Kerala Journal of PMR

2021

Vol 18 issue 3

FROM THE PRESIDENT'S DESK

The Covid scenario continues to haunt us. It is with a heavy heart that we mourn the loss of one among our fraternity, Dr. Rajagopal, who succumbed to Covid-19. Our prayers are with the bereaved family.

Into and past the second wave, the hurdles thrown before us reaffirm the need to promote and develop rehabilitation strategies in combating the psycho-physical and functional challenges faced by the patients. The need of the hour is to safeguard the specialty from being usurped by the so called 'unsung heroes'. Rehabilitation in post Covid times needs to be contoured to cater to both the differentially abled and the Covid challenged. The members of our chapter have been rendering yeoman services by actively engaging in frontline health care activities while diligently contributing to enhancing the significance of Physiatry. Kudos to all! Time is ripe for improvising and reorienting the outlook and scope of the specialty making it indispensible in post Covid management.

The specialty has always benefitted from the sound counsel of pioneering faculty. And our younger generations have always enthusiastically imbibed the best from them. In this context it would be most appropriate to recall the contributions of Dr. S. Abdul Gafoor, who recently retired as the Head of department of PMR, Medical College Thiruvananthapuram. After three decades of glowing service across the entire stretch of Kerala, Gafoor Sir steps down after a highly productive career that saw many improvisations and developments in the institutions where he was the Head.

My personal association with Gafoor Sir dates back to my PG days in Trivandrum in the early nineties when he was the dynamic tutor for an impressionable set of eager students. Even my first lessons of practice outside the hospital were learnt through his guidance. One among the earliest holders of MD in PMR, his academic credentials oversaw the evolution of the specialty. His easy going nature and his camaraderie set him apart as a wonderful Head and Colleague to all who had the privilege of working with him. I was lucky to experience this both as his student and later on as his colleague. Associates in various medical colleges fondly recall the genuinely humane concerns and highly approachable demeanor that distinguished Gafoor Sir. His recent stint at Medical College Kottayam was a testimony to his capabilities to lead a department and effectively infuse enthusiasm in all members of the staff. The focus on PG teaching ensured top performance from the residents bringing glory to the department. His focus on acute rehabilitation, subspecialty clinics and interest in research, all signal an exemplary future for the specialty.

Dr Abdul Gafoor will no doubt continue to render his expertise to the development of the specialty in future. On behalf of all of us, I wish Sir a productive and contented life ahead.

The journal of Kerala Chapter of IAPMR has earned special recognition at the National level. Dr. Ravi Sankaran & his team deserve special mention for their sustained effort in bringing out a much appreciated journal.

Wishing everyone good health & happiness.

FROM THE EDITOR'S DESK

Dear Readers,

Rheumatological rehabilitation is one of those topics Physiatrists can expound in detail. Prior to becoming an Internal Medicine subspeciality, PMR was one of the primary care providers for Rheumatology patients. In some ways we still are, when one considers people not the disease. This issue is packed with interesting articles from our clinicians. Everything from Psoriasis, to inflammatory arthritis, to fibromyalgia and hemophilia are covered here. Dr.Nittu, the pioneer of radiosynovectomy in Kerala, shares her expertise for the benefit of all. The smorgasbord inside is great for PGs and senior clinicians.

I have some good news to share. The KJPMR board has been formed. It is twelve members strong, welcome. Reeba madam oversees the communicator panel. Bineesh oversees the lifelong learner panel. We have space for more active members.

As always submissions are welcome. Please ensure they are in a word document and turned in by the deadline. Our next issue's theme is Meningomyelocoele. Happy reading.

TABLE OF CONTENTS

"Articles are the responsibility of the Authors"

Approach to a patient with Musculoskeletal and Joint Pain

Dr. Sreejith K, Professor and Head Dept of PMR MCH Kottayam

Evaluation and treatment of a patient with Musculoskeletal and joint pains forms a major chunk of cases in an outpatient setting of a physiatrist practicing musculoskeletal medicine. As clinicians we all follow non-analytical clinical reasoning based on evidence and solid experience. A two-step approach described in EULAR Rheumatology course material will be useful for approaching a patient with musculoskeletal complaints. The first step aims to define the generic type of pathology, or to set a main pattern. The second step is a more detailed investigation, which is adapted to each pattern and aims to differentiate between potential causes of that pattern, thus making a final differential diagnosis. Knowledge in electro diagnosis and ultrasound is an added advantage for physiatrists practicing musculoskeletal medicine

The main patterns are

1. General pain syndrome
2. Regional pain syndrome
3. Articular
4. Bone pain
5. Neck pain and back pain
6. Muscle syndrome
7. Systemic syndromes

Regional Pain

Regional pain syndromes are characterized by pain that affects a single musculoskeletal area (e.g., shoulder, hand or knee).

Regional pain may be

- Periarticular pain
- Joint pain
- Neurogenic pain
- Referred pain.

Periarticular pain originates in anatomical structures that are involved in joint motion but are located outside the joint capsule. These structures are often site of disease due to repetitive stress injuries or as local manifestations of systemic synovial diseases, like rheumatoid arthritis (RA). Inflammation of the tendon and ligament insertion points (enthesopathy or enthesitis) may occur in isolation, such as after trauma, and occasionally may have no apparent cause. On the other hand, recurrent and multiple enthesopathies can be a feature of seronegative spondyloarthropathies.

On clinical examination, patients with periarticular lesions will have more pain on active motion than on passive movement. Passive movement is not limited in periarticular lesions. This is to be expected as the involved structure remains rested during passive movement. Conversely, patients with joint lesions tend to have pain of similar intensity on both active and passive movement and may show equal limitation of both.

Resisted movements involving the periarticular structure will cause intense pain. Such maneuvers do not cause aggravation of pain when the problem is purely intra-articular. Palpation directly onto the inflamed periarticular structure will reproduce the patient‘s pain, whereas with arthritis tenderness is maximal along the joint line

Joint Pain

Unlike periarticular pain in joint pain all movements will be painful. Similarly both active and passive movements will be painful. Joint movement is often limited due to swelling or structural damage.

Neurogenic pain

Neurogenic pain is caused by compression or irritation of nerve roots or peripheral nerves. Eg.Radiculopathies, CTS ,TTS etc.

Referred Pain.

Referred pain denotes symptoms felt at a distance from their anatomical origin. For example, although hip pain is often felt maximally deep in the anterior groin, it may present as knee pain with no proximal discomfort. Usually children with Perthes disease may present with knee pain, so it is very important to examine the adjacent joints also. Localized musculoskeletal pain can also be referred from internal viscera. Such pain has an uncharacteristic rhythm, being more affected by the physiology of the viscera than by joint movement, and may be accompanied by other more suggestive symptoms. Local musculoskeletal examination is normal and does not reproduce the patient‘s pain.

