiograph may be diagnostic showing the sclerotic areas. Elevated serum alkaline phosphatase with normal calcium and phosphate levels support the diagnosis. A bone scan may be useful to find the extent of bone involvement.

ENTHESITIS

A vast majority of patients complaining of hip pain have enthesitis at multiple sites around hip and pelvic region. This could occur as part of spondyloarthritis or due to acquired postures or overuse. Highly sedentary persons are at a greater risk of developing enthesopathies due to unhealthy postures and overuse. Furthermore, entheses are subject to wear and tear with ageing and are susceptible to injuries.

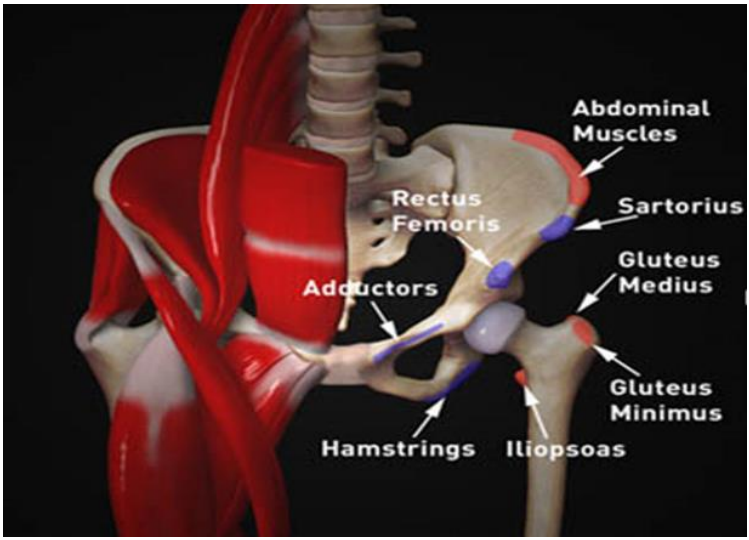


Figure 13.3 – Attachment of muscles related to hip

The pelvic bones and the upper femur have multiple muscle attachments (Figure 13.3). The patient may complain of hip pain due to inflammation at any of those sites of insertions. Enthesitis of a reachable site can be diagnosed by palpation of the localized tender points. It is

difficult to clinically diagnose enthesitis at deep seated sites. The possibility of enthesitis should be considered if the initial hip joint examination with palpation and ascertainment of range of movements is unremarkable. Iliac bone enthesitis can be identified by eliciting localized tenderness. Rotatory movements of trunk will be painful if the iliac crest entheses are involved. The adductor and hamstring muscle contraction may be painful if hip adductor or hamstring insertions are involved. The exact diagnosis is possible by an MRI although it is not recommended as a routine.

PIRIFORMIS SYNDROME

Hypertrophy, spasm or anatomical variation of the piriformis muscle may result in local entrapment of the sciatic nerve, resulting in posterior hip (buttock) pain radiating to the thigh. This is described as piriformis syndrome. Prolonged sitting and climbing steps are known to trigger the pain. Furthermore, it can be elicited by external palpation of the piriformis muscle in most of the affected people. Flexion, adduction and internal rotation at the hip joint tenses the piriformis muscle in reproducing pain. Imaging is not helpful to diagnose the condition. Piriformis syndrome should be a diagnosis of exclusion.

BURSITIS IN PELVIC REGION

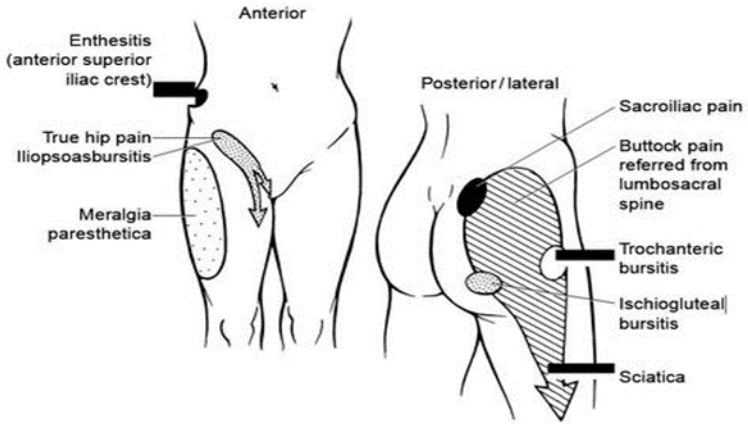


Figure 13.4 – Pain due to bursitis and miscellaneous pathologies of hip

Iliopsoas bursitis can present as anterior hip pain which may mimic a genuine hip pathology. There may be a history of trauma or inflammatory arthritis. The pain is localised to the groin or anterior thigh and is worsened by hyperextension of the hip. Patients prefer to position themselves by flexing and externally rotating.

The ischiogluteal bursa is a deep-seated structure with pain associated with it localising posteriorly. The pain is made worse by prolonged sitting on a firm or hard surface. Tenderness can be elicited over the ischial tuberosity.

Trochanteric bursitis presents as lateral hip pain which worsens on climbing stairs, lying on the affected side on bed and by crossing legs. It is easily elicited by palpating the tender area over the greater trochanter of the femur. It is a common condition in the older adult. A tear in the gluteus medius tendon at its' insertion to the trochanter can also give rise to a similar presentation.

Entheses being poorly vascularised will not respond to NSAIDs and rest, especially if they present late. Steroid injections are commonly used for peripheral enthesitis which shows a good response. However, this option can be used only for trochanteric bursitis in the pelvic region.

MISCELLANEOUS CONDITIONS

Meralgia paraesthetica is a condition where there is burning paraesthesia and numbness in the anterolateral aspect of the thigh. It is caused by entrapment of the lateral cutaneous nerve of the thigh by the inguinal ligament close to the attachment at the anterior superior iliac spine. The condition is brought on by sudden weight gain or by pelvic surgery. Although it is self-limiting usually, amitriptyline and gabapentin may be employed.

Sciatica is caused by impingement of the L4, L5 and S1 nerve roots. This gives rise to pain crossing the gluteal region to the posterolateral leg.

Polymyalgia rheumatica can also give rise to buttock, thigh and hip pain that is worse in the morning.

MANAGEMENT

Specific management will depend on the diagnosis in addition to symptomatic treatment. Orthopaedic or rheumatology teams should be consulted for management planning. Rehabilitation with physiotherapy and occupational therapy, assistive devices and home adaptations are essential components.

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14. The Knee and elbow

Dr. Shehan Silva

THE KNEE

In geriatric population, knee pain is a common symptom that is associated with frequent clinical encounters. Studies demonstrates that the self-reporting annual prevalence ranges between 33-47%. Knee pain is associated with significant restriction of activity of daily living and poor quality of life.

Anatomy of the knee

The knee is composed of an interface of four bones: femur, tibia, fibula and patella. Furthermore, there are 4 compartments namely, medial and lateral tibiofemoral, patella femoral compartments and the superior tibiofibular joint. (Figure 14.1)

The capsule of the joint is supported by the medial and lateral collateral ligaments. Cruciate ligaments are strong binders between the femur and tibia passing in an oblique fashion. The anterior cruciate ligament prevents forward displacement of the tibia on the femur (it becomes tensed on hyperextension). The posterior counterpart resists backward displacement (tensing in hyperflexion). The menisci (semilunar shaped cartilages) are attached to the tibial intercondylar area and the capsule of the joint. The medial meniscus is larger and less curved. The lateral meniscus is loosely adherent and the popliteus tendon intervenes between it and the lateral collateral ligament. Both these cartilages act as shock absorbers

All of these can be subjected to repetitive strain by dynamic movement and static endurance of excess weight, and by physical point injury or disease. In contrast low physical activity and immobility also weaken the knee muscles and reduce blood circulation, leading to diseases themselves. Age related degeneration is accompanied by high level of friction with adjacent tissues and cartilages.

History and Examination

A detailed history is crucial as always. The knee is subjected to trauma and directional force. These can cause laxity of the joint and deformities. A direct blow to the knee causes serious injury. Anterior force on the proximal tibia with the knee in flexion can cause posterior cruciate ligament injury. This happens when the knee impact on the dash board in an automobile accident. The medial collateral ligament is commonly injured by direct forces as in clipping in football. Rapid deceleration and sharp rotations cause sprain or rupture of the anterior cruciate ligament. Sudden twisting or pivoting can cause shearing of the menisci.

The site of pain should be carefully questioned as it can narrow down the diagnosis. Anterior knee pain is associated with the patella. Medial knee pain is commonly caused by medial meniscal tears, medial collateral ligamental injuries and arthritis. Similar pathologies involving the lateral aspect and with iliotibial band tendonitis gives rise to lateral knee pain. Posterior knee pain arises due to formation of Baker's cyst. Pain when descending steps is associated with patella pathologies. Early morning pain with stiffness and gelling signals inflammatory arthritis

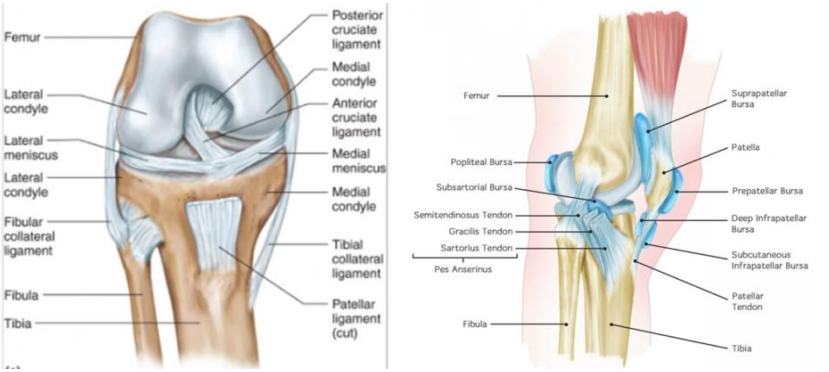


Figure 14.1 - Anatomy of the knee

Patients may complain of locking and catching sensation which may give clues to meniscal injury. Furthermore, a sense of giving away or the knee being unstable upon standing or walking points towards ligamental injury and laxity. Referred pain from the lumbar spine and

hip joint may radiate to the knee. It is therefore prudent to exclude pathologies unrelated to the knee.

In inspection, the presence of diffuse or localised swelling of the knee and the presence of valgus or varus deformities should be noted. Evidence of inflammation may relate to infection (septic arthritis and infected bursa) or even acute flares of gout. The range of movements are restricted in osteoarthritis, knee joint effusion or inflamed soft tissues. Extension is 0◦ whereas flexion is 135◦ degrees in normalcy. The impairment of flexion is demonstrated by the loss of angle or loss of heel to buttock distance while on a couch. The loss of extension is shown by the inability to get the back of the knee on to the surface of the bed.

Special manoeuvres are done to test the ligaments and menisci for injuries and joint stability. Furthermore, gait examination may demonstrate limping due to antalgic gait or even worsening deformities.

Patella tap test is used to confirm the presence of joint fluid. If the effusion is small, the bulge test can be performed by emptying the medial parapatellar fossa by applying pressure of the flat of the hand directing proximally. The bulge refills as the suprapatellar area is emptied. Posterior knee joint cysts (Bakers cyst) can be palpable in the popliteal fossa. An evaluation for effusion is carried out with the patient supine and the knee in extension. The suprapatellar pouch should be milked to determine whether an effusion is present.

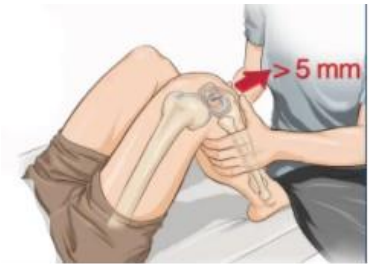
An effusion with rapid onset and tension point to the rupture of the anterior cruciate ligament or fracture of tibial plateau with haemarthrosis. Those that develop slower by 36 hours may have mild to moderate effusion is consistent with meniscal injury or ligamentous sprain. Recurrent knee effusion after activity is consistent with meniscal injury.

Point tenderness should be sort especially at the patella, tibial tubercle, patella tendon, quadriceps tendon, anterolateral and anteromedial joint line, medial and lateral joint lines. The presence of crepitus should be noted during palpation over patella. The Quadriceps angle (Q angle) of

greater than 15◦ is a predisposing factor for patella subluxation. The patellar apprehension test demonstrates an unstable patella; the examiner applies pressure on the patella. The patient may complain of sensation that the knee cap is going to pop out its groove.

a) Testing Cruciate Ligaments

The *anterior drawer* test helps to assess the anterior cruciate ligament. The patient assumes a supine position with the knee at 90. The patient's foot is fixed at slight external rotation by sitting on the foot and then thumbs are places at the tibial tubercle and fingers at posterior calf. With the hamstrings relaxes the examiner pulls anteriorly and assesses for anterior displacement of the tibia. The *Lachman test* is an alternative. While the patient is in supine position the injured knee is flexed to 30. The examiner stabilises the distal femur with one hand and grasps the proximal tibia in the other thereafter attempting to sublux the tibia anteriorly. Lack of clear end point indicates a positive test.



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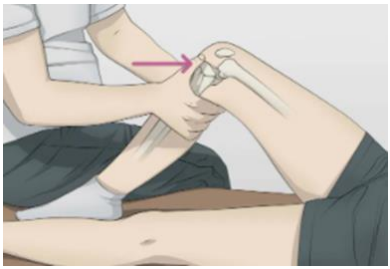

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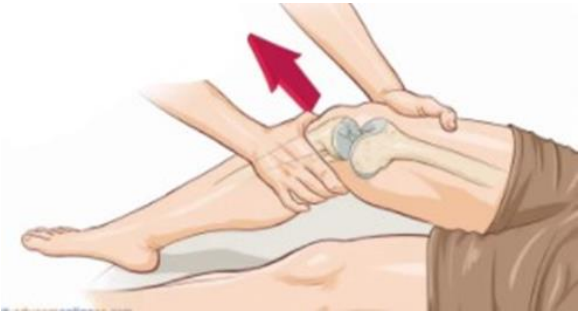

(c)

Figure 14.2 - (a) Anterior drawer test, (b) Posterior drawer test, (c) Lachman test

The posterior drawer test is used to examine the integrity of the posterior cruciate ligament. The patient is positioned as per the anterior drawer test. The examiner then looks for posterior displacement (posterior sag) while standing. Thereafter the patient's foot is fixed in neutral position by sitting on it and displacement posteriorly is applied as per the anterior drawer test.

b) Testing Collateral Ligaments

The medial collateral ligament is assessed by the valgus stress test. The patient's leg is slightly abducted and the examining hands are place on the medial aspect of distal tibia and lateral aspect of knee. Valgus stress is applied to the knee at 0◦ (full extension) and 30◦ of flexion. At full extension the posterior cruciate ligament and the articulation of femoral condyle with the tibial plateau should stabilise the knee. At 30◦ degrees the valgus stress will assess the laxity or integrity of the medial collateral ligament. Similarly, the varus stress test assesses the integrity of the lateral collateral ligament. The examining hands are placed in the medial aspect of the knee and lateral aspect of the distal fibula. Here again, varus stress is applied to the knee at full extension and 30-degree flexion. A firm end point indicates that the collateral ligament is intact. A soft or absent endpoint indicated complete rupture of the ligament (third degree tear).

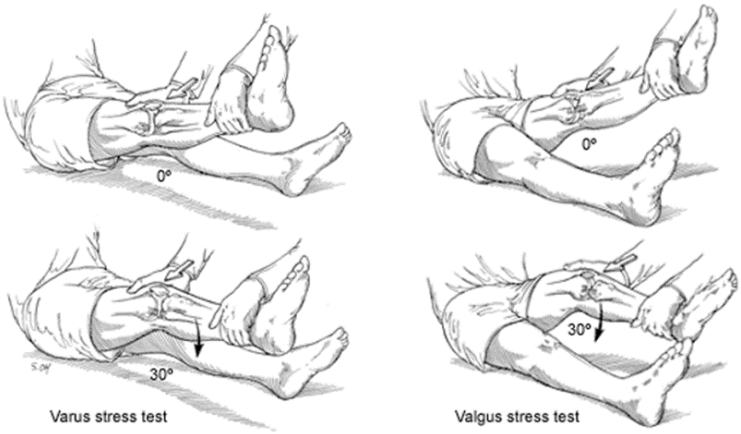

Figure 14.3 - Varus and valgus stress tests

c) Testing meniscii

Joint line tenderness is a nonspecific test for meniscal tears. *McMurray's* test can be employed to detect pathology. The patient lies supine and the examiner flexes the knee to 90 deg. A click felt over meniscus when the knee is brought from full flexion to 90 degrees. The examiner grasps the patient heel with one hand to hold the tibia in external rotation with the thumb at the lateral joint line and fingers in the medial joint line. The examiner flexes the knee maximally to in impinge the posterior horn of the meniscus against the medial femoral condyle. A thud, click or eliciting pain indicates a positive test. The Ege's test also serves this purpose. As the patient squats an audible and palpable click is elicited over the affected meniscus.

In the *Apley's* grinding test, the patient is placed in the prone position. The knee is flexed at 90 degrees. The thigh is kept secure to the bed with the examiners knee. The clinician should then laterally and medially rotate the tibia combined with distraction. Excessive movement, restriction or discomfort is noted. This manoeuvre is then done with compression rather than distraction. If rotation along with distraction is more painful or demonstrated increased rotation relative to normal side, the lesion is deemed ligamentous. If the rotation along with compression is more painful or demonstrated decreased rotation the lesion is most likely meniscal.

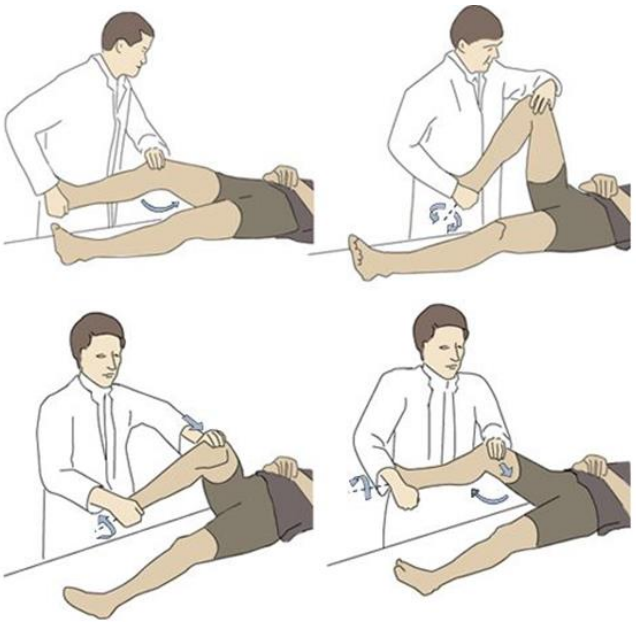

Figure 14.4 - McMurray Test

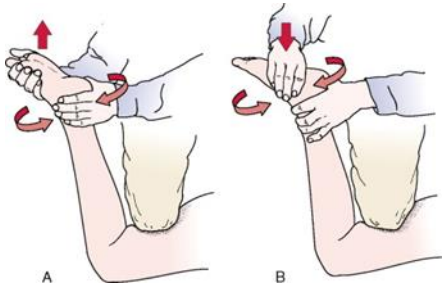

Figure 14.5 - Apley's test a) Distraction b) Compression

Osteoarthritis, inflammatory arthritis, crystal arthropathy and septic arthritis have been discussed elsewhere. Osteoarthritis, inflammatory arthritis, septic arthritis and crystal induced arthropathy are described elsewhere.