Generalized pain syndrome

Fibromyalgia accounts for the majority of cases of ‘multiple regional pain‘ or ‘chronic widespread pain‘ syndrome. Patients with fibromyalgia may present with different patterns of dominant symptoms, which can be misleading. It is not uncommon for these patients to be misdiagnosed and treated for a variety of conditions, including RA, ankylosing spondylitis (AS), connective tissue disease, disc herniation, etc. The diagnosis is purely clinical and depends on demonstration of widespread hyperalgesic tender sites (i.e., lowered pain threshold) affecting all four body quadrants. Similarly before arriving at a diagnosis of FM do minimum investigations like Blood routine, CRP, TSH etc

Low back pain and neck pain

These are the most common conditions seen in a general PMR practice. Most cases of back pain resolve within 3 to 6 weeks. In the vast majority of cases it will be impossible to make a precise diagnosis and treatment will be guided by general rules of pain management, exercise and risk factor control. Only a small percentage of cases involve a specific etiology requiring special diagnostic and therapeutic action. In patients younger than 45 years, about 5% of cases of chronic low back pain are due to spondyloarthritis. Features suggestive of inflammatory back pain, such as night pain and improvement of back pain with movement but not at rest, may be indicative of spondyloarthritis. In a very few cases the underlying causes are serious—such as malignancy and infection—and require urgent investigation and specific treatment. Such diagnoses cannot be missed. Avoid inappropriate imaging in back pain because it will complicate the clinical picture.

The recommended strategy for back and neck pain is based on the search and recognition of alarm symptoms and signs that indicate a higher probability for an underlying specific cause. Important points of differentiation from common mechanical pain include: progressive pain, night predominance, lack of relationship to spinal movement, and involvement of the thoracic spine.

Articular syndrome

Four fundamental features of the articular pattern are

Inflammatory or non-inflammatory

Acute or Chronic

Mono, oligo or polyarticular with or without axial involvement

Extra articular/systemic manifestations

The most important task is to differentiate between degenerative arthritis and inflammatory arthritis. Osteoarthritis is typically associated with ‘mechanical‘ or ‘usage-related pain’—pain that increases with repeated use of the joint, which is relieved by rest, and which is often worst towards the end of the day. Patients may describe pain that increases again after resting and this may be accompanied by ‘gelling‘, stiffness that subsides after just a few minutes. Early morning stiffness associated with OA is usually relatively brief, and is ‘worn off‘ in well under half an hour.

Conversely, in active inflammatory disease, pain is worst in the morning and is relieved as they get up and start to move their joints. Morning stiffness is often prolonged, lasting for more than 30 min and sometimes for several hours. Stiffness after rest may persist for more than 5 mins.

Patients with inflammatory arthritis may additionally complain of non-specific features such as fatigability, weight loss, night sweats. With inflammatory multi-system diseases, there may also be symptoms and signs of extra-articular manifestations (e.g., anterior uveitis, skin lesions, and lung or bowel problems).

Physical examination in case of OA shows coarse crepitus, localized joint-line tenderness and/or bony swelling (osteophyte) along the joint margin. Deformity may also be present in later stages of OA.

In inflammatory diseases, the synovium becomes inflamed, engorged and eventually hypertrophied, and the volume of synovial fluid increases. This increase in soft tissue and fluid occurs within the limiting confines of the capsule and can increase the pressure within the joint causing pain, stiffness and restriction of movement. A joint with increased intra-articular pressure is most comfortable in the position that minimizes the pressure. This position, generally mild to mid flexion, is mainly determined by the tightness of the capsule and is termed the 'loose-packed' position, in which the capsule is normally at its loosest and therefore can accommodate an increase in fluid and soft tissue. Conversely, the positions in which the capsule is naturally tight—the 'tightpacked' positions at the extremes of range of movement—are the positions that are the first to be painful when synovitis is developing, and the first movements to become restricted. As the inflamed joint is moved, actively or passively, through its range of movement the pain is minimal or absent in the loose packed position but increases as the joint moves into any of the tight packed positions. This uneven distribution of pain, maximal in all tight pack positions, is called 'universal stress pain'—the most sensitive sign of synovitis, occurring even before there is visible swelling or restricted movement

Patterns of inflammatory joint disease

It is better to represent the patient's condition with as few words as possible—for example, 'asymmetrical oligoarthritis, with inflammatory back pain' or 'acute arthritis with fever and rash'. This will make the final diagnosis a lot easier and reliable. The most common patterns are

- *Number of joints affected*

Monoarthritis: one single joint affected

Oligoarthritis: 2–4 joints affected

Polyarthritis: >4 joints affected

- *Acute versus chronic*

Acute: onset in hours or days

Sub-acute: up to 6 weeks

Chronic: onset over weeks (more than 6 weeks) or months

- *Additive versus migratory*

Additive: the affected joints are added progressively

Migratory: the inflammatory process flits from one joint to another

- *Persistent versus recurrent*

Persistent: once it has set, the arthritis persists over time (persistent ≥6 weeks)

Recurrent: episodes or crises of arthritis separated by symptom-free intervals.

- *Predominantly proximal versus predominantly distal*

Proximal: arthritis mainly affects large joints—that is, proximal to the wrist or ankle and the spine

Distal: the arthritis mainly affects the small joints of the hands and feet, with or without the wrist and ankle

Large and small joints affected—there is a mixture of joint sizes

- *Symmetrical versus asymmetrical*

Symmetrical: affects approximately the same joint groups on each side of the body

Asymmetrical: there is no clear relationship between the joints affected on either side of the body

- With or without inflammatory low back pain
- With or without systemic manifestations

Monoarthritis

It is useful to separate acute from chronic arthritis. Most common cause of acute arthritis is Crystal arthritis. Most common crystal arthritis is Gout.

Initial episodes of gout almost always follow a monoarticular recurrent pattern with symptom-free intervals, predominantly in the lower limbs. With time, intervals become shorter, the upper limbs also become involved, and more than one joint can be affected in each episode, occasionally leading to a polyarticular pattern. Acute calcium pyrophosphate (CPP) crystal arthritis (‘pseudo gout‘) presents a similar clinical picture but is mainly restricted to patients over the age of 60 years. It particularly targets the knee. Other common sites are the wrist, shoulder and ankle, although almost any joint can be affected. While a confident diagnosis of crystal synovitis may be made from the clinical characteristics, definitive diagnosis requires demonstration of sodium urate or CPP crystals in aspirated synovial fluid.

Not all patients with hyperuricemia develop gout. Uric acid levels may fluctuate and possibly decrease during an acute episode, but if normal, measuring uric acid 2 to 3 weeks after an acute attack has resolved will provide an accurate baseline and may aid in establishment of the diagnosis. Acute gout cannot be excluded by the presence of a normal serum urate level.

Estrogen protects women from gout because of its association with enhanced renal excretion of uric acid and estrogen in hormone replacement therapy has a similar effect. Gout occurs in only 0.2 per 1000 cases of women younger than 45 years. Women with gout tend to be older and have more comorbidities, such as obesity and kidney disease, compared with men. Diuretic use as opposed to dietary triggers is more common in women than in men with gout. Elderly women, particularly those taking diuretic agents, may have an initial polyarticular presentation, upper extremity dominant disease, or tophi that may be confused with rheumatoid nodules. Tophi may also be present in Heberden nodes. Approximately 25% of patients with gout have a family history.

Disease 5 to 10 years after onset may show tophi and typical erosions, called rat bite erosions, which have overhanging edges. Joint space is preserved until later in the course of the disease.

Ultrasound is more sensitive than radiography for identifying erosions. Ultrasonography (US) detects changes in gout in the form of the double contour (DC) sign and synovial hyperecchoic foci (SHEF) which is considered to represent MSU deposits on the articular cartilage or in synovium respectively. The location of these US changes may guide the physician where to aspirate relevant synovial tissue to examine for MSU. The double-contour sign should be differentiated from a cartilage-interface sign, which is dependent on the angle of insonation.

Although the MTP is the most common joint involved and may appear to have gout, gout is not always the correct diagnosis. Ultrasound also facilitates assessing the effectiveness of urate-lowering therapy (ULT) because the tophi and double-contour sign regress with successful treatment.

Ultrasound is a valuable tool for guiding arthrocentesis of synovial fluid. In 1 study of primary care internists, US-guided aspiration was successful in 93% vs 52% of patients when palpation was used. Ultrasound also facilitates local corticosteroid injections into an affected joint. Also, US guidance can be used for dry needling, a technique used for aspirating MSU crystals in an asymptomatic joint in a patient without a synovial effusion but with suspected gout. In 1 study, 29% of patients with asymptomatic hyperuricemia had evidence of MSU deposition. However, patients with gout tend to have more erosions and synovitis than asymptomatic patients with hyperuricemia and MSU deposition. Some concern has been expressed about

differentiating gout from asymptomatic hyperuricemia. However, recent findings showed that the double-contour sign and tophi retain high specificity and allow for differentiation between the 2 diagnoses.

Acute calcium pyrophosphate (CPP) crystal arthritis (‘pseudo gout‘) presents a similar clinical picture but is mainly restricted to patients over the age of 60 years. It particularly targets the knee. Other common sites are the wrist, shoulder and ankle, although almost any joint can be affected. While a confident diagnosis of crystal synovitis may be made from the clinical characteristics, definitive diagnosis requires demonstration of sodium urate or CPP crystals in aspirated synovial fluid. In recent years US has emerged as a valuable tool in diagnosis of CPPD. Ultraound is able to detect CPP crystals in hyaline cartilage, fibrocartilage, tendons and synovial fluids. The definitions used for the identification of the US features of CPP deposits were divided depending on the anatomical structure under examination. In particular, different definitions were used for CPP deposition at the level of hyaline cartilage, fibrocartilage, tendons and synovial fluid. In hyaline cartilage, CPP crystals are seen as hyperechoic deposits, placed within the layer of the cartilage, that reach large dimensions. At the level of the fibrocartilage, CPP crystals usually appear as hyperechoic, rounded or amorphous-shaped deposits placed within the structure. In the tendons, CPP crystals are usually seen as linear deposits within the fibrillar echotexture (multiple or single lines or thick solid band), but in some studies these deposits are described also as punctate. Finally, in the synovial fluid CPP crystals appear as hyperechoic spots or ovalar aggregates.

Septic arthris presents with acute or subacute arthritis with progressive signs and symptoms. Systemic symptoms such as fever and malaise may be present. Always do a synovial fluid analysis in a case of acute progressive monoarthritis because septic arthritis is a medical emergency. Other causes of acute monoarthritis include post-traumatic synovitis, palindromic rheumatism, reactive arthritis, psoriatic arthritis, and bacterial endocarditis.

Monoarthritis can have an indolent course, lasting from weeks to months. The main possible causes of chronic inflammatory monoarthritis include infection (Brucella, Mycobacterium, Borreliosis, others), monoarticular presentation of oligo- or polyarthritis (juvenile idiopathic arthritis, reactive arthritis, seronegative spondyloarthropathy), and a foreign body (e.g., plant thorns). Differential diagnosis must incorporate causes of non-inflammatory arthropathy, such as OA, recurrent hydrarthrosis, osteonecrosis, chronic regional pain syndrome, neuropathic (Charcot‘s) joints and tumours, including pigmented villonodular synovitis. The etiological diagnosis of chronic monoarthritis usually requires a synovial biopsy with pathological and bacteriological examination and in selective cases PCR technique. In a large number of cases, no specific cause can be identified.

Chronic symmetrical additive peripheral polyarthritis

This pattern describes joint inflammation involving, simultaneously, five or more joints (polyarthritis), for more than 6 weeks (chronic). Small joints of the hands and feet are predominantly affected, with or without the wrist, ankle and knee (peripheral), and approximately the same joints are involved on each side of the body (symmetrical). The affected joints have been added progressively (additive) over a relatively short time period (several months). There should be no inflammatory low back pain. The most common cause for this pattern is RA. Onset of especially aggressive disease may be accompanied by constitutional features such as fever and lymphadenopathy. This could suggest a ‘systemic syndrome‘, but articular manifestations dominate the clinical picture in the vast majority of patients. Other systemic manifestations may occur, but they are usually milder than the arthritis and tend to occur later in the course of disease

This pattern of arthritis is common to other rheumatic conditions, especially other connective tissue diseases, such as SLE, primary Sjögren's syndrome, polymyositis (PM) and mixed connective tissue disease (MCTD). Psoriatic arthritis can also present with this pattern, although it tends to be more asymmetrical and involve either the distal interphalangeal or the sacroiliac joints. OA with CPP crystal deposition (CPPD) deserves consideration, especially in older patients. A pre-existing pattern of generalized OA with recurrent inflammatory episodes may suggest this condition. Chronic polyarticular gout may have a similar pattern of distribution. However, almost invariably this is preceded by a period of recurrent monoarthritis. Viral arthritis, such as is associated with parvovirus B19, HIV and hepatitis, may follow a similar pattern, but tends to have a more acute onset than RA or the connective tissue diseases. The clinical contribution to differential diagnosis relies on careful evaluation of extra-articular manifestation. Further investigations may be useful.