Patellofemoral Pain Syndrome (Chondromalacia patellae, runners knee)

There is a vague history of mild to moderate anterior pain in a circular distribution (peripatellar and retropatellar) that usually occurs following prolonged sitting (theatre sign). It may worsen with excessive use and especially by ascending and descending stairs. The exact cause is unclear although overuse is attributed (training errors and overtraining, injury and focal weakness). Although it is common in younger individuals, older adults too can develop this condition. An effusion may exist along with patellar crepitus on flexion. Along with patella tenderness the pain can be reproduced by applying direct pressure to anterior aspect of patella. Patellar tenderness can be elicited by subluxing the patella medially or laterally and palpating the superior and inferior patellar facets. Radiography is not indicated. Treatment requires rest, physical therapy (braces, taping and insoles) and NSAIDs

Patellar Tendonitis

In patellar tendinitis (jumper's knee) there is inflammation of the patellar tendon at its attachment to the lower pole of patella. The characteristic pain is described when the patient jump, go upstairs or do deep knee squats. It is an overuse injury from repetitive overloading of knee. Low angle dorsiflexion, weak buttock muscles and tightness of calf and quadricep muscles and hamstrings are risk factors Usually there is no pain at rest. The condition can be complicated by patellar tendon rupture.

Popliteal Tendonitis

Pain in the posterolateral part of the knee joint nay occur in popliteal tendinitis. Running downhill is a definite risk factor. Other than PRICE and good analgesia and antiinflammation (NSAIDs and steroid injection), therapy in the form of strengthening and increasing flexibility should be carried out.

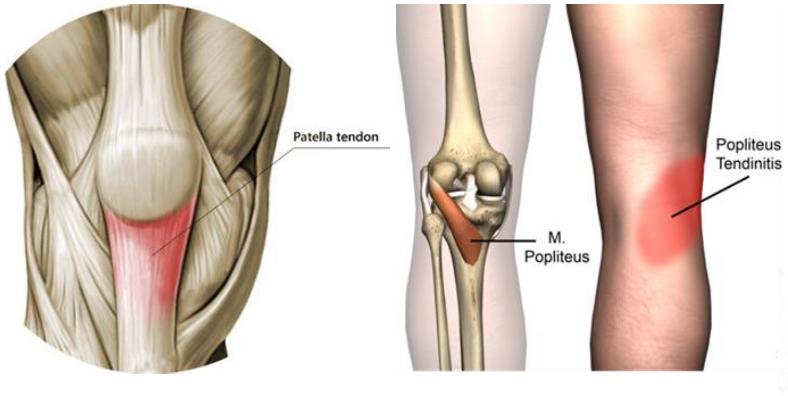

Figure 14.6 - a) Patella tendonitis b) Popliteal tendonitis

Medial Plica Syndrome

In this condition a redundance of the vestigial joint synovium (plica) medially can be inflamed with repetitive overuse. The plica can be caught on the femur or pinched between femur and patella. Acute onset of medial knee pain after marked increase of usual activity point to the diagnosis. A tender mobile nodularity at the medial aspect of knee (anterior to the joint line) may be demonstrated. Radiographs are not indicated. Analgesia, antiinflammation and physiotherapy are advocated in management.

Chondromalacia Patellae

There is inflammation in the posterior aspect of the patella with softening of the cartilage. The cartilage is a natural shock absorber. Overuse and chronic friction can give rise to increased deterioration. This condition although is common in young individuals engaging in sports, can be seen in older adults as well. Sometimes this condition is introduced as patellofemoral pain syndrome. However, the latter is now considered as conditions where there is no cartilage damage. A combination of PRICE, anti-inflammatory medications and physiotherapy are advocated.

Iliotibial band tendonitis

This common condition is seen in those engaging in running or cycling. The iliotibial band stretches from the ilium to the fibula as a thick band. The pain complained to exist at the lateral aspect of knee join is aggravated by activity as above and by running downhill or climbing uphill. Predisposing factors include tightness of iliotibial band, excessive foot pronation, genu varus and tibial torsion. Tenderness is present at the lateral epicondyle of the femur, 3 cm proximal to the joint line. Running must be avoided. Radiography is not indicated.

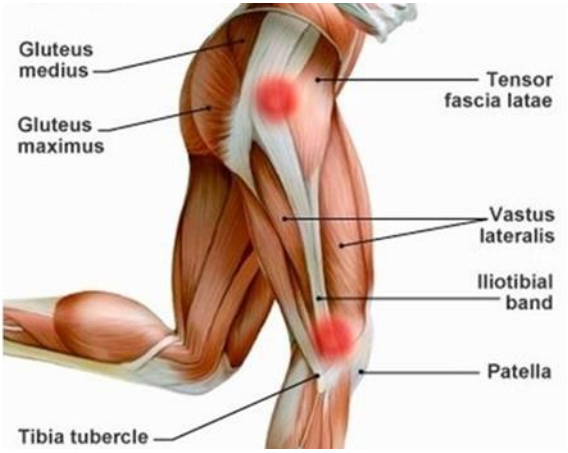

Figure 14.7 - Iliotibial band

Anterior Cruciate Ligament Sprain

ACL sprains are caused by non-contact deceleration forces as when a runner plants one foot and turns in the other way. The valgus tress leads to anterior displacement of the tibia giving rise to sprain or rupture. The patient reports hearing or feeling a 'pop' at the time of injury. Swelling of the knee within two hours after the injury that limits range of motion indicates consequent haemarthrosis following rupture. The anterior drawer test may be positive although the Lachman test should be positive. Radiography is needed to detect tibial spine avulsion fracture. MRI of the knee is indicated as pre-surgical evaluation.

Medical Collateral Ligamental Sprain

A misstep or collision causing valgus stress on the knee followed by immediate pain and swelling at the medial aspect is the characteristic history for this manifestation. Point tenderness at the medial joint line and valgus stress testing of the knee flexed to 30 reproduces the pain.

Lateral Collateral Ligamental Sprain

Although less common than its medial counterpart, this condition results from varus stresses to the knee as when a person plants one foot and turns towards the ipsilateral knee. There is acute onset lateral knee pain (and prompt cessation of activity) with point tenderness in the lateral joint line and positive varus stress testing.

Meniscal Tears

The resilience of menisci in youth gradually decreases upon maturity. Meniscal tears result due to sudden twisting injury of the knee as in sports. They may also occur in association with a prolonged degenerative process. Acutely there will be pain and tenderness in the medial or lateral aspect immediately. There is report of recurrent knee pain and episodes of catching or locking especially with squatting or twisting of the knee. A mild effusion is usually present. There will be tenderness at the joint line depending on the side that is affected. The McMurray test may be positive. MRI is the radiologic test of choice. The immediate treatment of PRICE approach is important. Although arthroscopic repair is practiced in younger individuals, older patients who also have osteoarthritis do not show promising results compared to physiotherapy. Furthermore, surgical intervention may increase the risk of secondary osteoarthritis.

Haemarthrosis of Knee

Haemarthrosis can be caused by trauma and by clotting or bleeding disorders such as haemophilia, sickle cell disease or von Willebrand disease.

Popliteal (Baker's) Cyst

The patient reports of insidious onset of mild to moderate pain in the popliteal region. Furthermore, there is palpable fullness at the media

aspect of popliteal region at the level of the gastrocnemio-semimembranosus bursa. Rupture of popliteal cyst gives rise to calf pain and swelling which may mimic as deep vein thrombosis. Intact large posterior knee cysts can cause venous hypertension of legs.

Bursitis

In prepatellar bursitis (house maid's knee/ beat knee/ miner's knee) there is inflammation of the prepatellar bursa which is found at the front of the knee. There is swelling at the knee with tenderness however without any restructuring of joint range of motion. Infrapatellar bursitis (clergyman's knee) presents with inflammation of superficial or deep infrapatellar bursa. Both these conditions may be brought on by occupations of frequent or prolonged postures of kneeling/ bending. Both conditions are treated with rest, NSAIDs and physical therapy.

THE ELBOW

The elbow can be affected due to epicondylitis or arthritis. Inflammatory arthritis can commonly affect the elbow, although osteoarthritis is rare.

Inspection of the joint may demonstrate the presence of bursa. Rheumatoid nodules may overlie the extensor aspect of the elbow must be examined for defined localised tenderness in epicondyles on resisted movement. Furthermore, an effusion may be discernible in the posterior triangle composed of the epicondyles and the olecranon of ulna. The thumb, 2nd and 3rd finger placed on lateral, medial and olecranon respectively lie in a straight alignment in extended position. It assumes a triangle upon flexion. Incongruence of these points may point to a supracondylar fracture.

Epicondylitis

The two insertion points of the common extensor tendons on the ulnar (ulnar epicondyles) can be inflamed giving rise to epicondylitis. These are commonly known as Tennis Elbow and Golfer's Elbow (Although they do not exclusively occur with regards to these sports). The former occurs at the lateral humeral epicondyle while the latter is seen on the medial bony insertion.

Pain is generated on tensing the relevant muscles. The pain in lateral epicondylitis is seen on gripping an object tightly, carrying a heavy object, shaking hands or opening a door as to create wrist extension. Furthermore, it may radiate to forearm and dorsum of the writs. Medial epicondylitis classically presents as pain on carrying a heavy object or by repetitive activities such as swinging or throwing. The pain is reproduced by resisting wrist flexion and pronation with the elbow being extended. The differential diagnosis of medial epicondylitis includes tears of pronator teres and medial collateral ligament tears and laxity.

Treatment includes rest (for at least 1 month) and early physiotherapy (once pain is controlled) by modalities such as massage and application of ice. A forearm band placed 1-2 inches inferior to elbow aids to diminish the tension on the extensor insertion. NSAIDS can be used for pain control. In severe conditions, local injection of steroids may be helpful at the point of maximum tenderness (with meticulous care in avoiding the ulnar nerve).

Olecranon Bursitis

This is the most common superficial bursitis in humans. It can present either as septic or aseptic manifestation as per prepatellar bursitis. Aspiration is avoided unless diagnosis in certain or to relieve symptoms.

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15. Hand and Foot Region

Dr. Dilrukshi Tennekone

HAND

With the advancement in age, the older person experiences difficulties in hand function and manual dexterity requiring finer movements of hands like gripping, as well as a reduction in power/strength. The causes are multitude (Table 15.1)

These may occur as a result of normal ageing process or due to disease conditions.

Table 15.1 - Factors Affecting Function in Ageing Hands

Intrinsic Factors	**Extrinsic Factors**
Genetic factors	Environmental factors (ultraviolet radiation, chemical irritants)
Endocrine factors	Physical activities (work related, recreational sports, and hobbies)
Metabolic disorders	Nutrition
Diseases (osteoarthritis, rheumatoid arthritis, osteoporosis)	Traumatic injuries
Pathological changes	
Soft tissues (muscles, tendons, blood vessels, nerves)	
Hard tissues (bone, hyaline cartilage, fingernails)	

There is significant reduction in muscle mass (25%-45%) - sarcopaenia of old age. This is responsible for the reduction in muscle strength and is significant beyond 65 years of age. The grip strength in hands are reduced. Hand joints, especially of synovial type undergo morphological and pathological changes common to ageing skeletal tissue. With advancing age, a reduction in cartilage volume, proteoglycan content, cartilage vascularisation and perfusion could occur. These result in narrowing of joint space and marginal osteophyte formation, changes seen in osteoarthritis. Tendons attach muscles to bone and transmit muscle force to skeletal system with limited stretch or elongation. However, they have a very poor blood supply (virtually avascular) near entheses. Tendons in distal palm and digits are enclosed in synovial sheaths that enhances the gliding of tendons. These are thickened in sections to form pulleys. With ageing, aberrations in these systems result in decreased range of joint motion and decreased flexion power (which may cause flexion contractures of overlying joints). Tensile strength is a measure of tendon elongation with tensile testing. It tends to decrease with age giving rise to stiffer tendons.

Arthritis

Degenerative and inflammatory arthritis manifest in hands with characteristic appearance. These are described elsewhere in separate chapters.

Carpal Tunnel Syndrome

Commonest peripheral nerve entrapment is that of median nerve at the wrist, giving rise to carpal tunnel syndrome. The carpal tunnel is formed by the two rows of carpal bones dorsally and by the transverse carpal ligament on the volar aspect. Median nerve that provides sensory and motor supply to the hand is particularly vulnerable to compression at the wrist while travelling through this tunnel. Repetitive flexion-extension movements at wrist, local trauma, swelling of tendon sheaths within the tunnel, stenosis of tunnel due to bone enlargement/fracture, thickening of transverse carpal ligament or any condition occupying the tunnel could be responsible for the entrapment. (Figure 15.1a)

Females are more affected and mostly present with unilateral symptoms. However bilateral involvement is common with the non-symptomatic side having subclinical involvement. Pain & paraesthesia (numbness/tingling) in the distribution of the nerve supply (thumb, index, middle and the radial half of the ring finger) are the presenting symptoms, worse at night and might awaken the patient. These are relieved by shaking the hand or by hanging it down the side. In severe involvement wasting of the thenar muscles could be seen resulting in hand weakness and a tendency to drop objects. (Figure 15.1b)

Patients with mild to moderate symptoms are managed conservatively with NSAIDs, wrist splints and local corticosteroid injections. Those resistant to conservative therapy and those with severe involvement are offered surgery to release the transverse ligament/ flexor retinaculum.

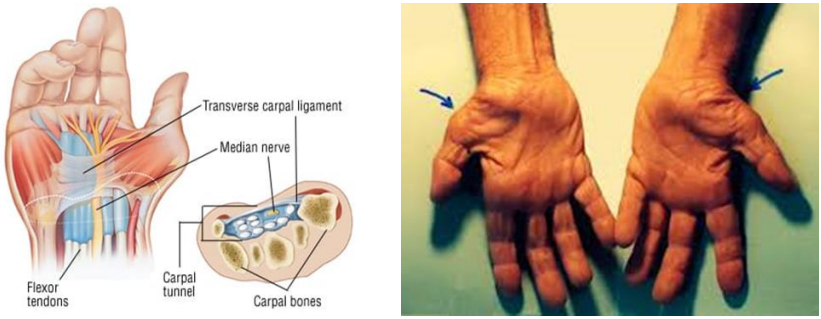

Figure 15.1 - a) The carpel tunnel b) Wasting of thenar muscles in carpel tunnel

De Quervain Tenosynovitis

De Quervain tenosynovitis involves the abductor pollicis longus and extensor pollicis brevis tendons at the first wrist extensor compartment. These tendons are enclosed in a thick fibrous sheath at the level of the radial styloid process. Repetitive movements at the wrist and overuse of thumb leads to thickening of the tendons and sheath giving rise to pain and swelling at the site. Pain is present especially with thumb movements and grasping and there is local tenderness on the radial styloid. Passive stretching of these tendons over the radial styloid in thumb flexion (Finkelstein manoeuvre) as well as resisted thumb

extension and abduction aggravates the pain. Ultrasound scanning could be used to confirm the diagnosis. (Figure 15.2)

Treatment is aimed at reducing the inflammation of the tenosynovial sheath, prevent adhesion formation and prevent recurrence. Ice application, splinting and NSAIDs are used for pain relief. Steroid injections are also used to reduce pain and inflammation and once acute symptoms resolve gentle exercises for the wrist should begin. Frequent recurrences or resistant cases may warrant surgical intervention (decompression of first extensor compartment with or without tenosynovectomy).

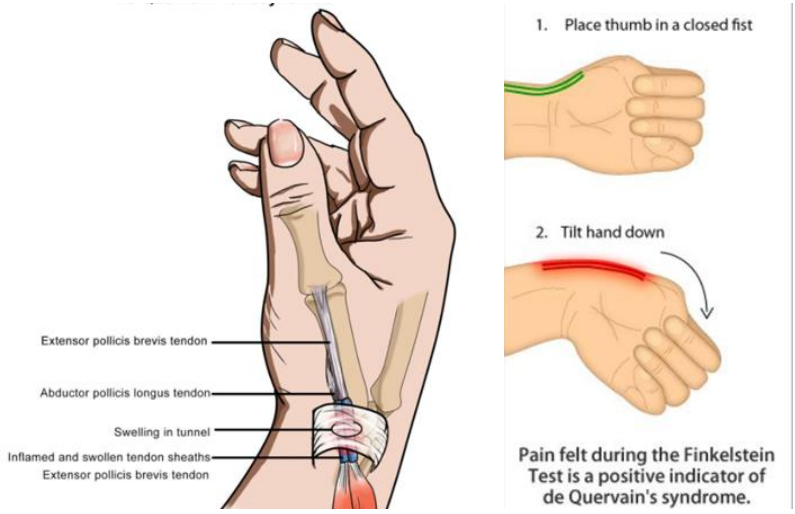

Figure 15.2 - a) De Quervain Tenosynovitis b) Finkelstein manoeuvre

Finger Flexor Tenosynovitis

Inflammation of the fibrotenosynovial tunnel surrounding the flexor finger tendons gives rise to the condition known as finger flexor tenosynovitis or trigger finger. This mostly occurs due to overuse and additionally seen in conditions like diabetes mellitus. The normal gliding of tendon in motion is affected and the tendon gets locked in flexion giving rise to dysfunction and pain. In some multiple fingers could be involved and patients may experience significant stiffness of hands thus mimicking symptoms of rheumatoid arthritis. (Figure 15.3)

Treatment aims at pain relief with NSAIDs, physical measures and stretching exercises. Steroid injections are often used and in resistant cases percutaneous or open surgical release of the tendon is performed.

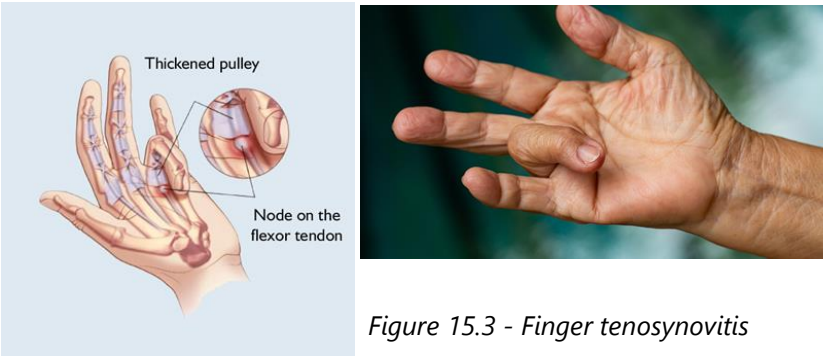

Figure 15.3 - Finger tenosynovitis

Dupuytren contracture

This is a painless progressive fibrosis and shortening of the palmar fascia resulting in contractures of the fingers particularly metacarpophalangeal or proximal interphalangeal joints. Fourth and fifth fingers are mostly affected. This condition is associated with work related overuse, alcohol consumption, diabetes mellitus, smoking, complex regional pain syndrome and malignancy.

Expectant management with observation is all that is needed for those with painless, slow/non progressive minimal contracture without functional impairment. Surgery is reserved for patients with more than 30-40% contractures and significant disability.