Chronic asymmetrical oligo/polyarthritis

This describes an asymmetrical arthritis present for more than 6 weeks (chronic), affecting two or more proximal or distal joints (oligo/polyarthritis). Dactylitis, enthesitis or involvement of distal interphalangeal joints is a common but not mandatory feature. The combination of synovitis with adjacent periarticular inflammation (one example being dactylitis) is strongly suggestive of seronegative spondyloarthropathy. Psoriatic arthritis is the most common cause of this pattern. A personal or family history of psoriasis will provide support for this diagnosis. When the arthritis is limited to proximal and distal interphalangeal joints, hand OA needs to be considered as an alternative diagnosis. Although this usually has a slow development, it may evolve with acute inflammatory flares of these joints. Chronic sarcoid arthropathy may evolve with a similar pattern—the inflamed joint tends to show a nodular asymmetrical aspect as opposed to the spindle shape of psoriatic synovitis. Other seronegative spondyloarthropathies, such as AS, may also cause this pattern of arthritis, mainly asymmetric oligoarthritis. Inflammatory back pain is expected in this condition, but may not be obvious. Reactive arthritis or arthritis associated with inflammatory bowel disease may show this pattern, but when distal joints are affected they usually adopt a symmetrical distribution. Other causes include incipient RA, juvenile idiopathic arthritis, polyarticular gout, OA with CPPD, and Behçet's disease. Again, careful enquiry about axial and extra-articular manifestations including enthesopathy and uveitis may provide critical clues to the diagnosis.

Proximal oligoarthritis

Patients may present with inflammatory arthritis involving predominantly proximal joints. The most common causes are the seronegative spondyloarthropathies. Complete questioning and physical examination is important for differential diagnosis. The presence of inflammatory low back pain, a personal or family history of psoriasis, inflammatory bowel disease, AS, uveitis, and the occurrence of any infectious disease (infectious diarrhea, urinary tract infection) in the weeks preceding arthritis must be explored in the enquiry. Other causes of proximal oligoarthritis include Behçet's disease, juvenile idiopathic arthritis and incipient RA. It is not uncommon for arthritis with this pattern to defy formal classification and is better described as unclassified oligoarthritis.

Polymyalgia rheumatica is in the differential diagnosis of proximal oligoarthritis. The patient is typically an elderly person, presenting with pain and stiffness in the neck, shoulders and hip girdle due to inflammation that mainly affects the peri-articular structures (tenosynovial sheaths, bursae). Peripheral joints can be involved. Some extra-articular manifestations must be explored in questioning. Malaise, fever, fatigue, anorexia and weight loss are common features. Jaw claudication, temporal

headaches and loss of vision suggest concurrent temporal arteritis.

Acute oligoarthritis or polyarthritis:

Febrile arthritis and arthritis with manifestations of the skin and mucous membranes

This is characterized by a rapid onset of oligo- or polyarthritis, often associated with a recent infection, fever or changes in skin and mucous membranes. This pattern suggests the possibility of reactive arthritis. In these cases, the patient is most often a young adult man. The enquiry should explore the occurrence of a significant infection within the 2–3 weeks before the arthritis. There is a wide variety of potential causal agents, but most frequently this will be gastrointestinal, genitourinary or upper respiratory infection. Reactive arthritis is often associated with manifestations in the skin (e.g., rash, erythema, keratoderma blenorrhagica) and mucosae (conjunctivitis, urethritis, oral or genital ulceration, circinate balanitis). The pattern of articular involvement varies. Proximal oligoarthritis is the most common, but in cases of poststreptococcal reactive arthritis or post-viral arthritis, such as parvovirus, HIV, hepatitis C and B and tropical viruses, it is often polyarticular and symmetrical. Still‘s disease (in adults or children) is characterized by an acute or sub-acute onset of arthritis, associated with fever, evanescent skin rash, weight loss, lymphadenopathy and/or splenomegaly. SLE, dermatomyositis (DM), Behçet‘s disease and, occasionally, RA may present with similar features and should be considered in the differential diagnosis. Lofgren‘s syndrome is a type of acute sarcoidosis that is most commonly accompanied by bilateral ankle arthritis and fever, erythema nodosum especially of the legs. A chest radiograph will commonly reveal bilateral hilar lymphadenopathy.

Inflammatory back pain

Inflammatory back pain is axial and symmetrical, affects multiple segments, is not relieved by rest but rather by movement, and is associated with prolonged morning stiffness. Low back pain may radiate to the buttocks and patients may describe alternating buttock pain.

Inflammatory back pain is a typical manifestation of seronegative spondyloarthropathies: non-radiographic axial spondyloarthritis, AS, psoriatic arthritis, reactive arthritis, and spondylitis of inflammatory bowel disease. Other conditions to consider are Behçet‘s disease and infectious or aseptic discitis. In seronegative spondyloarthropathy, the spine is very often affected and may or may not be accompanied by involvement of the peripheral joints. When the condition is strictly limited to the spine, non-radiographic axial spondyloarthritis or AS becomes the most probable diagnosis. If peripheral joints are also affected, the pattern of distribution and the associated manifestations are the key to the final diagnosis. Psoriasis, inflammatory bowel disease, uveitis, urethritis, conjunctivitis, venous and arterial thrombosis, and mucosal ulcerations must be explored in the history and examination.

Patterns of OA

OA typically develops in middle-aged or elderly people and in some joints, such as the hip or knee, symptoms may get slowly worse with time, whereas in others, such as finger interphalangeal joints, symptoms may improve. Target sites of OA are sternoclavicular joint, acromioclavicular joint, first MCP joint, PIP and DIP joints, Hip, Knee and first MTP joint. In axial skeleton target sites are cervical and lumbar spine. However, although joints such as the shoulder and elbow are not target sites for OA, the most common cause of arthritis in these joints is OA simply because of the very high prevalence of OA compared to other arthropathies.

Nodal generalised OA is characterised by multiple Heberden‘s and Bouchard‘s nodes and involvement of other common target sites for OA. It predominates in middle-aged and older women and there is often a family history. ‘Atypical‘ OA is suggested by uncommon characteristics, such as onset at a young age or an unusual joint distribution, in

a patient who has usage-related pain and signs of joint damage, without inflammation. Such features should alert the clinician of the possibility of an underlying predisposing condition. However, young-onset pauci- or polyarticular arthritis that clinically, and even radiologically, appears as OA should lead to consideration of specific conditions that predispose to joint damage such as Spondylo-epiphyseal and epiphyseal dysplasia (may have short stature and family history) , pre-existing arthritis (e.g., prior juvenile idiopathic arthritis, septic arthritis) , Haemochromatosis (often targets hip, wrist, metacarpophalangeal joints) , Neuropathic joint (hindfoot—diabetes; shoulder, elbow, wrist—syringomyelia; knee, spine—syphilis) , Osteonecrosis (mainly hip, shoulder, knee, elbow) , Endemic osteochondropathy (e.g., Kashin-Beck disease, Mselini disease).

OA itself is a risk factor for crystal deposition (CPP, basic calcium phosphates and sodium urate). Therefore, if a patient with apparent OA has an additional superimposed inflammatory component, especially with 'flares' of clinically overt inflammation, examination of a synovial fluid aspirate, particularly for CPP and urate, is warranted.

Bone pain conditions

Bone pain can have different presentations. One form that is important to identify is pain characterised by deep, diffuse, continuous pain, unrelated to movement and not typically confined to joints. Frequently, pain will be worse at night and disturb the patient's sleep. These features should raise consideration of bone tumours, metabolic bone diseases or inflammation of the periosteum. Metastatic tumours are the most common neoplasms of bone, and bone is the third most common site of metastasis after the lung and liver. Pain due to a metastatic lesion may be the first symptom of the underlying malignancy. Common sites affected include the spine, the pelvis and the proximal segments of the limbs. The local examination is usually normal and does not reproduce the patient's pain. Another form of bone pain results from fracture. This pain may be more acute or subacute in onset, is very well localised without any radiation, and is often worse on minimal movement. Risk factors for osteoporosis and fractures include postmenopausal status, early menopause, late menarche, low body mass index (BMI), sedentary lifestyle, low dietary dairy intake, and family history of osteoporosis or fracture. Secondary causes include malabsorption, hypogonadism, hyperthyroidism, hyperparathyroidism, excess alcohol use, and prolonged glucocorticoid use.

Muscle symptoms

Myalgia is a frequent symptom of viral infections, but is also commonly associated with systemic diseases such as lupus, vasculitis and PMR. These may cause pain and muscle tenderness, but usually not weakness. However, primary muscle disease is most commonly reflected by proximal weakness and muscular atrophy. Myopathic patients may have difficulty going up and down stairs, getting up from a low chair or combing their hair, but their handshake is firm and they can walk on tiptoe. Examination may show decreased proximal muscular power and muscle tenderness. Muscle atrophy is a later and inconsistent finding. PM, DM and inclusion body myositis (IBM) are the most typical causes of this pattern. Arthralgia and low grade arthritis may also occur, especially when inflammation of muscle occurs as part of a connective tissue disease, such as systemic sclerosis or MCTD. Systemic complaints such as low grade fever, fatigue, weight loss and skin rashes are often present. The differential diagnosis of adult PM/DM must include a broad array of conditions capable of affecting skeletal muscle. Hypothyroidism, sarcoidosis, and metabolic myopathies must be considered in this clinical context, as the myopathy associated with these conditions may dominate the clinical presentation. Muscle pain and stiffness of the proximal limb girdles, characteristic of PMR, may be interpreted as myopathy, but with PMR muscle weakness

is not a feature. Drugs which can cause neuromuscular complications, include glucocorticoids, hydroxychloroquine, colchicine, cyclosporine, statins and fibrates . Non-inflammatory myopathies must also be considered and they include an enormous variety of neurological conditions, such as spinal muscle atrophies, myasthenia gravis, myotonic diseases, congenital myopathies and storage diseases. Generalised muscle pain with 'tender points' is an integral part of fibromyalgia can lead to a subjective feeling of muscle weakness which in most cases is not confirmed on physical examination

Systemic syndrome

Inflammatory joint diseases may be accompanied by extra-articular manifestations. Examining for extraarticular manifestations should be an integral part of the investigation. Commonly, these manifestations occur together with polyarthritis and sometimes they can dominate the clinical picture. The term 'systemic syndrome' is a term used to further explore the diagnosis.

Main systemic manifestations associated with rheumatic diseases include constitutional manifestations such as fever, weight loss, severe fatigue. Skin manifestations include photosensitivity, alopecia, Heliotrope rash, Gottron's papules, Raynaud's phenomenon, Psoriasis. Mucosal manifestations consists of oral and genital ulcers, dry eyes and mouth. Another important systemic manifestation is serositis. Others include arterial or venous thrombosis, recurrent abortion, dysphagia, dyspnoea, lower limb edema, hypertension, lymphadenopathy, muscular weakness convulsions, psychosis, peripheral neuropathy, sinusitis, otitis media, deafness diarrhea etc.

In assessing a patient with musculoskeletal complaints a good history and thorough clinical examination is of utmost importance. The history should be comprehensive and focused. Relevant information should be explored, but irrelevant should be discarded. Irrelevant information creates distraction. To master this ability requires a clear view of what is the key information and what needs to be taken into account during the diagnostic process or treatment selection.

Physical examination will play a major role in clarifying the diagnosis. Examination is extremely important to determine the anatomical origin of the pain (articular, periarticular, muscular, neurogenic, etc.) and the nature of the disease (degenerative, inflammatory, entrapment).

Laboratory tests have limited usefulness in the diagnosis of most rheumatic conditions. Their influence varies according to the clinical picture and they should be chosen to clarify a clinically supported hypothesis. Ordering a battery of laboratory tests will often introduce information noise and cost rather than clarify the diagnosis.

Musculoskeletal ultrasound is a valuable tool in assessing many musculoskeletal and rheumatological conditions. For example in hip joint arthritis features of inflammation and effusion may not be clinically demonstrable. Here ultrasound will be helpful in demonstrating effusion and synovitis.

Several studies have demonstrated that high frequency US is accurate for detecting joint effusion and synovitis compared with magnetic resonance imaging and direct arthroscopic visualisation. US is more sensitive and reproducible than clinical evaluation in assessing joint inflammation.

Power Doppler (PD) US is a new technique of colour Doppler that improves the sensitivity to detect flow from small vessels and low velocity flow at the micro vascular level. PD US detects indirect signs of increased vascularization associated with soft tissue musculoskeletal inflammatory and infectious diseases and enthesitis in spondyloarthropathies. The PD signal correlates highly with local clinical evaluation of joint inflammatory activity in the knee, metacarpophalangeal (MCP) and interphalangeal joints of patients with RA and other inflammatory arthropathies.

PSORIATIC ARTHRITIS

Dr. Sofia Paulson, Senior Resident, Govt. Medical College Thrissur

Psoriatic arthritis (PsA) is an inflammatory arthritis associated with psoriasis and is a member of spondyloarthritis family. The prevalence of inflammatory arthritis is increased among patients with psoriasis, ranging from 7% to 42%, compared with a general population estimate of 2% to 3%. Among patients with psoriatic arthritis, in 85% of them skin psoriasis precedes arthritis, whereas in 15% psoriasis develops later after the onset of arthritis and rarely never at all (psoriatic arthritis sine psoriasis). Overall, this disease is equally common in both sexes with a mean age of onset is 30 to 55 years of age. The most common skin lesions are plaque psoriasis and psoriasis vulgaris but other patterns can be seen.

CLINICAL FEATURES AND CLASSIFICATIONS :-

Psoriatic arthritis is heterogenous in nature by its musculoskeletal manifestations like axial arthritis, peripheral arthritis, enthesitis, dactylitis and extra articular manifestations affecting the skin, nails, eyes and organ systems.