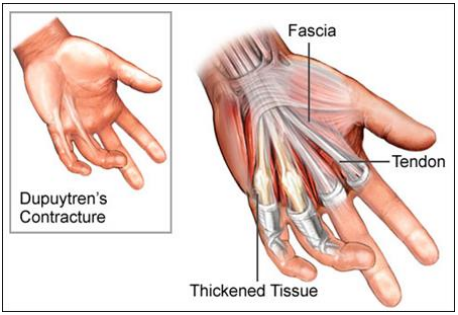

Figure 15.4 - Depuytren Contracture

FOOT

The foot and ankle provide support, balance and shock absorption during standing, walking and running. It is designed for support and locomotion. Ligaments are responsible for connecting the bones together and providing strength. Muscles and tendons are responsible for movements along with maintaining strength in the feet. All these structures undergo changes with age. (Figure 15.5)

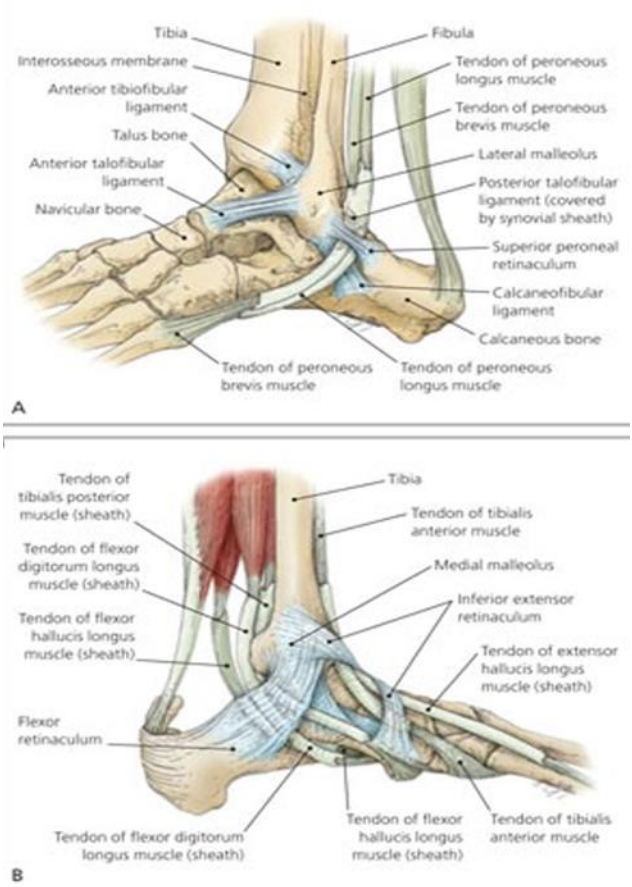

Figure 15.5 - Anatomy of the foot

Arthritis

Degenerative and inflammatory arthritis manifest in feet with characteristic appearance. These are described elsewhere in separate chapters.

Achilles Tendinopathy

The muscles in the leg and foot have powerful and wide insertions to large areas of bone through fascia. This provide strength and stability. The Achilles tendon is the largest and strongest tendon in the body.

Some superficial fibres of this tendon become continuous with plantar fascia. Due to its' large size and immense functional demands, the Achilles tendon is susceptible to a wide range of acute and chronic injuries. Most presentations of Achilles tendinopathy are related to overuse. The risk factors include male gender, systemic glucocorticoid therapy, advanced age and hypercholesterolaemia.

Varying degrees of pain, stiffness and swelling of the heel can be seen depending on the condition (Figure 15.6). In the presence of insertional tendinopathy (enthesopathy) the possibility of an associated spondyloarthritis should be explored. Enthesopathies are commonly associated with a retrocalcaneal bursitis where the bursal swelling is often seen on either side of the Achilles tendon. Tendon ruptures may occur resulting in difficulty of walking and a palpable gap in the tendon. Paratenonitis, tendinosis and tears are common in the mid-third of the tendon. These involvements can be confirmed by ultrasound scanning.

As in all core tendon injuries, the main focus of management is on controlled loading. In acute/ sub-acute conditions the management is based on protect, rest, ice, compression, elevation and support (PRICES) approach. Topical as well as systemic NSAIDs are used in the management along with stretching of gastrocnemius, soleus and hamstring muscles. Attention to biomechanical factors like walking patterns and shoe wear is needed. Glucocorticoid injections have no role in tendinosis as this may weaken the tendon resulting in rupture. Bursitis may however be treated with steroid injections and refractive cases with surgery.

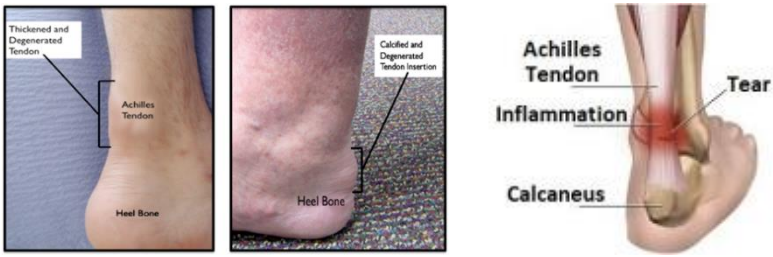

Figure 15.6 - Achilles tendonitis

Plantar Fasciitis

Plantar fasciitis is the inflammation of the insertion of the plantar fascia on the medial process of the calcaneal tuberosity. This condition is linked to overuse. Obesity, prolonged standing and weight bearing are risk factors. Characteristically there is pain in the sole of the foot with the first steps taken after inactivity.

Ultrasound scanning will demonstrate thickening, hypoechogenic changes and oedema of the fascia. Treatment consists of rest, activity modification, splints, footwear modification, NSAIDs and local corticosteroid injections. Surgery is an option in refractory cases.

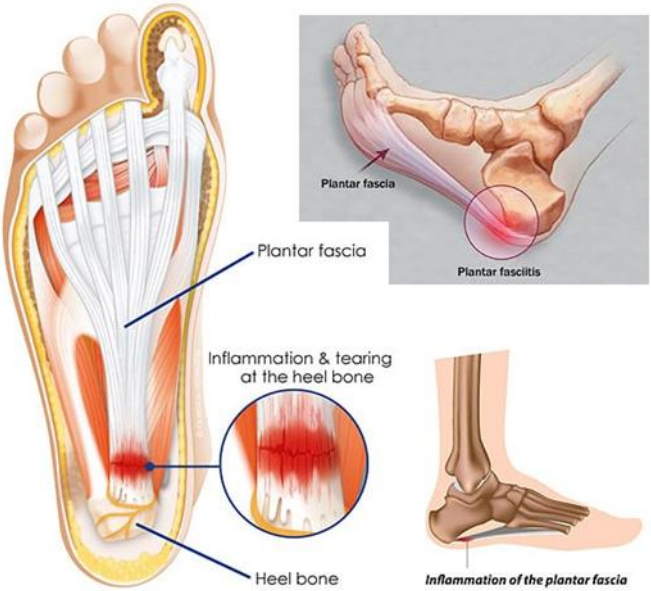

Figure 15.7 - Plantar fasciitis

Peroneal Tendinopathy

Inflammation of the peroneal tendon at the ankle presents as pain and swelling posterior and distal to the lateral malleolus. Pain is present with active eversion and resisted dorsiflexion of foot. Involvement of this tendon may cause lateral ankle instability as well. Management includes

protection, relative rest, ice, compression, elevation, medications and rehabilitative exercise modalities (PRICEMM). (Figure 15.7)

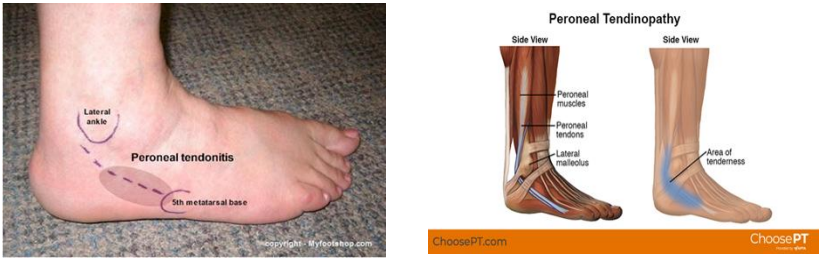

Figure 15.7 - Peroneal tendinopathy

Tibial Tendinopathy

The posterior tibial tendon performs plantar flexion, inversion of the foot, and stabilisation of the medial longitudinal arch. Injury to this tendon can elongate the hind- and midfoot ligaments, especially the spring (calcaneonavicular) ligament, resulting in a painful flatfoot (pes planus) deformity. Pain and swelling is present posterior to the medial malleolus. Worsening of pain with weight bearing and with resisted inversion and plantar flexion of foot may be seen. (Figure 8)

In anterior tibial tendinopathies pain and swelling is present over the anterior aspect of medial malleolus. Foot dorsiflexion may be weakened resulting in a foot drop. (Figure 15.8)

Management is similar to the other tendinopathies.

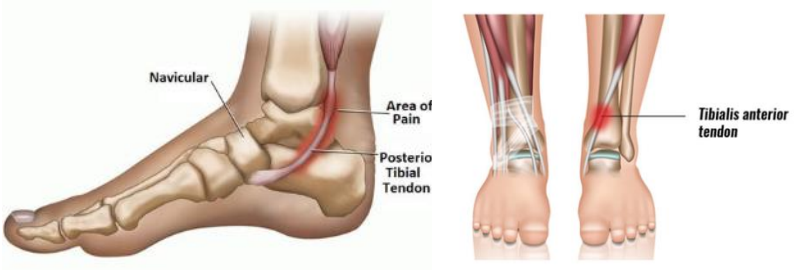

Figure 15.8 - Tibial tendinopathies

Further Reading

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16. Giant Cell Arteritis and Polymyalgia Rheumatica

Prof. Sarath Lekamwasam

Giant Cell Arteritis (GCA) and Polymyalgia Rheumatica (PMR) are the clinical ends of the same disease process and the two conditions can co-exist in some patients. While acute tissue ischemia predominates in GCA, PMR is associated with profound myalgia of girdle muscles. The newer variations of the condition such as cranial GCA and aortic GCA have been recognized. While histology of arterial wall confirms the diagnosis of GCA, no confirmatory test is available for PMR.

GCA, previously named as temporal arteritis or granulomatous arteritis, is an inflammatory vasculopathy predominantly seen in older adults. Although the disease commonly affects medium-size arteries, the involvement of large arteries such as the root of aorta and its major branches are well known. The disease is characterized by the presence of granulomas comprised on T lymphocytes and activated macrophages in the vessel wall leading to the occlusion of affected arteries and tissue ischemia. In GCA, symptoms are mostly due to the ischemia of retina, masseter muscles and the posterior circulation of the central nervous system. The involvement of the subclavian and axillary arteries can lead to ischemia of upper limb while aortitis is associated with dilation of proximal aorta.

The incidence of GCA in Sri Lanka is unknown. Studies have shown the incidence of GCA to vary from 15 to 25 per 100 000 in at-risk populations. The disease is commoner among women compared to men and the incidence increases with advancing age. It is uncommon for people younger than 50ys to develop GCA and when it occurs, alternative conditions such as Takayasu arteritis needs to be considered.

PATHOPHYSIOLOGY

The pathogenesis of GCA and PMR is complex and it involves both systemic component and arterial wall abnormalities. They are a result of immunopathogenic process where inflammatory cells infiltrate the vessel wall leading to structural damage and eventually occlusion of

arterial lumen. The pathogenesis of systemic inflammation, however, is ill understood. It is believed to be a result of highly activated innate immune system.

The cells involved with the pathogenesis of GCA include activated T cells and macrophages and they are responsible for the granulomatous reaction seen in the arterial wall histology. In response to an unknown trigger, T cell activation occurs and activated T cells secrete interferon-(IFN-), a cytokine capable of regulating macrophages. Macrophages under the influence of interferon acquire tissue-injurious properties. The local tissue damage triggers a cascade of reactions including secretion of matrix metalloproteinases, reactive oxygen intermediates, growth factors and angiogenic factors. All these abnormalities lead to the final result, lumen-occlusive intimal hyperplasia.

The disease is associated with an exaggerated acute-phase response which can either precede or accompany the vascular pathology. Furthermore, systemic inflammation can occur without vascular damage, as seen in PMR. Systemic inflammation is mediated through the activated circulating macrophages which release interleukin (IL)-1 and interleukin-6. These cytokines can lead to abnormalities in many organs including liver, central nervous system, bone marrow, and the immune system.

AETIOLOGY

The exact aetiology of GCA-PMR is unknown. Although it is largely considered as an autoimmune disease, there is no evidence that it is directed to a particular component of the vessel wall. As in other autoimmune diseases, many triggers have been found and the spectrum ranges from viral agents (parainfluenza, parvovirus B19, herpes virus) to other organisms like chlamydia and mycoplasma.

CLINICAL PRESENTATION

Although they are the two ends of the clinical spectrum of a single disease, some patients have features of both indicating an overlapping of the two. Studies have shown that nearly 20% of patients with PMR

have certain clinical features of GCA whereas this can be nearly 50% if routine temporal arterial biopsies are performed. Imaging such as CT and PET have shown the presence of large vessel arteritis, mostly asymptomatic, in one third of patients with PMR. The subsets such as cranial arteritis and aortitis may or may not have features of GCA or PMR at the presentation time making the diagnosis difficult.

PMR usually presents as severe pain and stiffness of shoulders and proximal arms. Some patients have evidence of involvement of other regions such as the neck, pelvic girdle and thighs. Nearly half of the patients with PMR have distal manifestations such as peripheral arthritis, hand swelling with pitting oedema and carpal tunnel syndrome. Ultrasonographic evidence of bursitis and tenosynovitis has also been described.

The above symptoms are not characteristics of PMR and can be seen in other diseases as well. Polymyalgic presentation is known in late-onset rheumatoid arthritis and spondyloarthritides. Conditions such as hypothyroidism, myositis, Parkinson disease and some malignancies may mimic PMR: hence requiring a comprehensive assessment at the initial presentation to rule out and closely supervised during follow up.

Patients presenting with PMR should be routinely screened for the presence of large-vessel inflammation and concomitant GCA clinically. Investigations such as temporal artery biopsy and imaging should be reserved for those with features suggestive of GCA or vasculitis. Features of GCA or vasculitis may appear during the follow up of PMR patients requiring higher doses of glucocorticoids. Clinicians should look for the possibility of GCA or vasculitis when a patient with what appears as simple or pure PMR has high inflammatory markers, anaemia or thrombocytosis in addition. An inadequate response to low dose glucocorticoids also should raise the possibility of underlying vasculitis in a patient with PMR.

GCA generally presents with recent onset unilateral headache confined to the temporal region. In addition, claudication of jaw, tongue and masseter muscles have been described. Tenderness over carotid arteries indicates the diffuse nature of the disease. Blindness due to the

occlusion of the central retinal artery is the most feared complication of GCA. It is not uncommon for patients to have mild fever during the course of the illness. Patients in some instances have presented as pyrexia of unknown origin or with focal neurological signs having GCA has been diagnosed based on advanced investigations.

In addition to the classic clinical features, GCA may take atypical form and presenting only with the features of the involvement of extra-cranial territories. In most of these cases diagnosis has been made at post-mortem studies or histological analysis of case series of vascular surgery of the aorta or its main branches. The widespread use of whole-body imaging techniques (PET/CT and CT or MRI angiography) and a greater awareness of the disease in patients with atypical manifestations will probably raise this incidence even more in the future.

CLINICAL FEATURES OF GCA

Typical presentation

- Temporal headache of recent onset
- Scalp pain/tenderness
- Jaw or tongue claudication
- Acute visual deficits
- Non-pulsatile and tender temporal artery
- Anterior ischemic optic neuropathy or central retinal artery occlusion
- Associated constitutional symptoms: fever, fatigue, weight loss
- Associated polymyalgia rheumatica symptoms

Atypical presentation

- Acute ischemic signs and symptoms of extremities
- Focal neurological signs due to ischemia

Non-specific manifestations without evidence of infectious o neoplastic disease:

- Fever including PUO
- Weight loss
- Fatigue/malaise

- Unexplained anaemia

Laboratory findings of GCA include high inflammatory markers, mild normocytic normochromic anaemia, thrombocytosis, high alkaline phosphatase and high serum globulin. Histology of the temporal artery confirms the diagnosis and this should be done before or soon after starting glucocorticoids. The characteristic histological changes may be missed due to the patchy nature of the arterial involvement and undue delay in performing biopsy. Although most patients with temporal-artery-biopsy-proven GCA have no clinical evidence of vascular ischemic manifestations during the acute stage, imaging techniques often show asymptomatic extra-cranial vascular involvement. Patients with GCA have a higher risk of developing aortic aneurysm or dissection especially when other cardiovascular risk factors such as hypertension are present.

The presence of other risk factors of atherosclerosis such as diabetes or hypertension at the time of diagnosis of GCA may enhance the risk of ischemic manifestations of the disease. Isolated extra-cranial GCA remains mostly undetected as the signs and symptoms may be nonspecific (fever, fatigue, weight loss). In addition, patients with acute ischemic manifestations are not routinely screened for the possibility of GCA. Patients with extra cranial vasculitis have more indolent disease with less pronounced laboratory abnormalities including histology. This leads to delays in the diagnosis and treatment.

Temporal artery biopsy is considered as the gold-standard for the diagnosis of GCA. However, the focal or segmental inflammatory changes and practical difficulties in performing the biopsy have limited its use. The sensitivity of biopsy varies depending on the length of the excised specimen, whether it is performed unilaterally or bilaterally and the delay after staring glucocorticoids. The traditional recommendation is to obtain an arterial sample of at least 1-2–cm of length, before or soon after commencing glucocorticoids.

As an alternative diagnostic aid, colour duplex ultrasonography has been proposed. It is a non-invasive and readily available investigation. The diagnostic yield is largely operator-dependent and demonstration

of arterial wall oedema during the acute phase (the 'halo' sign) has a specificity above 90%. Contrast enhanced MRI can demonstrate arterial wall thickening during the acute phase but the positivity decreases soon after the commencement of glucocorticoids. The central bright spot sign (optic nerve head enhancement on MRI) has been proposed as a useful tool to distinguish acute stage GCA from other non-arteritic anterior ischemic optic neuropathies.

MANAGEMENT

PMR usually responds to low-dosage prednisolone (10 to 20 mg per day) within days although the optimal response may take about two weeks. An attempt must be made to taper the prednisolone dosage once the patient is stabilized for two to four weeks. The tapering process is highly individualized and should be done carefully to avoid possible relapse of the disease. A general suggestion is to decrease the prednisone dosage by 1 mg per day each week until most symptoms have resolved. Some prefer to reduce the daily dose by 50% every two weeks. Most patients require treatment for two to three years and patients should be monitored for adverse effects of long-term glucocorticoids. Patients with a clinical diagnosis of PMR whose symptoms do not respond to low-dosage corticosteroid therapy should be evaluated for an alternative diagnosis such as GCA.

Polymyalgia Rheumatica Activity Scale (PMR-AS) has been developed based on five variables to assess the severity of disease: a visual analogue scale for pain from the patient, a visual analogue scale for the physician's assessment, CRP level, duration of morning stiffness (measured in minutes) and the assessment of the ability to elevate the upper limbs. PMR-AS scores less than 7 suggest low disease activity, scores of 7 through 17 suggest medium disease activity, and scores greater than 17 suggest high disease activity. Although this scale has been recommended for monitoring the patient's response to treatment, it is not widely used.

Prednisolone is the first-line therapy for GCA, however requiring higher dosages such as 40 to 60 mg per day to get the disease under control. Patients with visual symptoms require more vigorous treatment with

either prednisolone 80 mg daily or intravenous methylprednisolone. Treatment should not be delayed while awaiting temporal artery biopsy or imaging. Corticosteroid therapy has no effect on biopsy results for up to four weeks after initiation.

Higher dosage therapy should be continued for two to four weeks after remission before a gradual tapering is initiated. Dose reductions should be done carefully while monitoring the disease activity and it may take about 6months to reach low doses (7.5 to 10mg). Alternate-day therapy is not recommended as it may increase the risk of relapse of the disease. Generally, treatment with prednisolone is required for two to three years and relapses usually require stepping up of dosage.