In 1971 Moll and Wright classified psoriatic arthritis in to 5 clinical types:

1. Distal interphalangeal (DIP) joint arthritis
2. Arthritis mutilans
3. Symmetrical polyarthritis
4. Asymmetrical oligoarthritis
5. Spondylitis

The Classification criteria for Psoriatic Arthritis (**CASPAR**) have high specificity but less sensitivity. To be eligible for classification as PsA the patient must have inflammation of joint or spine or enthesis with more than 3 points from any of the following category:

1. Psoriasis: Current (2), history of (1), family history of (1)
2. Nail Dystrophy (1): Onycholysis, pitting, hyperkeratosis
3. Negative for rheumatoid factor (1)
4. Dactylitis: Current (1), history of (1)
5. Radiographs of hands or feet (1): Juxta-articular new bone formation.

However, the pattern of joint involvement in PsA is not fixed, it may fluctuate and overlap and can be influenced by treatment.

DIP arthritis :-

DIP arthritis is characteristic of PsA and is often associated with dactylitis and nail abnormalities. It occurs early in the disease.

Mono and oligoarticular arthritis

It is most common presenting pattern and the prevalence ranges from 11-70% and have a male preponderance.

Symmetric Polyarthritis :-

It involves the small joints of hands and feet with female preponderance. They often have longer disease duration and are associated with erosive arthritis simulating RA.

Arthritis Mutilans :-

Known as 'doigten lorgnette' or, *'opera glass finger'*. It is the most severe form of PsA. Severe destructive arthritis leading to bone resorption is the hallmark feature of arthritis mutilans. These bone resorptions often lead to joint instability with consequent flail joints. A greater degree of bone resorption can lead to digital shortening or telescoping.

Spondyloarthritis :-

Axial involvement in PsA can be of two different patterns- either an isolated spinal involvement or an overlap with peripheral arthritis. Severe peripheral arthritis and the presence of HLA B27 are risk factor for spondyloarthritis. Unilateral sacroilitis is common. It differs from AS by the lower incidence of zygapophyseal joint involvement. Atlantoaxial subluxation of cervical spine similar to RA can occur.

Dactylitis :-

Swelling of whole digit (sausaging) is called dactylitis and it is due to joint inflamation, soft tissue oedema, bone oedema and tenosynovitis.

Enthesitis :-

Enthesitis is a characteristic feature of PsA. Achilles tendonitis and plantar fascitis are the most common sites, while iliac crest, insertions of quadriceps patellar, rotator cuff tendons and epicondyles of elbows are the other common sites affected.

Extra-articular manifestations :-

Uveitis occurs in 7-18%, more B/L in patients with spinal involvement. Higher prevalence of inflammatory bowel disease has been noted in some studies.

Other Associated Features

The syndrome of synovitis, acne pustulosis, hyperostosis and osteitis (SAPHO) is associated with psoriasis vulgaris or palmoplantar pustulosis. Psoriatic onychopachydermoperiostitis is a rare manifestation of PsA. Nails changes have close correlation with arthritis. Nails changes including pitting, ridging, hyperkeratosis and onycholysis occurs in 60-80% of PsA patient . Arthritis occurs within 2 years of onset of nail changes and DIP arthritis is more commonly associated with nail changes.

Comordibidities :-

Several studies had highlighted the cardiovascular risk factors and metabolic syndrome in psoriatic arthritis.

INVESTIGATIONS:-

Laboratory :-

There are no specific laboratory markers for psoriatic arthritis. Acute phase reactants are raised and in particular ESR and CRP correlate well with inflammation. 5-16% patients have low levels of RF and in 5% ACPA can be positive. Hyperuricemia can occur and denotes renal impairment and metabolic abnormalities.

Radiography :-

Peripheral Joints :-

1. New bone formation at sites of enthesitis.

2. Erosive changes at DIP.

3. Marginal erosions with adjacent bony proliferation along shafts of metacarpals, metatarsals (whiskering).

4. Pencil in cup deformity - Resorption resulting in whittling of phalanx.

Spinal joints:-

1. Asymmetrical sacroilitis is more common
2. Syndesmophytes are thick and non marginal called flowing syndesmophytes.
3. Cervical involvement is more common.

Musculoskeletal Ultrasound:-

It detects early changes of PsA at entheseal sites, entheseal thickening, hypoechoic changes, increased vascularity in power Doppler, tenosynovitis and bony erosions.

MRI:-

MRI is not routinely done. It can detect bone marrow oedema at enthesitis and early sacroilitis.

DIAGNOSIS:-

Diagnosis is clinical. High index of suspicion in a case of inflammatory arthritis is needed to clinch the diagnosis. Detailed history of psoriasis including family history and examination for skin lesions are essential. Axial signs, enthesitis, dactylitis, and radiographic changes help in diagnosing the PsA.

MANAGEMENT:-

Management of PsA requires a holistic multidisciplinary approach consisting of non-pharmacological, rehabilitative and surgical therapies along with the help of the dermatologist. The goals of treatment of PsA include control of inflammation, preventing joint damage and functional disability and modifying the long term complications of the disease. Three important guidelines have been proposed in the last 5 years. European League Against Rheumatism (EULAR), GRAPPA (Group for Research and assessment of Psoriasis and Psoriatic Arthritis) in 2016 and American College of Rheumatology (ACR) in 2018.

GENERAL PRINCIPLES OF TREATMENT

1. Early diagnosis and treatment are the keys to improving long term outcomes.

2. Treatment decisions are based on disease severity and the clinical domains involved.

3. PsA is associated with a high prevalence of comorbidities. These should be screened and managed as appropriate.

4. Patient education is extremely important

Non- pharmacological Management:-

This includes patient education, family and employer counselling and rehabilitation.

Pharmacological treatment

The choice of treatment is PsA depends on the domains involved since the relative efficacy of drugs differs between domains.

Table 1 : Pharmacological agents in the treatment of PsA and efficacy for individual domains.

	Cutaneous	Peripheral arthritis	Enthesitis	Dactylitis	Axial	Nails
NSAIDs	X	+	+	+	+	X
Methotrexate	++	++	X	X	X	X
Leflunomide	+	++	X	X	X	X
Sulfasalazine	+/-	+	X	X	+/-	X
TNF inhibitors	+++	+++	++	++	++	++
IL 17A blockers	++++	+++	++	++	++	++
Ustekinumab	++++	+++	++	++	X	++
Apremilast	+	+	+	+/-	X	X
Tofacitinib	+	+++	+	+	X	X

NSAIDS :-

Both selective and non selective COX2 inhibitors are useful in relieving musculoskeletal symptoms.

Glucocorticoids:-

Steroids are generally avoided in psoriasis because of the risk of developing erythroderma and pustular psoriasis. If they are required, the smallest dose should be prescribed with a slow taper to prevent cutaneous complications.