Use of adjuvant drugs such as methotrexate has not been proven effective. Methotrexate has been given both alternatively with prednisolone and as a steroid-sparing drug but both forms have not been proven to have additional benefit.

MONITORING COMPLICATIONS

Patients should be monitored for adverse effects of prednisolone. As GCA and PMR are commonly seen in people above 60 years of age, they should be monitored for adverse effects particularly seen in old age. These include osteoporosis, corticosteroid myopathy, cataracts, bruising, emotional symptoms (e.g., insomnia and depression), hypertension, diabetes and fluid retention.

All patients receiving prednisolone for PMR or GCA should get adequate doses of vitamin D (800-1000 IU/day), and calcium (1200 mg/day). Specific drugs should be prescribed only for those with low BMD or high fracture risk to prevent fractures.

CONCLUSIONS

GCA-PMR is an inflammatory disease with two conditions representing the two clinical ends of the disease spectrum. Although PMR is a milder condition, GCA is associated with sinister complications such as irreversible visual loss and acute arterial ischemia. For better clinical outcome, especially in suspected cases of GCA, early diagnosis and

treatment is required. Close monitoring is needed for disease activity and adverse effects of glucocorticoids during follow up.

Further Reading

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17. Miscellaneous Pain Syndromes

Dr. F H D Shehan Silva

Chronic Pain Syndrome (CPS) is a group of conditions that casts a great deal of morbidity and distress to patient especially the older persons. CPS is considered when there is pain lasting more than 3 months' duration: generally, the time taken for tissue injury healing. Furthermore, there is no clear structural cause (thus differing from inflammatory or degenerative arthritides) or definitive curative treatment. There is coupling of psychological distress, insomnia and altered use of muscles due to fear avoidance or abnormal behaviour.

Although there is no structural abnormality seen the distress is non imaginary or artefactual as there is altered pain processing in the nervous system. A vicious cycle exists in combination of physical and psychological factors which makes management difficult. Patients are anxious and depressed. Furthermore, they are socially isolated or dependent on assistance. Thus, quality of life is severely affected. It is essential that the medical consultation address areas such as employment, physical relationships, dependence, socioeconomic constraints etc. A multidisciplinary effort is essential with a biopsychosocial approach.

FIBROMYALGIA

Fibromyalgia (FM) is a CPS with significant musculoskeletal pain and tenderness. It is seen in female preponderance (3:1 in community and 9:1 in clinical setting) with similar prevalence in all socioeconomic classes (although cultural factors influence therapy seeking behaviour). Approximately 2-8% of population are projected to be affected by the illness although 3/4th may not be diagnosed. This condition is common with advancing age until the 6th decade of life. Thereafter it declines for unknown reasons.

FM can manifest in patients with comorbidities of musculoskeletal system (degenerative and inflammatory conditions), chronic infections, metabolic disease (diabetes, dyslipidaemia) or psychiatric illness. The

corollary of having FM like symptoms in the above comorbidities is encountered as well. The American College of Rheumatology ranks FM as a functional somatic syndrome while the expert committee of European League Against Rheumatism (EULAR) classifies it as a neurobiological disorder. The ICD-10 classification lists it as a musculoskeletal system and connective tissue disorder (M79-7), accepting it as a functional somatic disorder rather than a psychiatric illness. FM has been historically addressed as muscular rheumatism, fibrositis, psychogenic rheumatism and neurasthenia.

This disease is a pervasive and persistent one although there is no degree of degeneration or fatality. Pain and psychological factors (work status, helplessness, education and coping skills) have independent significant relationship to severity and function.

The cause of FM is not known. Se real hypotheses have been stated. Central sensitization describes that patient may have a low threshold for pain. Neuropathic pain and major depression coexist with fibromyalgia due to shared genetic which lead to neurotransmitter sizzling

Clinical Features and diagnosis

Patients present with generalised body aches viz. above and below waist with involvement of both sides and the axial skeletal structures. The pain is often poorly localised, severe in intensity and difficult to ignore. Further, it affects the functional capacity of the individual. Generally, FM is considered when the above description is present for the greatest number of days in a period of 3 months. There is accompaniment of tenderness which is elicited by low intensity stimulation such as skin rolling, thumbnail application to cause blanching (¬ 4 kg/m2) or even measurement of blood pressure (allodynia).

Other symptoms that can be associated with FM include

- Systemic – weight gain, cold symptoms
- Eyes – visual blurring
- Jaw – pain and temporomandibular dysfunction
- Muscular system – muscular spasm, twitches

- Gastrointestinal – nausea, functional bowl disturbances
- Urogenital system – dysuria, dysmenorrhoea
- Nervous system - headaches

Patients with FM may also complain of neuropsychological symptoms such as fatigue, stiffness, insomnia, cognitive affect (impaired concentration – 'fibrofog'), anxiety and depression. These symptoms may be present in similar or greater influence in life. Overall functioning can be affected with inability to conduct usual activities with prolonged time off work.

The American College of Rheumatology required elicitation of 11 out of 18 defined sites to aid diagnosis. A history of widespread pain lasting more than 3 months in all 4 corporal quadrants (above and below waist) are taken into account. However, currently strict categorisation of tender points is not necessary to diagnose as tenderness is a continuous variable. Furthermore, multisite pain and presence of neuropsychological symptoms are taken into account. The revised criteria adopts a widespread pain index (WPI) and symptom severity scale (SS). The WPI is summed up of the 19 body sites with pain. The SS rates the severity of fatigue, unrefreshed waking, cognitive symptoms and general somatic symptoms, each described on a scale 0 to 3, with a. total composite of 12. The revised criteria for diagnosis include

- WPI>= 7 and SS>= 5 *OR* WPI 3-6 and SS>= 9
- Presence of symptoms at similar lever for 3 months and no other diagnosable disorder explainable for pain.

It is of great importance that the clinician target treatment of the comorbidities mentioned above and also treat pain in chronic illnesses so that FM manifest. Treatment of central pain is considered other than treating the peripheral causes itself.

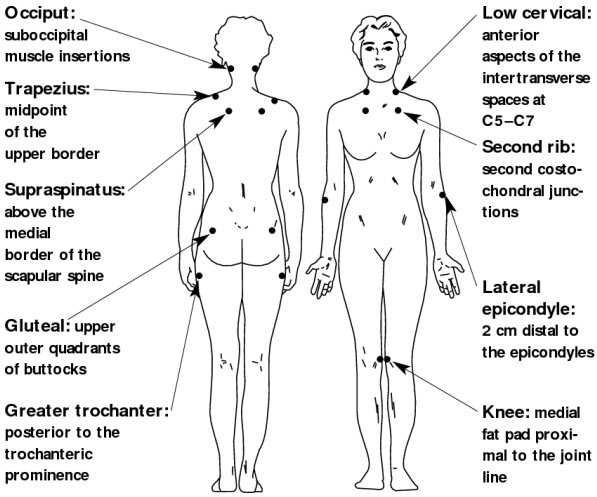


Figure 17.1 a) - 18 Tender points in fibromyalgia

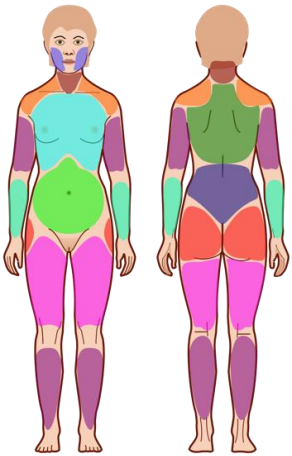


Figure 17.1 b) - Widespread pain index (19 areas)

Differential diagnosis of FM

The following are differential diagnosis

- Inflammatory – Polymyalgia rheumatica, inflammatory arthritis, connective tissue diseases

- Infections – Hepatitis C, HIV infection, Lyme disease, Parvovirus B19 and Epstein Barr viral infections
- Non inflammatory – Degenerative disease, myofascial pain syndrome, bursitis, tendinitis, repetitive strain injury
- Endocrine – Hypo/hyperthyroidism, hyperparathyroidism
- Neurological – Multiple sclerosis, neuropathic pain syndromes
- Psychiatric - Major depressive disorder, generalised anxiety disorder
- Drugs – Statins, aromatase inhibitors

It is essential that the above diseases are excluded when evaluating for FM by careful history taking and examination. Routinely a FBC, ESR, CRP and TSH should be done. The following needs to be done after guided assessment.

- Metabolic panel – FBS/ HbA1C, lipid profile
- Creatine Phosphokinase
- Antinuclear antibodies, rheumatoid factor/ anti CCP, Anti SSA and Anti SSB
- Hepatitis C antibodies,
- Spinal and articular radiography

Red flags of serious disease include

a) History – Fever/ sweats, unexplained loss of weight, morning joint stiffness of more than 30 minutes, new onset Raynaud phenomenon, visual disturbance, dry eyes and mouth
b) Examination – Synovitis, tender small joints, lymphadenopathy, rashes, neuromuscular signs

Management

There is no universally accepted treatment or cure for FM. Adequate symptom control is an essential concept. Integrated pharmacological and non-pharmacological approaches are useful. Explanation of the disease should acknowledge pain, the absence of structural damage, the impact in daily life and a realistic reassurance that symptoms are manageable. Patients may link the illness with a traumatising and emotional events. The clinician rather than confronting this should advise on preventing a trigger event.

Pharmacological Options

Pregabalin and duloxetine are approved by the FDA for management of FM. Although the FDA endorses milnacipran, the European Medicines Agency refused marketing authority.

Antidepressants improve pain, depression, fatigue, sleep disorders and quality of life. A small proportion benefit is seen from SNRIs: duloxetine and milnacipran, and TCAs: amitriptyline. (These are accompanied with more adverse effects than benefits). Amitriptyline takes approximately 3 months while duloxetine, milnacipran and pregabalin take 3-6 months. Rapid cessation may cause withdrawal symptoms. Anticonvulsants gabapentin and pregabalin have shown to reduce pain although it is not possible to predict who would reap benefits. However, patients may experience unpleasant side effects of dizziness, ataxia and fluid accumulation.

Tramadol and other weak opioids are recommended as opposed to stronger agents, by the EULAR. Some reviews suggest the combination of paracetamol and weak opioids over single medication. The action is suggested via serotonin and norepinephrine reuptake inhibition rather than the opioid receptor agonist. NSAIDs should be used when FM is associated with peripheral pain such as osteoarthritis.

Growth hormone in a systematic review for a period of 9 months showed reduction of symptoms by normalising IGF-1. Sodium oxybate, a drug used for narcolepsy and muscle weakness raises growth hormone levels but is not approved by FDA due to concerns of abuse: it is a prodrug of gamma hydroxy butyrate (a date rape drug). Muscle relaxants cyclobenzaprine, carisoprodol with acetaminophen and caffeine, and tizanidine are useful agents. The use of NSAIDs are not considered beneficial. Dopamine agonist pramipexole and ropinirole have shown benefit in a few but can be accompanies by impulse control behaviour. Low quality evidence exists regarding the use of quetiapine.

Non Pharmacological Options

A Cochrane review in 2020 showed that cognitive behaviour therapy may have beneficial effect of reducing pain and distress. Exercise and psychoeducation (including sleep hygiene) along with CBT have shown

added benefits. Furthermore, CBT improves self-efficacy, coping skills with pain, recurrent consultations. There is also improvement of depressed mood.

Mind-body therapy on holistic care of the individual improves physical and psychological well-being and self-empowerment of controlling the disease. Movement therapy (yoga, tai chi), psychological therapy (including CBT), spiritual upliftment (prayer and meditation) and biofeedback (use of technology to give objective measurable quantities of physiological processes) are used.

Physical activity has been shown to improve fitness, sleep, pain and fatigue. Some patient may find cardiovascular exercise effective. High intensity resistance training may improve pain and strength. Aerobic exercise enhances quality of life along with decreasing pain (although fatigue and stiffness are not changed). Combination of flexibility and aerobic training may improve stiffness. Graded exercise programs with small frequent activity building up is employed.

CHRONIC FATIGUE SYNDROME

Fatigue is a common symptom found in multiple conditions. Chronic Fatigue Syndrome (CFS) which is also known as myalgic encephalomyelitis (ME) is a chronic condition with symptoms based on fatigue which is unexplainable by other conditions of at least 6 months. Other synonyms for this entity include post viral fatigue syndrome, chronic fatigue immune dysfunction syndrome and systemic exertion intolerance disease.

CFS is most often seen in ages 40- 60 years, however it is increasingly seen in older adults as well. The severity is not different on gender basis although women are more affected than men. There is no discrepancy between ethnicities and socioeconomic groups. People who are overweight and inactive are more likely to develop the disease. Stress appear to be a factor. It causes a great effect on the quality of life with regards to happiness, productivity and health in the broader sense. It is of sudden onset or insidious development with remission and relapses (which makes the disease more difficult to manage. CFS occurs as a

spectrum. Patients may lead normal lives. In the other extreme of the continuum they may be totally bed bound and completely dependent on care. There may be chronic disabling pain. The functional status may be more intense than multiple sclerosis or end organ disease. Cognitive symptoms occur with deficits in attention, memory and reaction time. Simple as well as complex processing of information can be affected with impaired memory. Perception, motor speed, language, reasoning and intelligence are not affected generally.

Clinical Features & Diagnosis

The Centres for Disease Control and Prevention (CDC) of the United States adopts the following for diagnosis

a) Greatly lowered ability to do activities that were well usual before the illness. *The drop-in activity occurs along with fatigue, lasting six months or more*

b) Worsening symptoms after physical or mental activity that would not have caused a problem before the illness. *The amount of activity that may aggravate is difficult to predict although the decline often occurs 12-48 hours post activity, lasting days, weeks or longer. (Post exertional malaise – Hall mark of the disease)*

c) Sleep problems. *Weariness after full nights of sleep (unrefreshed sleep) or struggling to stay awake (hypersomnia), fall asleep or stay asleep (insomnia).*

In addition, one of the following must be present

i) Cognitive dysfunction (thinking and memory) – 'Brain fog'
ii) Orthostatic intolerance – light-headedness, dizziness, weakness, fainting, vision changes

Many patients may have the following symptoms

- Myalgia, arthralgia (with/without inflammation), headaches
- Tender cervical or axillary lymphadenopathy
- Soreness of throat
- Irritable bowel syndrome

- Chills and night sweats
- Allergies/ sensitivity to food, odours, chemical, light, sound etc.
- Shortness of breath
- Irregular heart beat

A history of antecedent infection with a prolonged fatigue may present.

Patients are usually type A personality (competitive, time urgent, failure hating, hostile/aggressive). They may be depressed and frustrated. However, they do not seek a secondary gain from the symptoms.

Physical examination may reveal no abnormalities although lymph nodes, adrenal and thyroid systems should be assessed. Pure CFS will not have trigger points.

There are no specific investigations although basic tests are advised to exclude the presence of other diseases. Therefore, diagnosis is made by exclusion

Differential Diagnosis

Certain symptoms of CFS may present with treatable illness. Therefore, it is essential that they are excluded in the diagnostic process

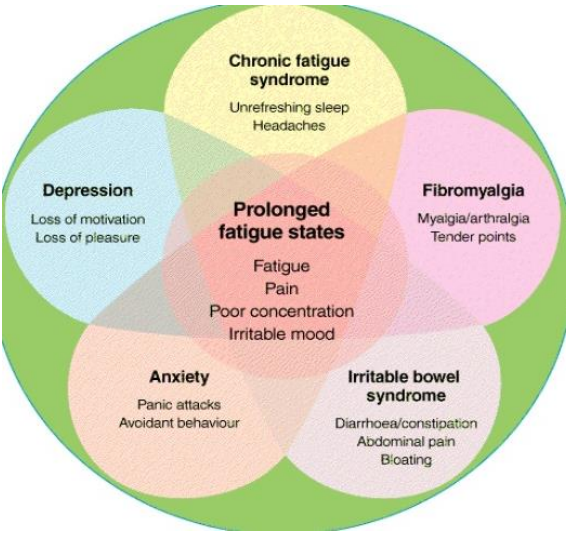
- Infections - Lyme disease, Infectious mononucleosis, influenza, HIV, tuberculosis
- Metabolic - Diabetes mellitus, dysthyroid disease, adrenal insufficiency, Cushing disease
- Connective tissue disease – Lupus, giant cell arteritis, polymyositis/dermatomyositis, Sjogren syndrome, giant cell arteritis/ polymyalgia rheumatic, autoimmune disease, Ehlers Danlos syndrome
- Hepaticological disease – anaemia, lymphoma, occult malignancy
- Psychiatric diseases – bipolar disorder, schizophrenia, delusional disorder, dementia, eating disorders
- Neurological diseases – Parkinsonism, multiple sclerosis, obstructive sleep disorder

- Toxicological diseases – Alcohol/ substance abuse, heavy metal poison
- Medication – Statin induced myalgia

The following red flags of serious illnesses are important

- Localising/ focal neurological signs
- Signs and symptoms of inflammatory arthritis/ connective tissue disease/
- Signs and symptoms of cardiorespiratory disease
- Weight loss and anorexia
- Sleep apnoea
- Clinically significant lymphadenopathy

Patients with fibromyalgia may have muscle pain, severe fatigue and sleep disturbance. The presence of extensive tender points and allodynia differentiates the former. However, the two diseases may coexist. CFS can also coexist with depression, anxiety and irritable bowel syndrome.



Management

It is essential that the physician connect and maintain a good rapport with the patient. Considerate listening is a key ingredient Decision making should be shared between the patient and clinician. The relationship should support and foster attentive care and reassurance. The physician should acknowledge and explain that the symptoms are real but nor malingering. Debate of the concept of mind over matter needs to be avoided although primary or secondary depression and/or anxiety needs to be addressed while maintaining trust. Furthermore, explanation of the variability of symptoms needs to be discussed. The patient needs to be the centre of care and care planning. Family members and caregivers should be also involved and educated

Symptoms need to be addressed promptly. Simple analgesics leading to neuroleptics, antidepressants and sedatives may be required if in the presence of related symptoms. Pharmacological methods need to be regularly reviewed for their efficacy and potential side effects

Sleep hygiene is essential with no excess or deficient sleep hours. Daytime sleep should be avoided. Half hour rests at a time are permitted. Relaxation techniques such as yoga, massage and complementary medicine may have a role. Healthy diet with regularised meals and good hydration is a must.

A graded exercise program should be adopted at a comfortable level for the patient with the help of a sports medicine specialist/ therapist. Too much rest is counterproductive in terms of cardiovascular and muscular deconditioning. A deconditioned patient who likely commence or improve activity may encounter unpleasant experience. Patients should be advised not to do more/ overdo exercise as it may lead to a relapse and reinforcement of the idea that it is harmful for the body. Patients who have all or nothing phenomenon who attempt to do extra on a 'good day' may relapse, requiring rest for longer period, vacillating pattern to a general downward trend.

Many therapies have been tried in CFS. However cognitive behavioural therapy and graded exercise demonstrate significant benefit which should be commenced by therapists. Cognitive behavioural therapy is

a collaborative treatment approach to reduce symptoms, disability and distress. Graded exercise therapy aims to improve symptoms and functioning by increasing activity in a custom made approach.

Fitness for work and recreation should be advised. Liaison with relevant third party individuals and agencies may require.

CHRONIC (WORK RELATED) UPPER LIMB PAIN SYNDROME

This entity is also known as Repetitive Strain Injury. The patient complains of pain in one or both upper limbs, confined to a part or that of the whole entity. It is seen in musicians and those working with computer keyboards, synchronous with stressful environment including shortage of staff, workplace conflicts or change of work practice. Pain may originate as a muscular type of neck pain or as a defined entity such as carpal tunnel syndrome. As time goes on the pain generalises to the whole limb and is not related to work.