Conventional synthetic disease-modifying antirheumatic drugs (csDMARDs):-

Methotrexate is often the first drug that is used in patients with peripheral arthritis. Even though there is paucity of evidence supporting the efficiency of csDMARDS. Observational data and clinical experience suggest that methotrexate is effective in PsA. EULAR guidelines recommend a trial of methotrexate as the first line for 3-6 months before biological DMARDs are considered for peripheral arthritis. Hepatotoxicity has been a concern in methotrexate treated psoriasis patients than in RA patients. Leflunomide and sulfasalazine are considered only if methotrexate is not tolerated. Leflunomide is given as 10 to 20 mg once daily dosage. Sulfasalazine is effective for peripheral arthritis at a dose of 2gm per day. Cyclosporine A is effective alone or in combination with methotrexate in severe resistant psoriatic skin lesions and arthritis.

Biological DMARDs (bDMARDs) :-

Biological DMARDs, in particular, TNF inhibitors are initiated when there is inadequate response to csDMARDs. All five TNF inhibitors are FDA approved for use in PsA and four are available, namely Etanercept, Infliximab, Adalimumab and Golimumab. They bring about a marked reduction in the signs of inflammation and delay radiological progression.

Biological DMARDs blocking IL-11/23 or IL-17:-

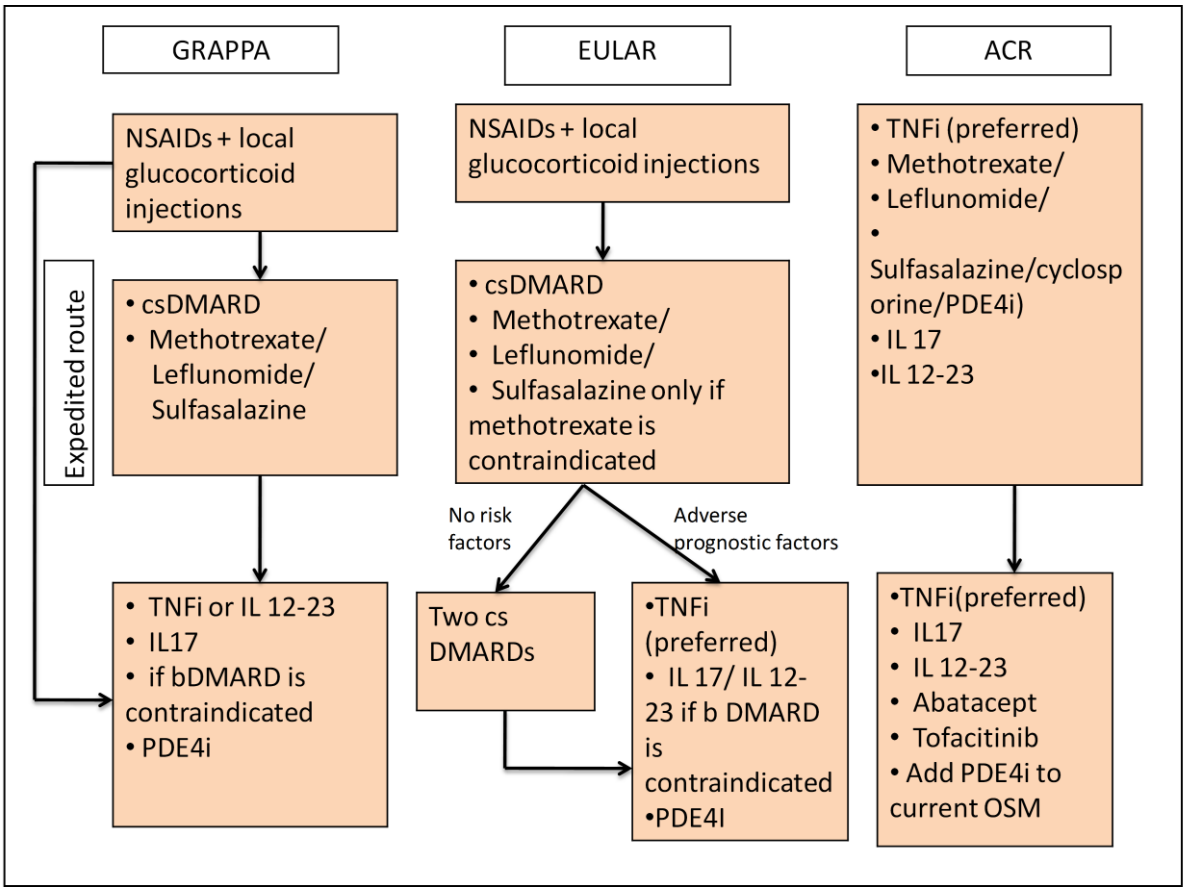
Secukinumab is an anti-IL-17 monoclonal antibody which is effective in both psoriasis and PsA. The other anti-IL-17 agents are ixekizumab and brodalumab. IL 12/23 inhibitor is a monoclonal antibody to the shared p40 subunit of IL-12 and IL-23 and has demonstrated efficacy in perpheral arthritis. bDMARDS blocking Il-12/23 or IL-17 pathways are used when there is inadequate response to csDMARDs and TNFi.

Targeted Synthetic DMARD (tsDMARD):-

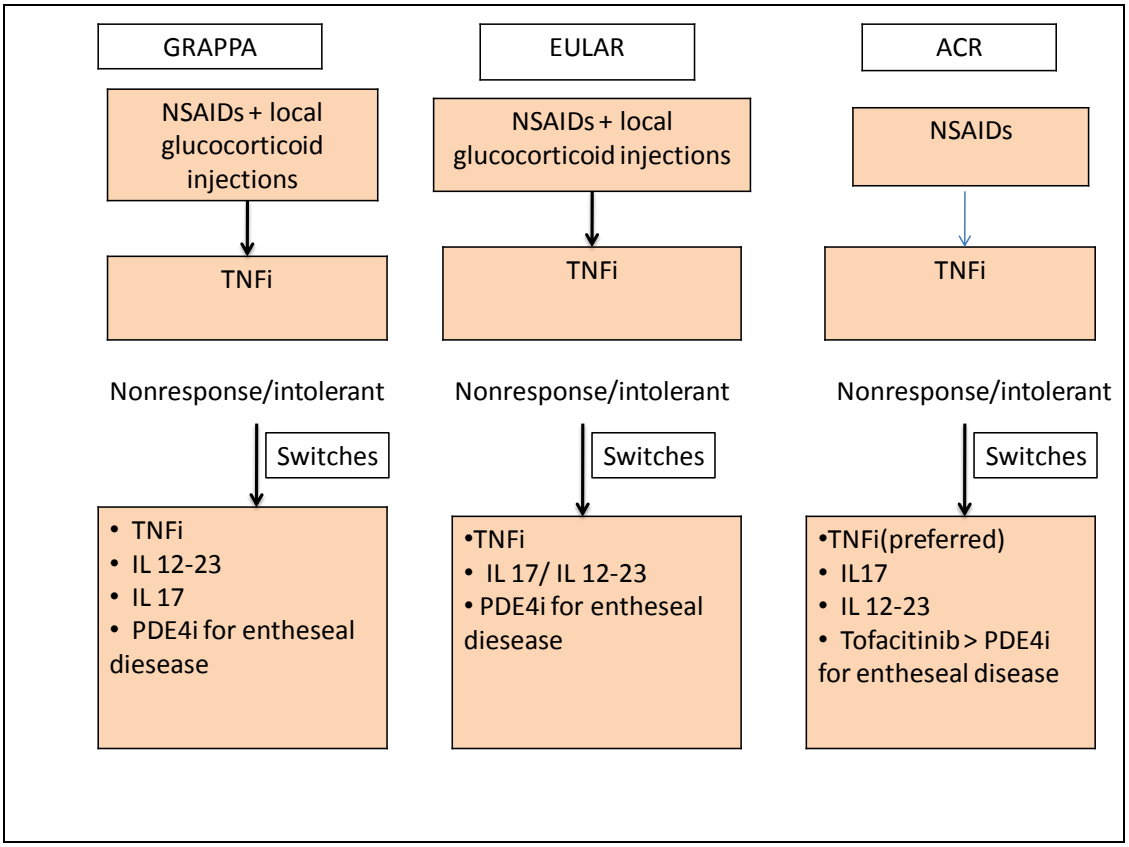
Apremilast is a novel phosphodiesterase 4 inhibitor and has an excellent safety profile. ACR recommends adding apremilast to csDMARDs rather than monotherapy. It is recommended when there are contraindications to bDMARDs.