This disease causes severe distress: treatment of regional pain entities should be therefore targeted so that chronicity is not achieved. Adequate analgesia with NSAIDS and physiotherapy is essential in the early phase. Medication such as pregabalin and amitriptyline are useful. A brief period out of work with gradual introduction to the work environment is essential. Adaptation of work environment to optimum conditions (ergonomics) is useful after consultation of an occupational therapist or other specialists. These include proper positioning of chairs, desks and computer peripherals, and improvement of techniques (e.g. positioning in playing an instrument)

TEMPOROMANDIBULAR PAIN DYSFUNCTION SYNDROME

This syndrome is seen among anxious persons giving rise to unilateral or bilateral temporomandibular joint pain. There is associated nocturnal bruxism (grinding of teeth) or masticatory abnormalities. Dental pathologies such as caries need to be attended to if detected (however unnecessary dental treatment may worsen it). There is a place for low dose antidepressants and benzodiazepines.

COMPLEX REGIONAL PAIN SYNDROME

Complex Regional Pain Syndrome (CRPS) is a neuropathic entity with abnormal sensory (hyperaesthesia), motor or autonomic (abnormal blood flow and sweating) disturbance. Trophic changes may be present. It is not essential that all of these features be present. There are two entities. CRPS I (previously known as Reflex Sympathetic Dystrophy Syndrome) do not show a definable nerve lesion. In comparison, CRPS II (previously known as causalgia) will demonstrate a definite lesion such as injury to a nerve trunk. CRPS is seen after a trauma or a central nervous system pathology such as a stroke.

The pain is disproportionate to time and intensity of the initial pathology and hypersensitivity to non-painful stimulus is present (allodynia). The neurological manifestations are not present in one single nerve distribution. At first there is hyperthermia and swelling in the region which may be difficult to identify although it may be reversible. This is followed by a phase of painful dystrophic changes with articular stiffness, cold skin and localised osteoporosis. Advanced CRPS is associated with skin and muscle atrophy giving rise to disabling contractures.

Diagnosis of CRPS requires a high index of clinical suspicion due to the presence of unusual pain distribution. The Budapest Consensus Criteria requires 2 signs in 4 categories: sensory, vasomotor, sudomotor/oedema and motor/trophic. There are no specific investigations although regional osteopaenia can be visualised in radiography. Management of a patient is often difficult. Adequate analgesia with NSAIDS coupled with tricyclic antidepressants, serotonin-norepinephrine reuptake inhibitors and pregabalin or gabapentin is used. Pharmacotherapy needs to be coupled with adequate physiotherapy. Bone strengthening medication need to be considered. Patients with uncontrollable pain should be referred to specialist pain clinics.

MUSCULOSKELETAL CHEST PAIN

Costochondritis is well known to affect the third, fourth and fifth costochondral junctions mainly. It is seen more in women. Tietze syndrome is described by some as costochondritis in absence of swelling. Costochondritis is also seen with rheumatoid arthritis, ankylosing spondylitis etc. Older patients may have xiphoidalgia and slipping rib syndrome (involving the tenth rib). Malignancies of breast, prostate and plasma cell cytoses and sarcomas can give rise to metastasis producing similar symptoms.

MYOFASCIAL PAIN SYNDROME

This entity is characterised by numerous regions of localised musculoskeletal pain and tenderness with multiple tender points. The pain is described as a deep pain or an ache with burning sensation. It is followed by overuse, by static contraction (e.g. reading, writing or typing in prolonged position) or by trauma. Tenderness can by elicited at the middle of a muscle belly and palpation can give rise to a twitch.

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18. Bone and joint Infections

Dr. Chandana Karunatileka

SEPTIC ARTHRITIS

Infections of bones and joints can increase mortality and morbidity in older person by irreversible joint destruction and sepsis. In older persons, the diagnosis of infective arthritis is complicated by atypical clinical manifestations. A variety of coexisting factors complicate the antibiotic usage and surgical intervention. Early intervention is essential as there is poor physiological reserves due to related comorbidities. Delayed or inadequate treatment of joint sepsis can lead to rapid and irreversible joint damage. The mortality associated with septic arthritis and subsequent sepsis is approximately 11%.

A hot swollen joint is a common medical emergency. The differential diagnosis can range from acute rheumatoid arthritis, trauma, inflammatory arthritis to septic arthritis which is the most serious problem is septic arthritis. Septic arthritis is defined as joint infection caused by the presence of pathogens in the joints, which can be infected directly or indirectly through blood-borne transmission.

Clinical Features

The diagnosis of septic arthritis is a clinical diagnosis that is accompanied by analysis of joint aspirate. A recent systematic literature review showed that clinical diagnosis is the gold standard while laboratory investigations are supportive. Patients with joint infections usually have a triad of fever (40-60%), pain (75%) and impaired range of motion. These symptoms may last from a few days to a few weeks. Fever is usually low grade (<102°F) and is not the basic symptom of diagnosis. However, symptoms of general malaise and discomfort may appear more commonly. Knees, hips or large joints are affected in up to 60% while smaller joint involvement is less common.

In patients with prosthetic joint infection, septic arthritis present with a prolonged low-grade course accompanied by gradually increasing pain. Early prosthetic joint infections are characterized by high-grade fever,

focal swelling, redness, cellulitis and the presence of a draining sinus tracts. Most of the late prosthetic joint infections are usually secondary to bacteraemia due to some other cause. Radiography may show evidence of peri-implant loosening. The ability of microorganisms to produce a biofilm around the implant is an important factor to understand in the treatment process. A biofilm is formed by the bacteria, to change their behaviour in response to antibiotics and host immune system. The pathogen may go dormant and hide for long periods of time.

Aetiology

Infections can be divided into bacterial and fungal infections. Bacterial infections can be further divided into Gram-positive or Gram-negative and as mono-bacterial or multi bacterial origin. Culture-negative infections create specific problems in diagnosis, treatment options, and patient compliance.

Staphylococcus aureus and *Streptococcus sp.* are the most common pathogens. *Staphylococcus epidermidis, Staphylococcus aureus, Pseudomonas aeruginosa* and *Escherichia coli* are usually isolated in patients with chronic implant-related infections. Hospital acquired methicillin resistant *S. aureus* (HA-MRSA) could be a causative agent among older persons' patients with lengthier hospital stay, increased comorbidities and previous antibiotic exposure. More recently, strains of community-acquired MRSA have been emerging as a causative agent. Gram-negative bacilli infections are also common in the older adults. Risk factors for Gram-negative infections include recent urinary tract infections, catheterisation, and immunosuppressive therapy.

Immunosuppressed patients are at risk of developing atypical infections and may have different presentations compare to normal immunity. Tuberculosis and fungal infections are rare. However, they can present among the immunosuppressed patients with complications and non-healing wounds. Newer diagnostic methods can help to identify pathogenic bacteria and treat them effectively in the case of negative cultures.

Diagnosis

The diagnosis of septic arthritis is based upon exclusion of the differential diagnosis. A thorough medical history should help to rule out traumatic events, rheumatism, crystal arthropathies and viral arthropathies.

The presence of an acute or past history of joint pains in other none affected joint supportive to exclude the possibility of degenerative joint disease, such as osteoarthritis and chondrocalcinosis, which is more common among the older persons. In the case of underlying polyarticular diseases (inflammatory diseases and degenerative diseases), the infected joints usually show symptoms which are more out of proportion to the background disease in the non-infected joints. It is generally believed that septic arthritis affects only single joints, but there is evidence that as many as 22% of cases involve multiple joints.

Ageing itself is a risk factor for the development of septic arthritis. The lack of typical symptoms and signs of infection, diagnosing an infection in the older persons may be a challenge. In the older adult the body tends to mount a less temperature response to pathogenic insults and so minor temperature elevations may indicate significant bacteraemia.

The clinical changes caused by infections in the older persons are usually subtle and non-specific. Signs that may indicate sepsis include changes in mental status, cognitive impairment and functional deterioration, and systemic features such as anorexia and weight loss. Other risk factors for the development of septic arthritis are

- Rheumatoid arthritis or osteoarthritis
- Prosthetic joints
- Abnormal joint structure
- Intravenous drug abuse
- Diabetes mellitus
- Previous intra-articular injection of corticosteroids
- Skin ulcers
- Application of oil and other pastes (herbal) to the joints which precipitate inflammation.

If septic arthritis is suspected, joint aspiration and subsequent microscopic analysis and synovial fluid culture must be performed before starting any antibiotic treatment. The synovial fluid is screened by polarized light microscope for crystals and Gram stain is used for bacterial screening (sensitivity 63 - 96%). Joint aspiration is performed under strict aseptic conditions to prevent the introduction of any infection in to the joint.

Macroscopic appearance of synovial fluid may show cloudiness and turbidity suggest an infection. Infected joint fluid is typically turbid, cloudy, yellowish or yellow-green in colour and viscosity is increased. Microscopic assessment will demonstrate elevated levels of nucleated cells and demonstrating a predominance of polymorphonuclear leukocytes. Gram staining and microscopy of synovial fluid will only reveal the presence of organisms in up to 50% of cases of septic arthritis. Subsequent culture of the joint fluid and concomitant blood cultures will increase the diagnostic accuracy, but there still remains a proportion of cases of joint sepsis where no positive microbiological proof is available. It is important to note, however, that septic arthritis and crystal arthritis can coexist. The presence of one diagnosis does not exclude the other.

The only absolute contraindication to needle aspiration of a swollen joint is the presence of a prosthesis.

In such a circumstance, referral to an orthopaedic surgeon is recommended so that arthrocentesis can be performed under aseptic conditions inside an operating theatre. Warfarinisation and the presence of overlying skin cellulitis are relative contraindications.

Normal ranges of WBC, ESR or CRP will not exclude the diagnosis of septic arthritis. They should be used in conjunction with the clinician's level of clinical suspicion.

Management

The focus of the treatment of septic arthritis is the adequate draining of infected synovial fluid, appropriate antibiotic treatment and to immobilize the joints to control the pain. In native joint infections,

parenteral antibiotics are usually required for at least 2 weeks. Methicillin-resistant Staphylococcus aureus (MRSA) infections require at least 4 full weeks of intravenous antibiotic treatment. In acute prosthetic joint infection intravenous antibiotics without surgical interventions can be employed, if it is an early type or secondary to blood-borne spread, without any evidence of soft tissue involvement and without any evidence of joint instability.

Pharmacological Treatment

A meta-analysis of a study of antibiotic treatment for joint sepsis did not draw clear conclusions about the clinical or bacteriological superiority of one regimen over another. In the older adults many other factors complicate the decision on the type of antibiotic. Antibiotic selection must be made with caution, considering the potential polypharmacy and drug interactions, decreased renal reserve and age-related changes in both pharmacodynamics and pharmacokinetics. The creatinine clearance and estimated glomerular filtration rate (eGFR) are useful measure of renal function evaluation before begins the antibiotics.

It is prudent to start the medication with the minimum dose and have clinical benefits. Current drug treatments should be reviewed, and any unnecessary drugs should be stopped or replaced with safer alternatives. However, it is always wise to discuss the choice of antibiotics with the microbiologists in the hospital, who can provide guidance based on local demographics patterns. The antibiotic treatment regimen should be adjusted based on the results of the synovial fluid and blood cultures, which may available within a few days of the presentation.

- In all cases of suspected acute septic arthritis and acute osteomyelitis, blood cultures should be obtained before commencing antimicrobial therapy.
- Joint aspirates or bone biopsies should ideally be obtained for culture.
- Empirical antibiotic therapy should be reviewed according to sensitivity test results.

- In chronic osteomyelitis bone biopsies should be obtained before commencing antimicrobial therapy.

Table 18.1 - Empirical and Prophylactic Use of Antimicrobials in Bone and Joint Infections (Sri Lankan National Guidelines 2016)

Condition	Primary therapy	Alternative therapy	Comments
Acute septic arthritis (Non prosthetic joints)	flucloxacillin/cloxacillin 2g IV 6 hourly or ceftriaxone 50-80mg/kg IV once daily In adults if an infection with a Gram-negative organism is suspected add cefotaxime 2g IV 8 hourly or ceftriaxone 2g IV daily	In immediate penicillin or cephalosporin hypersensitivity clindamycin 600mg IV infusion 6 hourly or vancomycin 1g IV infusion (over 100 minutes) 12 hourly or teicoplanin 400mg IV 12 hourly for 3 doses then 400mg IV daily	Acutely infected joints may require washouts in addition to antibiotics. Initial empirical therapy should ideally be guided by Gram stain results. Adjust therapy according to culture and susceptibility results. Renal function should be monitored with vancomycin therapy.
Prosthetic joint infection			No empirical treatment. Antibiotic therapy should be guided by sensitivity of culture isolates. Multiple samples should be collected for culture during surgical procedures. Duration is from weeks to months depending on the management plan and causative organism. .

Synovial fluid drainage by surgical intervention

The infected material must be removed from the inflamed joint. Arthrocentesis can be achieved through arthroscopy or open arthrotomy or through closed needle puncture. The literature review did not find any evidence to solve the superiority of these methods. The initial repeated needle aspiration may be sufficient to prevent a large accumulation of infectious fluid. If frequent drainage is required, surgical drainage is more effective.

Indications for surgical drainage include the following

- The appropriate antibiotic and percutaneous drainage fails to clear the infection after 5-7 days.
- The infected joints, which are difficult to aspirate (e.g. hip)
- When adjacent soft tissue infected

Arthroscopic drainage is preferred over the open arthrotomy.

Joint immobilisation and physical therapy

It is not essential to immobilize the infected joint unless the pain is severe. The patient should not bear weight until the clinical signs and symptoms of synovitis disappear. The initial passive range of motion helps to maintain the functional range. If the patient responds to treatment, gentle mobilisation of the joint. Most patients require active physical therapy to achieve the maximum range of motion.

Surgical intervention of artificial joint infection

For patients with prosthetic-joint infection within 30 days after implantation or prosthetic-joint infection within 3 weeks after the onset of symptoms, open arthrotomy and joint irrigation is required. If the prosthesis looks well fixed and in the absence of wound sinus tract, prosthesis preservation should be considered. In advanced cases, removal of the prosthesis, thorough joint irrigation, wound debridement and antibiotic treatment for 6 weeks is essential. Revision arthroplasty can be considered when all the inflammatory and infective parameters are normal. The usage of antibiotic impregnated cement is vital. It will improve the antibiotic diffusion into the surrounding tissues.

OSTEOMYELITIS

Spontaneous osteomyelitis is not common among the older patients. Older patients are more likely to have chronic osteomyelitis due to previous injuries, wounds, or diabetes infections. Osteomyelitis may linger and be dormant for 20 years or more: bacteria form a biofilm and remain inactive. Reactivation of bacteria can cause natural drainage after a few years. An old open fracture or a gunshot wound are examples. Diabetic foot infection is the most common cause of osteomyelitis in the older persons. Lack of protective sensation can lead to neuropathy and subsequent ulcers at pressure points. Subsequent infection of the toe or foot can occur. The rate of diabetic foot infections is estimated to be 0.3% per year.

The most common site of osteomyelitis is in the foot (43%), followed by the femur and tibia (20%). Bones can be infected with different organisms. The most common is *Staphylococcus aureus* (42%), followed by *Streptococcus sp.* (25%) and *Staphylococcus epidermidis* (22%). *Pseudomonas*, anaerobic bacteria and *Escherichia. coli* are other less common pathogens.

Risk factors include diabetes, neuropathy, peripheral vascular disease, immunosuppressive therapy, pressure point overload, lifestyle habits (smoking, drug or alcohol abuse), genetic or acquired immunodeficiency, and advanced age.

The Cierny/Mader classification system is used to classify osteomyelitis.

- Type I or intramedullary osteomyelitis refers to a nidus of infection inside the bone. This makes up about 2% of cases.
- Type 2 or superficial osteomyelitis refers to infection in the cortex of the bone that extends through the soft tissue. Most commonly this is seen in a pressure ulcer.
- Type 3 of localized osteomyelitis has a full-thickness cortical sequestrum. Removal of the sequestrum still allows for continuity of the bone.

- Type 4 or diffuse osteomyelitis has the components of types 1, 2, and 3 osteomyelitis but either the bone is unstable or debridement of the infection leads to instability.

The treatment of osteomyelitis is suppressive or curative, and is guided by the host and infection driven factors. The key step is to identify the specific microorganisms, select the appropriate antibiotic and management methods. If the patient is not already on any antibiotics, microbiological cultures of the bone pieces or wound discharge should be obtained prior to administration. Suppressive treatment is considered for the older persons, who are extremely poor in physiological reserves, they may not tolerate surgical drainage.

Curative treatment process;

- Type 1 or medullary osteomyelitis is treated surgically by opening the bone and debriding the nidus of infection. A course of intravenous antibiotics is also given after the debridement.
- Type 2 osteomyelitis is also treated with excision; however, the soft tissue defect must be covered with using a plastic surgical techniques. Surgical treatment may be difficult and may have worse outcomes which requires multistage surgery or even amputation.
- Type 3 osteomyelitis requires excision of the infected bone part but leaves the bone in continuity. Reconstruction must include dead space management and soft tissue reconstruction.
- Type 4 osteomyelitis has a bone that is unstable which must be reconstructed. Options include bone transport or vascularized bone grafts.

After surgical debridement, a prolonged course of intravenous antibiotics is typically recommended for 4 to 6 weeks. There is little literature evidence regarding the exact duration of antibiotic treatment needed.

The treatment of diabetic foot infection is complex and involves a multidisciplinary approach. Glycaemic control is essential to success. Excellent foot care combined with shoes designed to offload pressure

points on the feet can reduce plantar ulcers. In the case of deformity, surgery may be required to reduce the pressure of the ulcer. If ulcers or osteomyelitis develop, surgery may be required. The most typical is amputation. It is important to conduct a vascular study of the leg and foot before the surgery. If the patient cannot tolerate multiple debridement's and subsequent reconstruction, other methods must be considered, such as amputation which is more straight forward and one stage surgery.

A rare complication of chronic osteomyelitis is malignant transformation of the skin edges. More commonly this condition will occur after several years of treatment. Wound edges can transform to squamous cell carcinoma due to the chronicity of wound.

SUBACUTE OSTEOMYELITIS

Subacute osteomyelitis is a distinct form of osteomyelitis. Brodie abscess is one type of subacute osteomyelitis. Subacute osteomyelitis is difficult to diagnose because the characteristic signs and symptoms of the acute form of the disease are absent. It is a low-grade pyogenic abscess of the bone. Subacute osteomyelitis is one of the many clinical presentations of hematogenous osteomyelitis. Causative organisms are similar to acute osteomyelitis but there can be atypical varieties of organisms like mycobacterium species, salmonella and brucella. Subacute osteomyelitis is difficult to diagnose, but once diagnosed, it is having a good prognosis.

TUBERCULOUS OSTEOMYELITIS OF THE SPINE (POTT DISEASE)

Pott disease, also defined as a spondylitis, is a classic manifestation of extrapulmonary tuberculosis (TB). It is associated with high morbidity and may cause severe dysfunction and deformity. Bone and joint involvement are the third most common disease, accounting for 9.8% after lymphatic and pleural diseases in extrapulmonary tuberculosis.

Pott disease usually results from an extraspinal source of infection due to haematogenous dissemination. It manifests as a complex manifestation of osteomyelitis and arthritis that usually involves

multiple vertebrae. Commonly affects the thoracic spine. The anterior part of the vertebral body adjacent to the subchondral plate is usually affected. Tuberculosis may spread from this area to adjacent intervertebral discs.

Progressive bone destruction leads to collapse of the vertebrae and kyphosis. Abscesses, granulation tissue, or direct dural invasion can cause spinal stenosis, which can lead to spinal cord compression and neurological deficits. Compared with the lumbar spine, changes in thoracic spondylosis can easily lead to kyphosis. If the infection spreads to adjacent ligaments and soft tissues, a cold abscess will occur. Abscesses in the lumbar area may descend down the psoas muscle sheath to the femoral triangle and eventually erode the skin.

Diagnosis

Is often delayed due to nonspecific early manifestations and low suspicion. The diagnosis method is very complicated and should be based on the evaluation of chronic and non-relieving back pain (especially the thoracic spine), assessment of other comorbid and epidemiological factors, imaging, assessment of samples for bacteriological, pathological, or molecular confirmation.

Treatment

Treatment requires several months of anti-tuberculosis drug treatment, unless the disease is not accompanied by spinal deformity or compressive neurological symptoms, the drugs must be very effective. When compressive neurological symptoms appear due to abscess formation, surgical intervention should be performed. Spinal braces may help to minimize the pain and kyphosis.

Patients' compliance and resistance to long-term multi-drug therapy are other factors that significantly affect individual outcomes. Paraplegia resulting cord compression caused by active disease usually responds well to chemotherapy. However, due to permanent spinal cord injury, paraplegia may manifest or persist during the healing process. Surgical decompression will help for rapid recovery. Long-term complications need to be monitored, such as disease reactivation, advanced spinal instability and increased kyphosis.

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19. Management of Soft Tissue Injuries

Dr. Chathuranga Ranasinghe

The World Health Organisation (WHO) states that falls are one of the main causes of unintentional injuries that is on the rise among older adult population globally, which significantly impairs their state of health and quality of life. According to the WHO Global Report 2007, approximately 28-35% of people aged more 65 years fall each year leading to 20-30% of mild to severe injuries. Study conducted in the Colombo district in 2011 showed a prevalence of falls of 38%. Major underlying causes for fall-related hospital admissions are hip fracture, traumatic brain injuries and upper limb injuries. Soft tissue injuries are associated with severe cases of fractures/ falls would need hospital admissions while less severe cases are often managed at the outpatient level. The basic knowledge and skills of the clinician managing these soft tissue injuries will be very crucial in the long-term functional ability and quality of life of an older adult. Prompt management of soft tissue related injuries is cost-effective to the patient and to the health care system, avoids unnecessary hospital admissions and prolong hospital stay.

SOFT TISSUE INJURIES

Soft tissue is a term that encompasses all body tissue except the bones. These include skin, muscles, vessels, ligaments, tendons, and nerves. The injuries can range from trivial (e.g. abrasions) to critical (including internal bleeding).

Closed wounds

- An injury where there is no open pathway from the outside to the injured site and can be divided into:
- Contusion: a traumatic injury to the tissues beneath the skin without a break in the skin.
- Ecchymosis: discoloration under the skin caused due to leakage of blood out into the surrounding soft tissues.

- Oedema: swelling as a result of inflammation or abnormal fluid under the skin.
- Strain: stretching or tearing of a muscle or tendon resulting from overstretching or overexertion e.g. quadriceps muscle tear.
- Sprain: a joint injury involving damage to supporting ligaments and partial or temporary dislocation of bone ends, partial tearing or stretching of supporting ligaments e.g. ankle sprain.

Open wounds

These occur when the skin is interrupted or broken, exposing the tissues underneath. The following are included: abrasions, lacerations, incisions, punctures, avulsions and amputations. Depending on the severity these conditions the surgical management is required. The discussion is beyond the scope of this review.

PRINCIPLES OF SOFT TISSUE INJURY MANAGEMENT

The Acute stage - PRICE

The acronym PRICE (protection, rest, ice, compression and elevation) has been central to acute soft tissue injury management for many years. Over the time, acronyms guiding the management have evolved from ICE to RICE and then to PRICE.

Case example

Mr. X, a 68-year-old male presented to the general practice with an acute right lateral ankle sprain after a roll over injury of the foot. He had type 2 diabetes mellites controlled with oral hypoglycaemics. No significant other medical or surgical conditions were noted. On examination pain, oedema, a localised tenderness at the right lateral malleoli near the anterior talofibular ligament (ATFL) insertion was noted without ecchymosis. The ankle had loss of movement mainly due to pain. Clinical diagnosis was made as ATFL strain (Grade 1-2).

According to the traditional PRICE regime, the initial management (in the first 48-72 hrs.) consists of the following

Protecting the ankle from further injury by gently supporting the site without initial weight baring

Rest to the joint so that affected muscle, tendon and ligaments are in relaxed position (use of crutches is walking is desired), reassurance and pain control with medication (mostly paracetamol or non-steroidal anti-inflammatory drugs/NSAIDs)

Ice (Cryotherapy)

Compression with supportive strapping or bandaging (Check circulation, motor, and sensation before and after)

Elevation of the injured lower limb (Elevation of the right limb higher than the heart to promote interstitial fluid flow out of tissues).

If a severe injury to the ligament (completely torn ligament) or fracture is suspected, the ankle will need immobilisation and splinting. Furthermore, surgical intervention and follow up is required.

Cryotherapy

Cryotherapy is one of the most important components of the PRICE regime which uses an external cold source (ice bags with crushed ice, cold gel packs, ice massage, cold water submersion, gaseous cryotherapy, and continuous-flow cryotherapy devices) to reduce the tissue temperature. Most often the practiced protocol in the acute stage, includes application of cryotherapy for 10-20 minutes to/around the injured site every 6-8 hrs (depending on the severity) for the first 48-72 hrs after the injury. Current evidence does not favour one method of cryotherapy application, protocol, or both. The decrease in tissue temperature results in decreased tissue oedema and microvascular permeability, reduced delivery of inflammatory mediators, reduced blood flow via vasoconstriction, overall net decrease in tissue metabolic demand. It has shown to increase the threshold of painful stimuli with a significant benefit for pain control. Cryotherapy can result in complications like nerve palsies, mostly involving more superficial nerves such as the peroneal nerve. Care must be taken to provide sufficient insulation between the skin and the cryotherapy source, especially in patients with minimal subcutaneous fat. Nerve injuries can range from brief paraesthesia to complete axonotmesis. Frostbite has

also been a concern but, rarely reported in the literature as a complication of treatment.

Effect of PRICE on the inflammatory process

The PRICE protocol has been widely followed despite a paucity of high-quality, empirical evidence to support its various components or as a collective treatment package. Even though ice/cryotherapy is the cornerstone of the PRICE regime, there is no high-quality evidence of the efficacy on tissue healing process except for pain control. It is argued that ice could potentially disrupt inflammation, angiogenesis and revascularisation, delay neutrophil and macrophage infiltration as well as increase immature myofibers leading to impaired tissue repair and redundant collagen synthesis. But cold-induced analgesia, the assurance and support provided by compression and elevation are enough to retain ICE within the management. Even though not included in the acronym, the widespread use of NSAIDs observed in clinical practice is been challenged, particularly in the management of ligament and muscle injuries. It is argued since various phases of inflammation help repair damaged soft tissues; inhibiting inflammation using medications may negatively affect long-term tissue healing, especially when higher dosages are used. Additionally, this can be of caution to an older patient due to the side effects of NSAIDs. However, it is still premature to propose that NSAIDs are not useful to the physician in managing soft tissue injuries. Both dose and duration minimisation should be prioritised and combined with simple principles of PRICE, which allows NSAID-sparing.

The subacute stage - POLICE and optimal loading

Acute management should always follow a subacute and long-term rehabilitation stages for optimal functionality. Even though aggressive ambulation or exercise are to be avoided just after trauma, complete rest should be of limited duration (a 'relative rest'). Longer periods of unloading can produce adverse changes to tissue biomechanics and morphology whereas progressive mechanical loading is more likely to

restore the strength and morphological characteristics of collagenous tissue.

For Mr X, supervised functional rehabilitation which involves ankle muscle strengthening and early weight-bearing usually with a crutch support will be effective and is superior to unloaded total immobilisation.

POLICE, a new acronym, represents ***P****rotection,* ***O****ptimal* ***L****oading,* ***I****ce* ***C****ompression and* ***E****levation*. This has been proposed to aid clinicians to ponder on strategies for safe and effective mechanical loading or "Optimal Loading" during acute soft tissue injury management. Optimal loading replaces rest with a balanced and incremental rehabilitation programme with early activity encouraging early recovery. The difficult clinical challenge in an elderly patient is finding the balance between loading and unloading during tissue healing. If tissues are stressed too aggressively after injury, the mechanical insult may cause re-bleeding or further damage. A loading strategy should reflect the unique mechanical stresses placed upon the injured tissue during functional activities, which varies across tissue type and anatomical region.

Patient and family education on, benefits of an 'active approach' to recovery is of extreme importance. Excessive use of passive modalities (such as electrotherapy, manual therapy or acupuncture etc.), overtreatment and therapy-dependent behaviour can be counterproductive on recovery in the long term. For Mr. X the healing time and recovery would be much longer than a person of a younger age and can be further complicated with his glycaemic status. Clinicians should strongly advocate for setting realistic expectations with patients about recovery times instead of chasing the 'magic cure' approach.

Subsequent chronic stage - treat with LOVE

In 2020, Dubois et al. proposed the acronym LOVE (Load, Optimism, Vascularisation and Exercise) highlighting the value of long term rehabilitation in soft tissue injury management, which is usually missed in practice. While maintaining optimal loading to promote tissue repair,

supporting optimistic patient expectations are associated with better prognosis as patient can have anxiety, depression and fear which can become barriers to recovery. During the chronic stage there is tendency for the patient to detach from the normal pre-injury lifestyle and become more physically inactive even with rehabilitation. While the acute stage is resolving, pain-free symptom allowed aerobic/cardiovascular exercises should be started to boost motivation, regain confidence and increase blood flow (vascularisation) to the injured structures. There is a strong level of evidence supporting the gradual introduction of a holistic general exercise program during the recovery of the injury.

Prescribing specific muscle strength training to the ankle and balance training will improve Mr Xs' functionality and reduce possible recurrent ankle sprain in the future. In addition, a general exercise program will help to restore endurance, mobility, strength, proprioception and early regain active life which will be a beneficial lifestyle modification for the control of his glycaemic status. Pain should be avoided and used as a guide for exercise progressions.

No HARM at the acute stage

While PRICE focuses on minimising bleeding and swelling, **HARM** (**H**eat, **A**lcohol, **R**unning/exercise, and **M**assage) encompasses the factors to avoid during the acute stage which will increase circulation to the damaged area and aggravate the injury. Application of heat with hot towels/packs is a common traditional household remedy in Sri Lanka which should be avoided in the acute stage. Consumption of alcohol should be avoided which can dilate blood vessels, mask pain and the severity of the injury which can increase the risk for re-injury. Running/exercise is the opposite of 'relative rest'. Massage of the injured areas can damage underlying injured structures which will also stimulate the flow of blood to the area.

CONCLUSION

Soft tissue injuries, if poorly managed, will result in poor long-term functionality and quality of life. Managing soft-tissue injuries is more than short-term damage control. Similar to other injuries, clinicians should aim for favourable long-term outcomes and treat the person with the injury rather than the injury of the person. PRICE regimen has been widely and successfully used in acute soft tissue management for a long time even with paucity of high-quality research evidence. It has been further improved with acronym POLICE, which emphasises 'relative rest' and 'optimal loading' of the injured soft tissue, to promote healing which needs to be introduced at the subacute stage. The acronym LOVE, highlights the long term more holistic care in recovery from injury, improving general health and fitness. The clinician can customize these concepts to own context and use them according to the type, site and severity of the injury. The longer duration need for recovery from injury, comorbidities and social dependency are important additional considerations to an older adult. Prevention of these injuries can be achieved by providing an environment in which the elderly citizens has a safe surrounding, improved awareness (patient and family) about what possible danger injuries might occur, a clear path to elevation of such danger in the time of need.

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20. Nursing care in Musculoskeletal Disorders

Dr. Nirmala Rathnayake

Musculoskeletal disorders are typically associated with pain (either intermittent or persistent) and limitations in mobility, dexterity and functional ability, reducing older people's ability to work and participate in social activities. Integrated and coordinated multidisciplinary and multi-professional services are needed for the successful management of musculoskeletal problems in older people. In such multidisciplinary care teams, the nurse has a definite and specific role.

GENERAL NURSING CARE FOR OLDER PATIENTS WITH MUSCULOSKELETAL DISORDERS

The role of the nurse in caring musculoskeletal disorders is multi-dimensional and it varies from caregiving to educating the patient and care givers. This spans across physical, psychological, environmental and spiritual care.

Older adults with musculoskeletal disorders have age specific, complex and multifaceted needs. They are affected in unique ways by the combined effects of the ageing, disease process and the environment which challenge their sense of self and influence their perception of quality of life (QOL). The nurse should be knowledgeable, skilled, vigilant, sensitive, proactive, respectful and positively motivated about caring to provide nursing care to older adults. The main objectives of providing nursing care to older adults with musculoskeletal disorders are to

a) promote and maintain optimal level of health and function with the existing musculoskeletal disorders
b) prevent deterioration of the existing musculoskeletal disorders
c) prevent complications of the musculoskeletal disorder.

The organized framework through which nurses delivers care involves assessing, identification of needs (nursing diagnoses), planning, implementing and evaluating nursing care according to the nursing process. They need to assess the patients' physical, mental and

cognitive skills, understand their acute and chronic health issues, and the specific concerns connected to the musculoskeletal disorder and common health issues of older age (i.e. falls, incontinence, changing sleep patterns etc.)

Different functions of nurses that can contribute to the optimum health and overall wellbeing of the older people with musculoskeletal disorders include supportive, restorative, educative, life-enhancing and managerial care. The supportive role includes psychosocial and emotional support, enhancing lifestyles and relationships, facilitating self-expression and ensuring cultural sensitivity. The restorative functions include maximizing the independence and functional ability, preventing further deterioration and/or disability, and enhancing the QOL. This is undertaken with a focus on rehabilitation that maximizes the older person's potential for independence, including assessment skills and undertaking essential care elements such as washing, dressing etc. On the educative role, nurses should teach self-care activities, such as self-medication for pain management, continence promotion and health screening. Nurses can teach older persons about the importance of weight management, engaging in physical activities to reduce the impact of musculoskeletal disorders on bodily functions, and stress management. In learning to manage their own health, older adults can retain more independence and possibly lessen the need for medical treatment. They can also assist family members, friends, and others to enhance knowledge and skills regarding the management of specific musculoskeletal disorders including safety, disease prevention, and adherence to medication etc. The life enhancing activities of a nurse include relieving pain and ensuring adequate nutrition for palliative management and rehabilitation. Furthermore, the managerial role of the nurses includes the supervision of care delivered by other staff and the overall management of the home environment.

Nurse must follow the following general guidelines when caring for older patients with musculoskeletal disorders since older people are extremely sensitive, emotionally unstable compared to young adults.

a) Consider individuality of the older patients. Do not attempt to alter the lifelong character and behaviour.
b) Be patient, kind, sympathetic while providing care.
c) Handle them gently and maintain privacy while providing care.
d) Communicate effectively. Make sure they can hear properly.
e) Encourage independence as far as possible.
f) Assist to achieve emotional stability. Support them during their periods of anxiety. Give them time to express their feelings. Praise even minimal achievements. Encourage contact with others.
g) Protect them from injuries, falls, and accidents with proper instructions / arrangements.
h) The older patients with musculoskeletal disorders are highly prone to develop bedsores. Therefore, provide comfortable bed, and smooth and wrinkle free bed linen.
i) Ensure adequate hydration and nutrition.
j) Encourage to do active range of motion exercises.
k) Maintain body alignment, posture, and mobility.
l) Help them to establish good sleeping pattern. Try to engage them in certain activities during day time, so that they can sleep well during night time.

The specific nursing care described for different musculoskeletal disorders are structured according to the nursing process, which include, nursing assessment, nursing diagnosis, planning and implementation and evaluating nursing care.

NURSING CARE FOR OLDER PATIENTS WITH LOWER BACK PAIN

Lower back pain is characterized by an uncomfortable or acute pain in the lumbosacral area associated with spasm of the para-spinal muscles, usually with pain radiating to the lower extremities.

Nursing assessment

a) Obtain history to determine when, where, and how the pain occurred, aggravating and relieving factors, relationship of pain to specific activities, presence of numbness or paraesthesia.

b) Perform a physical examination of neurologic system, localized weakness of extremities and reflex and sensory loss.
c) Perform musculoskeletal examination for changes in strength, tone, and range of motion exercises (ROM).
d) Assess coping ability of patient and partner, family for close relatives.
e) Assess effect of illness on daily living work.

Nursing diagnoses

a) Acute or chronic pain related to injury.
b) Impaired physical mobility related to pain.
c) Psychological disturbance related to pain.
d) Sleep pattern disturbances related to pain.
e) Risk for impaired skin integrity (bed sores) related impaired mobility.
f) Risk for addiction and medication errors due to self-administration of medications.

Planning and implementation

Relieving pain

a) Advise older patient to stay active as far as possible and avoid bed rest.
b) Keep a pillow between flexed knees while in side-lying position that minimizes strain on back muscles.
c) Provide lumbosacral support to provide abdominal compression and decreases load on lumbar intervertebral disks.
d) Apply heat (moist towels/packs) or ice, to relax muscle spasm and relieve discomfort.
e) Administer or teach self-administration of pain medications and muscle relaxants, as prescribed.
f) Administer non-steroidal anti-inflammatory drugs (NSAIDs) with meals to prevent gastrointestinal (GI) upset and bleeding, since muscle relaxants and opioids may cause drowsiness.

Promoting mobility

a) Encourage ROM or perform passive exercise (if unable to perform ROM) of all uninvolved muscle groups.

b) Suggest gradual increase of activities and alternating activities with rest in semi-fowler's position.
c) Avoid keeping the patient in prolonged periods of sitting, standing, or lying down.
d) Encourage older patient to discuss problems that may be contributing to backache.
e) Encourage patient to do prescribed back exercises since exercise keeps postural muscles strong, helps recondition the back and abdominal musculature, and serves as an outlet for emotional tension.
f) Avoid activities that may strain the back until healed, but bed rest is to be avoided as well, because it may significantly decrease the rate of recovery, increase pain and disability, and lengthen time spent absent from work.
g) When the patient is in bed, sleep in a supine to semi-fowler's position with hips and knees flexed to relieve painful muscle and ligament sprain, heal soft-tissue injury, remove stress from lumbar sacral area, relieve tension on sciatic nerves, and open the posterior part of the intervertebral spaces.
h) Isometric exercises should be done hourly while in bed, if possible.

Enhance psychological status

a) Provide adequate pain management.
b) Administer psychotropic medications as prescribed. They may be used for treatment of depression and anxiety, which potentiate pain.
c) Provide distraction to relieve the pain and cope with the functional disability (music, hymns, pirith chanting. etc.).
d) Teach relaxation methods and engage in relaxation methods (such as prayers, meditation. etc.) with them.
e) Assist them adequately for their day to day activities and basic needs.

Prevent the impairment of skin integrity (bed sores)

a) Provide a comfortable bed, and smooth and wrinkle free bed linen.
b) Ensure adequate hydration and nutrition.
c) Encourage to do active ROM exercises.
d) Maintain body alignment, posture, and mobility.

e) Avoid prolong bed bounding due to the pain

Promote sleeping

a) Help them to establish good sleeping pattern by introducing sleeping rituals.
b) Try to engage them in certain activities during day time, so that they can sleep well during night time.

Avoid medication related issues

They are prone to use self-medication to resolve the issues of pain and sleep.

a) Caution them about self-use of drugs especially analgesics and narcotics.
b) Reinforce verbal instructions with written instructions about their drugs, because of the poor eyesight and forgetfulness they may not be able to understand the instructions or the importance of drug treatment.
c) Explain side effects of drugs and watch for them.
d) Arranging drug schedule coinciding with the regular activity helps patients to remember taking drugs (reminding and keep alarms etc).
e) Monitor the drug dosage strength. It may be 30-50% less than the younger persons.

Provide patient and family education for health maintenance

a) Instruct family members or patient caregiver to avoid recurrences by following methods.
    1. Standing, sitting, lying, and lifting properly are necessary for a healthy back.
    2. Suggest alternate periods of activity with periods of rest.
    3. Ask them to avoid prolonged sitting (intra-discal pressure in lumbar spine is higher during sitting), standing, and driving.
    4. Change positions and rest at frequent intervals.
    5. Emphasize to avoid assuming tense, cramped positions.
    6. Sit in a straight-back chair with the knees slightly higher than the hips. Use a footstool if necessary.

7. Avoid knee and hip extension. Place a cushion in the small of the back for support.
8. Avoid fatigue, which contributes to spasm of back muscles.
9. Use good body mechanics when lifting or moving about.

b) Encourage daily exercise which is important in the prevention of back problems
   1. Do prescribed back exercises twice daily strengthens back, leg, and abdominal muscles.
   2. Walking outdoors (progressively increasing distance and pace) is recommended.
   3. Reduce weight if necessary, decreases strain on back muscles.

Evaluation: expected outcomes

a) Verbalizes relief of pain with rest and medication.
b) Performs back exercises correctly.
c) Physical mobility is optimized.
d) Psychological status is calm and balanced.

NURSING CARE FOR OLDER PATIENTS WITH ARTHRITIS

Nursing assessment

a) Obtain history of pain and its characteristics, including specific joints involved.
b) Take a history on past injuries to the joints, as this is a risk factor for osteoarthritis.
c) Assess the stiffness of joints, which is mostly experienced in the morning or upon awakening, usually lasts less than 30 minutes and decreases with movement.
d) Review functional impairment results from pain on movement and limited motion caused by structural changes in the joints.
e) Evaluate ROM exercises and strength.
f) Assess effect on ADLs and emotional status.
g) Assess the area over the affected joint may reveal tender and enlarged joints.

Nursing diagnoses

a) Acute or chronic pain related to joint degeneration and muscle spasm
b) Impaired physical mobility related to pain and limited joint motion/stiffness
c) Bathing, Hygiene, Feeding, and Toileting self-care deficits related to pain and limited joint movement
d) Knowledge deficit related to the lack of exposure of sources of knowledge

Planning and implementation

Relieving pain

a) Advice patient to take prescribed NSAIDs or OTC analgesics as directed to relieve inflammation and pain. May alternate with opioid analgesic, if prescribed.
b) Provide rest for involved joints; excessive use aggravates the symptoms and accelerates degeneration. Use splints, braces, traction, lumbosacral corsets as necessary and have prescribed rest periods in recumbent position.
c) Advise patients to avoid activities that precipitate pain.
d) Apply heat as prescribed to relieve muscle spasm and stiffness; avoid prolonged application of heat may cause increased swelling and flare symptoms.
e) Teach correct posture and body mechanics postural alterations lead to chronic muscle tension and pain.
f) Advise sleeping with a rolled terry cloth towel under the neck for relief of cervical osteoarthritis
g) Provide crutches, braces, or cane when indicated to reduce weight-bearing stress on hips and knees.
h) Teach use of cane in hand on the side opposite to the involved hip/knee.
i) Advice wearing corrective shoes and metatarsal supports for foot disorders also helps in the treatment of arthritis of the knee.
j) Encourage weight loss to decrease stress on weight-bearing joints if the older person is obese.

k) Support patient undergoing orthopaedic surgery for unremitting pain and disabling arthritis of joints.
l) Older patients are at greater risk for GI bleeding and renal failure associated with NSAID use. Encourage administration with meals, and monitor stool for occult blood.

Increasing physical mobility

a) Encourage activity as much as possible without causing pain.
b) Teach ROM exercises to maintain joint mobility and muscle tone for joint support, to prevent capsular and tendon tightening, and to prevent deformities. Avoid flexion and adduction deformities.
c) Teach isometric exercises to improve muscle strength around the involved joint.
d) Advise putting joints through ROM after periods of inactivity.

Promoting self-care

a) Suggest performing important activities in morning, after stiffness has been abated and before fatigue and pain become a problem.
b) Advise on modifications, such as wearing looser clothing without buttons, placing bench in tub or shower for bathing, sitting at table or counter in kitchen to prepare meals.
c) Help with obtaining assistive devices, such as padded handles for utensils and grooming aids, to promote independence.

Provide patient and family education for health maintenance

a) Encourage following
    1. Weight loss - Weight loss is an important approach to pain and disability improvement.
    2. Assistive devices - Canes and other ambulatory devices are very helpful for ambulation.
    3. Exercise - Exercises such as walking should begin moderately and increase gradually. Suggest exercises as a form of non-stressful exercise to preserve mobility.
    4. Analgesic - Adequate pain management is essential to the success of an exercise program.
    5. Physical therapy - A referral for physical therapy for people with similar problems can be very helpful.

6. Adequate diet and sleep to enhance general health.

b) Make aware of the use of complementary or alternative therapies such as herbal and dietary supplements, relaxation exercises, meditation etc.

c) Advise patient and family about the prevention of injuries. As one of the risk factors for osteoarthritis is previous joint damage, it is best to avoid injuries that might befall the weight-bearing joints.

Evaluation: expected outcomes

a) Report reduction in pain while ambulatory

b) Performs ROM exercises

c) Dressing, self-bathing and grooming with assistive devices

1. Identify negative factors affecting activity intolerance and eliminate or reduce their effects when possible.
2. Use techniques to enhance activity intolerance.
3. Report measurable increase in activity intolerance.
4. Report pain is relieved or controlled.
5. Follow prescribed pharmacologic regimen.
6. Participate in ADLs and desired activities.

NURSING CARE FOR OLDER PATIENTS WITH OSTEOPOROSIS, FRAGILITY FRACTURES AND ASSOCIATED RISKS

Nursing Assessment

a) Obtain a health history - The health history includes questions concerning the occurrence of osteopaenia and osteoporosis and focuses on family history, previous fractures, dietary consumption of calcium, exercise patterns, onset of menopause, and the use of corticosteroids as well as alcohol, caffeine and smoking.

b) Assess symptoms - Any symptoms the patient is experiencing, such as back pain, constipation, or altered body image need to be are explored.

c) Perform physical examination - Physical exam may disclose a fracture, kyphosis of the thoracic spine, or shortened stature.

Nursing diagnosis

a) Acute pain related to fracture and muscle spasm.

b) Risk for impaired bowel habit (constipation) related to immobility or development of ileus.
c) Risk for injury (additional fractures related to osteoporosis) related to weakened bone density
d) Psychological disturbance related to disease process and poor endurance.
e) Deficient knowledge about the osteoporotic process and treatment regimen.

Planning and implementation

Relieving pain

a) Advise the patient to rest in bed in a supine or side-lying position several times a day.
b) Provide mattresses that should be firm and non-sagging.
c) Encourage knee flexion that increase comfort.
d) Provide intermittent local heat and back rubs that promote muscle relaxation.
e) Encourage good posture and teach body mechanics.

Improving bowel movement

a) Encourage consumption of high fibre diet with adequate hydration and assist appropriately.
b) Increase the quantity of fruits, fruit juice, and vegetables.
c) Reduce the content of processed foods and refined sugars.
d) Avoid junk foods that are dense in fat and low in fibre.
e) Include foods with active cultures of Lactobacillus acidophilus (e.g. yoghurt).
f) Drink water throughout the day.
g) Advice the use of prescribed stool softeners to assist, prevent or minimize constipation.
h) Allow adequate toilet times.
i) Encourage patients own constipation relieving methods (i.e. home remedies).
j) Mobilize the patient while taking appropriate precautionary measures to prevent falling

Preventing injury

a) Encourage walking and activities with precautions.
b) Enhance good body mechanics.
c) Keep good posture and daily weight-bearing activity outdoors to enhance production of vitamin

Enhance psychological status

a) Provide adequate pain management
b) Psychotropic medication may be used for treatment of depression and anxiety, which potentiate pain. Therefore, administer psychotropic medications as prescribed.
c) Provide distraction to relive the pain and cope with the functional disability (music, hymns pirith chanting. etc).
d) Teach relaxation methods and engage in relaxation methods (such as prayers, meditation. etc.) with them.
e) Assist them adequately for their day to day activities and basic needs.

Provide patient and family education for health maintenance

a) Encourage following,
    1. Diet - Identify calcium and vitamin D rich foods and discuss on calcium supplementation.
    2. Exercise - Engage in weight-bearing exercise daily.
    3. Lifestyle - Modify lifestyle choices: avoid smoking, alcohol, caffeine, and carbonated beverages.
    4. Posture - Demonstrate good body mechanics.
    5. Early detection - Participate in screening for osteoporosis such as DXA scanning or bone marker assessment.
b) Educate family for outdoor safety
    1. Wear low-heeled shoes/slippers.
    2. Use handrails when going up and down.
    3. Look carefully at floor surfaces in public buildings (polished, tiled floors, wet surfaces, carpets etc).
    4. Good illumination, mobilise out of home during the day time.
    5. Use assistive devices such as walkers or cane.
    6. Wear hip protective devices if available.
c) Emphasize for domestic arrangements for the prevention of falls.

1. Place items more often used by the older person within easy reach.
2. Use assistive devices to avoid strain and injury.
3. Remove all loose wires, cords on floors.
4. Keep the floor free of clutter.
5. Keep the older person in a room where the floor is non-slippery.
6. Keep furniture in its usual place. Do not alter the places.
7. Use safety place to bath the older patient, if a tiled bathroom is used, bathing should be assisted by another person.
8. Avoid water spills at home or kitchen; clean them as early as possible.
9. Keep the patient in downstairs.

Evaluation – expected outcomes

a) Achieve pain relief.
b) Demonstrate normal bowel elimination.
c) Experience no new fractures.
d) Acquire knowledge about osteoporosis and the treatment regimen.

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21. Physiotherapy in musculoskeletal disorders

Mr. Iranga Aluthge

Musculoskeletal disorders of the older adults involve inactivity due to pain with several associated problems such as loss of mobility, restriction in joint motion, altered balance and gait. Restricted activities due to persistent pain results in grave problems in the community such as possibility of social isolation and sometimes psychological problems such as depression. Painful and weaker body elements will decondition due to disuse. Physiological changes that occur with ageing in the human body will result in reduced ability to bear pain (pain intolerance). Therefore, physical modalities and exercises should be the core of any approach that manage pain in geriatric community. Physical relief of chronic, sub-acute or acute musculoskeletal pains can be done by exercises that will target the source of pain.

Physiotherapy and physiotherapists assist patients to understanding the transformation between 'hurt' and 'harm'. Exercises or given targets may cause discomfort or hurt. However, it does not imply that damage or harm is being caused the system. Physiotherapists have numerous interventions and modalities to assist the elderly community for musculoskeletal disorders to relieve pain by improving the functional mobility, balance, strength, endurance and independence. Evidence from the previous researches demonstrate the positive impact of their effectiveness in treating chronic pain in elderly population.

Disability is generally regarded due to a pathological process, or injury, and not prima facie 'old age'. The effects of biological ageing reduce the efficiency of the body systems. However, throughout life, optimum function is maintained in each individual by continuing to use these systems to their maximum capacity. Physiotherapists have a key role in enabling older people to use a number of body systems to fully to enhance mobility and independence. When neither improvement nor even maintenance of functional mobility is a reasonable goal, physiotherapists can contribute to assisting older people to remain

comfortable and pain-free. Prevention of development of problems in later life through health promotion is also another principle

TREATMENT MODALITIES

Superficial heating is commonly used for pain relief, reduce muscle spasms and accelerate circulation locally by vasodilation. It also permits relaxation and prepares muscles and joints for the exercises. Heat also acts as the counter-irritant as it changes the sensory input to the skin and block the pain perception as well as the tissue response to it. Superficial heat penetrates 1-2 cm deep and decreases the sensitivity of pain trigger points. Heat reaches the skin and superficial subcutaneous structures, and improves the mobility and the elasticity. Infra-red radiation lamps, hydrocollator packs, paraffin wax baths, heating hydrotherapy, short-wave diathermy and ultra sound therapy are common example for the superficial heating modalities. Further electrical stimulation, soft tissue mobilization, joint mobilization and exercise therapy may have an additional benefit for relieving musculoskeletal pains.

The indication of superficial heat therapy is to minimized the generalized pain and tenderness associated with bursitis, tendonitis, trigger points, muscle spasm, myofascial pains, fibromyalgia, several types of arthritis, back pains and cervical pain. Response is well-known but sometimes this may be a temporary effect that is not lasting many days.

Infra-red radiation generating lamps emit wavelength of 760nm-3000nm infra-red A and B bands. Various lamps are used for therapy (e.g. non-luminous generators and the luminous generators.)

Hydrocollator packs or the hot packs are filled with hydrophilic silicate dioxide sand which when kept in hot water temperature between 70^0-80^0C can absorb many times its' weight in water, producing a gel like substance, that can be conformed to the body part needed. Six to eight cotton towel layers must be applied between the skin and the pack to prevent burns. Wet heat penetrates deeper than dry heat. The application time is 15 -20 with the temperature persisting throughout.

Several studies have shown that pain decreases after a longer period of time following wearing of heat wraps rather than brief heating through hot packs.

Heating pads are often used by the older adults to relieve the pain in joints and muscles. It heats up to 52^0C. However, temperature can fluctuate at a wider range. Extreme caution should be used when recommending this as patient may fall asleep with heating pads on their skin and resulting in partial thickness burns.

Paraffin wax baths are ideal for the distal extremities. These are used as a combination of wax and oil maintained at temperature 45^0-54^0C. The affected body part is dipped in to the melted paraffin waxes many times and wrapped in a plastic bag. Then they are wrapped with towels for 20 minutes until the heat is retained. Wax can be painted if the region is practically difficult to be dipped. Tissue temperature is raised with the oil providing a lubricant effect to the skin.

Hydrotherapy or the whirlpool treatment are the one of oldest method for managing pain and musculoskeletal dysfunctions. The sizes and the shapes of the tanks vary to accommodate specific body parts. Most of the whirlpool tanks have a turbine engine for water agitation. The direction and the intensity of the agitation can be adjusted to assist or resist exercises. The duration of the session is about 20 minutes in water temperatures 36.5^0-40.5^0C. Cleaning of the tanks for individual patients is important to prevent contamination and infection.

Aquatherapy in a pool is recommended for those with pain and joint stiffness. These include muscle weakness, osteoarthritis and rheumatoid arthritis, obesity, neurological disorders, orthopaedic conditions, joint replacements, polio, ankylosing spondylitis, osteoporosis. Aquatherapy promotes relaxation, increase circulation, restore mobility, strengthen muscles, improve balance, increase proprioceptive inputs and increase metabolic activity. The main benefit of aqua therapy is to perform exercises with decreased weight-bearing stress on joints. Elderly individuals with cardiovascular diseases best tolerate water temperature below 38^0C.

In ***Short wave diathermy (SWD)*** treatment the affected body part is placed inside an electromagnetic field created by high frequency alternating currents. There are two modes: continuous or pulsed. For medical purposes 27.12 MHz frequency and 11 meters' wavelength is used. There is resistance of the current with generation of heat. Heat penetration of the SWD is more than that of the infra-red radiation machine it will be of 1 cm depth. There are many contraindications and precautions of this modality which will limit usage of SWD in current days.

Cryotherapy or cold therapy is used to decrease pain, oedema, inflammation, and muscle spasms. There is local or general body cooling for therapeutic purposes by transfer of energy away from the tissues. Cooling can be achieved in several ways, such as, evaporating liquids, blowing cold air over the skin, with crushed ice. Heat transferred by conduction from the skin and energy is used in changing solid ice to water. Cryotherapy temporarily decrease spasticity, increase response to the passive stretch, increase deep tendon reflexes and clonus before commencing the exercises. This include cold packs, ice massage, cold baths, whirlpools, vapocoolant sprays and cold compression units.

Cold packs are silica-gel filled packs stored at -5^0C and applied to the affected area for 10-15 minutes. A wet cloth layer is placed between the cold pack and the skin to conduct cold better and to promote the personal hygiene

Ice massage is done by the formed ice and constant motion over the smaller to mid-sized affected areas for 5-10 minutes or until the patient goes through the four sensory stages of intense cold, burning, aching and analgesia. In the absence of other impairments, active range of motion exercises can be combined with ice massage to improve the earliest function of the affected body part.

Cold baths or whirlpools are used in applying ice for oedematous extremities and temperature should be maintained at $13\text{-}18^0$C. Treatment lasts for 10-15 minutes and patients are advised to move the involved extremity throughout the session to promote the pumping of excessive fluid from the affected area. Older adults need to be closely

supervised during the session due to preexisting decreased peripheral circulation.

Vapocoolent sprays of fluoromethane are used in treating trigger points or restricted muscles which are held in a passive stretch. When the spray is applied from the proximal end to the distal end along with the length of the muscle it is easy to stretch the muscle and the technique is called "spray and stretch".

Cold compression units are often used postoperatively. The mechanism is to use the gravity to allow iced water to flow form a cooler in to a sleeve that fits snugly over the affected area. Warm water can be drained from the sleeve back to the cooler. Some units have the pumps that keep the cool water circulating constantly.

Ultrasound therapy applies thermal as well as non-thermal effects to achieve pain relief, muscle relaxation, oedema reduction, increase collagen tissue extensibility, improving circulation, and promote healing. Ultrasound frequencies used in physiotherapy are in the range 0.5 MHz – 5MHz and pulsed and continuous modes are used. Ultrasound is used to treat soft tissues disorders such as tendonitis, bursitis, trigger points, muscle spasms, and evidences are there about the good results in treating lateral epicondylitis, calcified tendonitis, adhesive capsulitis and rotator-cuff tendonitis.

Electrical stimulation is an extremely versatile modality and by changing the wave form that can be modified to different types of clinical electrical stimulation. Frequency, wave form, and intensity are the different parameters that can be changed and modified according to the treatment goals. Transcutaneous electrical nerve stimulation (TENS) and interferential therapy are the commonly used forms of electrical stimulations for controlling the chronic and acute pains and muscle spasms. Interferential therapy currents penetrate deeper than the TENS while operating at higher frequencies (4000Hz-8000Hz) achieving more symptom control.

These types of electrical stimulations can be used to treat acute pains, chronic pains, phantom limb pains, neurological pains, osteoarthritis, fibromyalgia, tendonitis and bursitis. Nausea control in patients on

chemotherapy, regaining motor functions following a stroke, decreasing angina pectoris, decreasing urge incontinence, decreasing joint contractions, and decreasing spasticity are less common but also having favorable responses.

Soft tissue mobilisation techniques will also be helpful in reducing the pain, decreasing muscle spasms, improving bold circulation, improving lymphatic drainage, decreasing trigger points, and decreasing blood pressure. Soft tissue mobilisation techniques are often demonstrated in individuals with chronic pains, acute pains, oedema, fibromyalgia, myofascial pains, muscle spasms, trigger point bands, arthritis, reduced range of motions, headaches and pain related to cancers. These will promote the muscle relaxation and flexibility and loosening of scar and connective tissues by stimulating the muscles and fascial tissues.

Joint mobilisations are generally used to decrease the pain and to improve the joint range of motion directly. Also, this will break the scar tissue adhesions and promote the joint nutrition. Indications for the joint mobilisations are chronic pain, painful joints, post-surgical scaring, loss of range of motion, and functional limitations. Indirect effects involve with the sympathetic nervous system (SNS) and SNS lies along the spine at the level of T1 through L2. The ganglia containing the cell bodies, lie very close to the spinous

Joint mobilisations can be applied to any joint from larger hip joint to the smaller facet joints of the spine if required. Mechanism of the mobilisation is to apply gentle or aggressive force to stretch the capsule by distracting the joints, pulling the articulating surfaces apart and gain the range of motion. Also, extreme care should be observed when mobilizing joints of the elderly, those who are having osteoporosis, severe osteoarthritis, rheumatoid arthritis, chronic pains, joint replacements, post-surgical sites and elders with chronic steroid use. Mobilisation of joints are contraindicated in unstable joints, infected joints and near a healing fracture.

Exercises are also very important in managing the musculoskeletal disorders and as the requirement type of exercise may vary. All the types of exercises are promoting the circulation. Mobility and stretching

exercises are further reducing the muscle tensions as well. The overall increase in connective tissue with gradual decrease in tissue elasticity will leads to decrease in range of motion. It is very important to consult a physiotherapist before the exercises, because it can aggravate the current condition, if not properly assess and designed the exercises programme. Exercises are the obvious solution for the chronic pains in elderly, stiff muscles, decreasing the muscle mass, strength, power, endurance and range of motion. Exercises can be categorized in to several types such as mobilisation exercises, strengthening exercises, coordination exercises and stretching exercises.

Protective and supportive devices also assist in decreasing pain and increasing maximum functions for elderly persons with joint instability or malalignment. Kinesio taping for patellar realignment is much effective in decreasing pain and optimizing functions in elderly persons with osteoarthritis of knee joint and it will improve the blood circulation and relax the soft tissue structures underneath.

Although there are several interventions for treating musculoskeletal disorders in older adults, number of challenges also identified. They are the inappropriate pain management approaches, economic barriers, social barriers, regulatory or policy barriers and ethical barriers.

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22. Occupational Therapy for Musculoskeletal Disorders

Mr. Nandana Welage

Disorders of the musculoskeletal system are common and can have a great impact on the quality of life of the elderly, such as mobilisation, socialisation, recreation and leisure. Rehabilitation strives to maximize physical, functional, psychological, social and role status, and is beneficial for people with musculoskeletal disorders. Occupational therapy aims to improve a person's ability to perform daily activities and valued life roles at home, work, leisure and social situations to facilitate successful adaptations in lifestyle and to prevent or minimize functional and psychological problems. A wide range of interventions is used for people with musculoskeletal disorders to maximize their participation. These interventions include restoration of self-care, work, and leisure, self-management education (physical and psychological), family education and support, environmental modifications, and provision of assistive devices. The type and amount of treatment provided will vary depending on the stage of the disorder and on other interventions that person is receiving. In chronic diseases such as rheumatoid arthritis, occupational therapist may be involved in treating the person intermittently over many years.

These occupational therapy interventions are based on several approaches or frames of reference.

- Biomechanical frame of reference is based on improving physical abilities such as joint range of motion, strength, and endurance. This approach is helpful in mobility training and maintain day to day physical activities.
- Learning frame of reference is a useful approach as people with musculoskeletal disorders need to be educated in the management of their condition. This is of twofold characteristics:

    a) cognitive approach which provides advice on time management and energy conservation

b) behavioural approach which provides pain and anxiety management techniques to cope with the everyday challenges.

- Compensatory frame of reference is used when the progressive nature of the condition lead to reduced function and requires compensation. This also consists of two approaches:

  a) adaptive approach which provides behaviour modification and rearrangement of their physical environment

  b) rehabilitative approach which provides alternative help or support in care assistance or use of appropriate assistive devices and equipment.

In the early stages, occupational therapy is designed to enhance behavioural changes such as energy conservation and joint protection to manage symptoms and prevent deterioration.

JOINT PROTECTION AND ENERGY CONSERVATION

The aims of joint protection and energy conservation in occupational therapy are to reduce pain, inflammation and internal and external joint stresses. It can also be effective in reducing fatigue. Joint protection includes respect for pain and altering movement patterns of affected joints.

RESPECT FOR PAIN

Ignoring pain may lead to further joint damage and pain, while being too sensitive to pain may lead to reduced activity and muscle wasting and lead to joint instability.

The Occupational therapist identifies the activities that may need alteration by asking the person with musculoskeletal disorders to self-monitor the amount of pain on selected days and activities or keep record of activities, pain and fatigue levels over the time. Both these strategies can increase the awareness of the need to use joint protection and energy conservation.

ALTERING MOVEMENT PATTERNS

Joint protection education is directed towards altering movement patterns at affected joints. The principles use here are as follows.

1. Distribute the load over several joints.

Occupational therapists teach how to increase the surface area by using hand or both hands e.g. using the palms of both hands to carry plates, mugs, trays etc. with the fingers in extension (Figure 22.1a).

2. Use stronger, larger joints.

Occupational therapists teach the advantage of using stronger and larger joints for activities, as the stronger the joints are able tolerate a given amount of load e.g. using the hip to close drawers or door (Figure 22.1b).

Figure 22.1 -
a) Using the palms of both hands to carry plates
b) Using the hip to close drawers.

3. Use joints in their most stable and functional positions.

Certain positions enable greater efficiency of muscles and leverage to be applied during an action. Most joints are stable in straight alignment. Flexed and deviated positions and applying rotational force during activity increases stress on ligaments.

Correct lifting techniques is an important aspect in preventing and correcting low back and knee injury (Figure 22.2)

Figure 22.2 - Correct lifting technique

4. Reduce effort

Using less muscular effort to perform daily tasks reduces internal joint stress. E.g. technical devices most commonly used in the kitchen are the jar opener, tap turner (Figure 22.3a) and easy vegetable peeler.

5. Avoid positions of deformity.

Stresses contributing to common patterns of deformity should be avoided e.g. finger twisting actions promote ulnar deviation. The palm should be used to press spray bottles (Figure 22.3b), turn taps and open jars with the fingers held straight.

Figure 22.3 - a) Tap turner, b) Palm to press a spray bottle

REST, POSITIONING, AND SLEEP

Inflamed and painful joints should be rested. However, research have shown that prolonged rest can have negative effects on muscle strength. Occupational therapists therefore encourage person with musculoskeletal disorders to have adequate rest and activities to enhance natural recovery process.

Advice is also offered on correct rest positions. It is important to support joints well when sitting and lying. Chairs should have a firm arm rest and adequate seat depth to support the upper legs and allow the hips, knees and ankles to be positioned at 90^0 -120^0 to have a supportive back.

Occupational therapists provide support to overcome sleep disturbances, by providing advice and discussions on ensuring that the bedroom is a comfortable environment (bed, noise, temperature and lighting level), avoidance of beverages containing caffeine before bedtime and setting aside a relaxing period prior to sleep (e.g. having a warm bath) and to have a regular sleep schedule.

ENERGY CONSERVATION

Energy conservation techniques can assist in reducing fatigue. The main principles of the energy conservation are as follows.

- Balance rest and work by taking regular short breaks during prolonged periods of activity.
- Encouraging correct body positions. Good posture balances the weight of the head and limbs on the bony framework so that the force of gravity assists in keeping correct joint position. More energy is used to maintain poor posture as muscles have to work against the effect of gravity to maintain the position. Hunched shoulders, craned neck and bent backs cause muscle tension, pain and tiredness.
- Avoiding staying in one position for a long period which can lead to stiffness. It is recommended to change the position every 20 – 30 minutes.

- It is important to have a correct work height which allows the head and neck to be held as straight as possible while sitting or standing. Work surface should be approximately two inches below elbow when the shoulders are relaxed.
- Advice to avoid activities that cannot be stopped if they become too stressful and could therefore, strain weakened muscles e.g. working with machinery in a production line of a factory.

RELAXATION, PAIN AND STRESS MANAGEMENT

Pain is a major concern in musculoskeletal disorders such as rheumatoid arthritis. It is a primary determinant of physical dysfunction and is associated with psychological symptoms such as helplessness and depression. A variety of cognitive behavioural techniques are used by occupational therapists to modify the perception of pain and behaviour.

When training relaxation, a common method employed is the use of guided imagery in which persons are asked to repeatedly imagine performing specified activities in a sequential manner without imagining any pain in the mental activity.

Occupational therapists often use Jacobsen technique for relaxation. This technique however involves muscle contraction and relaxation. Therefore, when selecting participants those who are with swollen joints are avoided to practice this technique. Occupational therapists teach cognitive coping strategies such as attention refocus by introducing pleasurable activities to engage and vivid imagery which is thinking about a pleasant scene or events that bring happiness to mind. These kind of techniques are coupled with joint protection techniques and problem-solving techniques such as identifying activities causing pain or events which increase stress and introduce methods to resolving them.

SPLINTS

Splints are used to relieve pain and decrease local inflammation. Correctly positioned joints minimize joint contractures, increase joint stability, improve strength, range of movement, function, and prevent

or correct deformity. Splints can be divided into resting and dynamic splints (or orthoses). These are commonly provided in rheumatoid arthritis. Occupational therapists use plastic prefabricated form materials and elastic materials to fabricate splints. There are two major types of splints.

1) Resting splints are usually worn at night and for short periods in the day if necessary for pain relief and inflammation reduction. Generally, the joints are supported in an anatomically correct position (Figure 22.4a).

2) Dynamic splints enhance the unstable joint to remain stable while assisting weak muscles to perform movements and function (Figure 22.4b).

Figure 22.4 - a) Rest splint. b) Dynamic wrist splint

JOINT PROTECTION

Joint protection maintains functional ability by altering working methods and involves educating in proper joint and body mechanics and by the use of appropriate assistive devices. Theoretically, reducing the load and effort required to perform daily activities reduces strain on joint structures, pressure on pain receptors, localized inflammation, and fatigue.

ENVIRONMENT MODIFICATION

Assistive devices and environmental modifications reduce dependence and maintain function by compensating for muscle weakness and movement limitations, as well as improving safety aspects.

SAFETY AT HOME

Occupational therapists assess home environment and provide suggestions to make the home a safe place for persons with musculoskeletal disorders. Loose rugs, worn carpets and slippery floor surface should be changed or eliminated to minimize falls. Stair rails and banisters should be securely fixed. The rails should provide a comfortable grip. Height of the rail is decided to the height of the person and requirement of the person with musculoskeletal disorders. Prior to providing guidelines to change the home environment, the occupational therapist should always adopt a sensitive approach with a clear explanation of the reasons for changing the person's own home.

ACCESS TO HOME

Whenever possible, the occupational therapist should conduct a home visit to assess the accessibility issues. The front entrance should be accessible to the user. Steep steps can be made easier to negotiate by adding a half step and also by the use of a grab handle fixed near the door. If the person is unable to walk, then occupational therapist assesses the entrance for a ramp to replace the steps. Mobility inside the home is also assessed. Strategically placed grab rails can be helpful to prevent falls and also to support when walking. Changing the arrangement of furniture can provide a safe and comfortable mobility inside the home. Some doorways may be exceptionally narrow to pass through with a walking frame or wheelchair. Bathroom and toilet doors are the most common examples. Occupational therapists consider adaptation or widen the doorway to provide access to such places.

EQUIPMENT FOR EVERYDAY LIVING

The occupational therapist delivers advice and recommend equipment for reducing the strain on affected joints. Alternative methods of performing tasks which may be less stressful to joints are also suggested.

1) Bathing

Bathing can be difficult when musculoskeletal disorders affect upper and lower limbs. So, equipment such as bath boards, bath seats and non-slip mats be considered with proper installation instructions.

2) Toileting

People with musculoskeletal disorders may find commodes of lower seating level difficult to use. This can be eased by raising the commode seat height. A grab rail or frame may also be useful to support when sitting and getting up from the commode. If a person using a squatting pan commode find it hard to use, a commode chair or installation of a seat commode is advised. In the presence of financial constraints, liaison needs to be done to acquire funds from the social service department.

3) Dressing

Training the person to be seated at a comfortable height while dressing and to make use of assistive devices such as long handle reacher (Figure 22.5a) and easy fastening methods can be done.

4) Eating

In musculoskeletal disorders, the ability of using fingers for eating and drinking is usually affected due to swelling, pain and deformities. These symptoms slow down the pace of eating and drinking as the disease progress. Therefore, a person with musculoskeletal disorders is supported with maintaining correct posture and movements. When deformities prevent proper use of fingers or cutlery to eat, assistive devices such as adapted cutlery are fabricated (Figure 22.5b).

5) Attending to Work

Many people with musculoskeletal disorders in employment, find problems at work, especially with the pain, mobility and hand function. If the person finds it difficult to hold a pen, occupational therapist helps to increase the diameter of the pen by introducing pen grips or padding the pen to increase the diameter (Figure 22.5c).

Figure 22.5 - a) Reacher b) Adaptive cutlery c) Pen adaptations

Occupational therapists introduce labour saving devices and environment modifications to create a comfortable work environment e.g. ramps, stair lifts etc.

6) Leisure

Encouragement should be provided to maintain existing leisure activities and hobbies. These types of enjoyable activities enhance both physical and emotional wellbeing. In leisure activity such as gardening, instructions are provided to use a stool to sit instead of kneeling when weeding plants. Advice is given to use gloves to protect hands. Methods to distribute weigh over the strong joints and large surface area and using raised flower beds or containers for easy planting and handling are taught.

CONCLUSION

Evidence suggests that seeing an occupational therapist is beneficial for people with musculoskeletal disorders to be independent in activities of daily living, work and leisure. When a person is referred, the occupational therapist conducts an in-depth assessment to identify functional problems and demonstrates ways to practice difficult tasks more easily or provide advice about using strategies, techniques, gadgets, or equipment and new technologies. The occupational therapist may also suggest some useful changes at work or home environment to promote and prolong health and wellbeing of persons with musculoskeletal disorders.

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23. A Synopsis of Arthroplasty

Dr. Chandana Karunatileke

JOINT IN THE PERSPECTIVE OF ORTHOPAEDICS

Anatomically a joint can be described as a physical location where two or more bones connect each other. They comprise of several different elements of connective tissues such as tendons, ligaments and cartilage. Furthermore, they are surrounded by a fibrous capsule that limits the bone movements. The inner surface of this capsule produces synovial fluid which lubricates the joint. Joints allow movement of bones in guided specific directions and assists in locomotion.

Arthritis, especially of the hip and knee typically causes significant and progressive pain along with deterioration in function, ambulation, and mobility. It is one of the leading causes of physical disability in the older population. Total joint replacement surgery provides pain relief and restores function in individuals. Advances in technology, surgical technique, and perioperative care over the past few decades have made total hip arthroplasty (THA) and total knee arthroplasty (TKA) more suitable for a wider age group of patients, including those older than 80 years. Validated outcome measures indicate that there is no ceiling on the age at which joint replacement surgery should be considered.

ARTHROPLASTY

Patients with significant pain and functional disability due to pain and disability are candidates for referral to an orthopaedic surgeon for arthroplasty. A painful knee or hip can make life miserable. The decision for joint replacement rests upon how much it hurts and how much it is affecting the patient's life. Furthermore, it should be arrived after carefully balancing the patient symptoms and disability against his or her physical requirements. The following criteria needs to be considered.

1. Patients having significant pain
    a) which keeps them awake at night despite the use of medications

b) that keeps the patient preventing from being able to walk or bend over
c) that is not relieved by rest
d) that is not helped by non-surgical approaches.

2. Patients that can no longer complete routine daily tasks without help.
3. When patients have either primary or secondary osteoarthritis and feel that the disease is wearing patient down physically, emotionally, and mentally.
4. When patient is suffering from severe side effects from the medications taken for painful knee or hip.
5. When investigations show advanced arthritis or significant joint damage. (Non-invasive treatments such as medications and physical therapy are of limited value in advanced arthritis)

The goals of arthroplasty are to

a) relieve pain
b) aid the joint to work better
c) improve the mobility of the person.

Although hips and knees are replaced the most often other joints such as shoulders, interphalangeal joints, ankles, and elbows are also regions that are intervened with surgery. Joint replacement surgery removes the damaged or degenerated parts of a joint and replaces them with new, metallic and plastic parts and liners.

COMPLICATIONS AND SPECIAL CONSIDERATIONS

Multicentre studies elaborate lower rates of postoperative complications following joint replacement. This is most likely due to increased awareness and medical screening. With the advancement of anaesthesia, orthopaedics, orthogeriatric, post-operative care and implant technology, the risks following joint replacement surgery are minimal. The overall rate of complications within 30 days of surgery in a review of more than 10,000 patients after elective joint replacement was 2.2%. This included myocardial infarction (0.4%), pulmonary

embolism (0.7%), deep venous thrombosis (1.5%) and death (0.5%). The frequency of these events can increase with age.

Infection is a relatively rare but potentially devastating complication can occur following joint replacement. Infection rates vary from about 0.2% to 2.5%. The risk of infection is slightly higher in TKR than in THR. Risk factors for infections include age, diabetes mellitus, rheumatoid arthritis, a previous surgery on the same joint, chronic infection elsewhere in the body, chronic kidney or liver disease, malnutrition, steroid use, smoking, and early wound complications or hematoma. Other risk factors may include a higher American Society of Anaesthesiologists (ASA) score, morbid obesity, bilateral surgery, allogenic blood transfusion, atrial fibrillation, myocardial infarction, and prolonged hospitalisation.

There is evidence that prolonged wound bleeding, haematoma formation at the surgical site and reoperation for evacuation of haematoma places a patient at higher risk for deep infection. For this reason, wound care and monitoring by the medical personnel, nursing staff, and therapists is important.

Periprosthetic joint infections may be superficial or deep. However, all infections should be considered deep until proven otherwise in order to avoid devastating complications.

The incidence of venous thromboembolism following joint replacement with thromboprophylaxis is extremely low.

Postoperative delirium is a common and serious problem in hospitalized older patients following elective joint replacement. Patients experiencing postoperative confusion and delirium can be disruptive and distressing to other patients and the health care team. Risk factors for postoperative delirium include age, pre-existing cognitive impairment or dementia, history of delirium and a history of alcohol dependence. Several preventative strategies have been employed including the use of regional anaesthesia, limiting use of postoperative narcotics, rapid mobilisation and return to familiar environments, and antipsychotic medications.

Advancement in physiotherapy resulted in early mobilisation and reduce the joint stiffness. Pain following joint replacement can be managed with patient controlled epidural lines and physiotherapy.

Wearing away of the joint surface may become a problem after 15 to 20 years.

ADVANTAGES OF JOINT REPLACEMENT

Total joint arthroplasty has become an extremely effective and successful treatment for the hip and knee. These procedures are among the most successful treatments available as quantified by several measures including pain relief, improved walking, improved self-care and function, and increased quality of life years gained. There is also substantial evidence that older patients undergoing total joint arthroplasty receive these benefits along with an associated decrease in health care costs as compared with usual care.

Joint replacement had documented significant improvements in health outcome including improved pain, emotional reaction, sleep, and physical mobility as early as 3 to 12 months after the operation.

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