Tofacitinib is an oral Janus kinase inhibitor effective in PsA. It is considered in ACR guidelines as an option for patients who prefer oral medication and do not have severe skin involvement.

Flowchart 1 : Comparison of EULAR/ACR/GRAPPA guidelines for management peripheral arthritis.



FLOWCHART 2 : Comparison of EULAR/ACR/GRAPPA guidelines for management axial and entheseal disease



CONCLUSION:

In short psoriatic arthritis requires early diagnosis and treatment to improve the outcome. NSAIDs and Methotrexate play a major role in the management especially in resource-limited settings. Biological agents especially TNFi are currently the most efficacious drugs for PsA and are backed by long term safety profile. Newer targeted therapies has come up which can bring us closer to realm of remission which was previously difficult to achieve.

References:-

1. Manual of Rheumatology- 5th edition.

2. Rheumatolgy clinics spondyloarthritis.

3. Firestein& Kelley's Textbook of Rheumatology-11th edition

Rheumatology Practice & COVID Vaccines: 5 Points to remember.

Dr. Santhosh Babu M R, Senior Medical Consultant (PM&R),
National Institute of Physical Medicine & Rehabilitation, Iringalakkuda, Thrissur.

1. **Can DMARDs reduce immune response to COVID vaccines?**

   Yes. Even though studies in this regard are less, available evidence shows that protective antibody response of patients on DMARDs after COVID vaccination is reduced. The effect varies with different vaccines and type of DMARDs. No research on adenovirus J &J vaccines is published so far. Sulfasalazine, Methotrexate, TNF Inhibitors, JAK inhibitors, IL Inhibitors produced modest reductions.10 fold reductions are seen in patients on regular corticosteroids while 30 fold reductions are reported with B cell inhibitors. Since all these groups developed protective antibody response, the benefit of COVID vaccination should not be deferred to these patients.

2. **Should we discontinue DMARDs prior to COVID vaccination?**

   Not necessary. Whether deferring vaccines will lead to improved protective antibody response remains unknown now. However it is prudent to consider avoiding high dose corticosteroids & Biologics during vaccination period. These can be given if essential at least 2weeks prior to first dose and three weeks after second dose in cases resistant to conventional DMARDs.. More over biologics should be started during the Pandemic if only absolutely essential and instead conventional DMARDs should be preferred.

3. **Will vaccines flare up of autoimmune rheumatic diseases?**

   There are few reports of relapse of moderate disease activity of RA after COVID vaccination, even after prolonged remission. But this is more likely in people with very minimum or no drugs. The American College of Rheumatology issued guidance regarding COVID-19 vaccination on Feb 8, 2021, and acknowledged a theoretical risk of flare of autoimmune diseases after vaccination with moderate consensus.

4. **Are COVID vaccines safe in people having autoimmune rheumatic diseases?**

   So far there are no reports to believe that the available vaccines are unsafe in people with autoimmune rheumatic diseases. COVID vaccines made by mRNA subunit technology are believed to be safer in immune-compromised patients to those on immune-suppressant drugs. One should note that in vaccine trials both these groups were excluded.

5. **Should we discontinue any rehabilitation programs during vaccination period?**

   Not necessarily. No authoritative recommendations are available in this regard. In view of possibilities for mild adverse effects during immediate post vaccination period, avoiding physical exertion and therapeutic heat modalities three to four days immediately after each vaccination dose is a better strategy.

Fibromyalgia, Pain for a lifetime, Facts and Fallacies!

Dr. Vipin P Vijayan, MD
(PMR) Junior Consultant
Department of Health Service – Kerala

Working as a Pain-Rehabilitation Physician, I have seen many patients who come with total body pain, aches, muscle cramps, sleeplessness and fatigue. Most of them have consulted Doctors of almost all Specialities available in their area and had undergone various investigations and eventually all reports were normal.

While taking the history, they reveal that, all these difficulties were there very early in their life, but were categorized under the broad term, laziness. Because of the excessive fatigability, most of them were scared to work continuously.

It may be due to Fibromyalgia, a neurologic chronic health condition that causes pain all over the body and other symptoms, but all other conditions with similar presentation have to be ruled out. Widespread pain, abnormal pain sensitivity, and additional symptoms, such as fatigue, sleep disturbance, and mood symptoms. Exact aetiology and pathogenesis of Fibromyalgia are still unknown, it has been suggested that stress or psychological factors may play a key role in the syndrome.

It is believed that the disease now called Fibromyalgia first began to be recognized and studied in the 16th century. The first description of such a condition was provided by Guillaume de Baillou in 1642, who used the term ―muscular rheumatism. E.W. Boland (1947), in the absence of physical findings corresponding to the symptomatology, modified the concept and proposed the term ―psychogenic rheumatism, defined as musculoskeletal expression of functional disorders, stress states, or psychoneurosis. In 1976, P.K. Hench coined the term fibromyalgia (from the Latin word ―fibro, which means fibrous tissue, and the Greek words ―mio (muscle) and ―algia (pain) as a form of non-articular rheumatism. However, it was not until 1981 that the medical community started accepting the disease under the term ―fibrositis or ―fibromyalgia. In fact, in 1981, Yunus et al. introduced the following formal set of criteria to diagnose primary FMS:

(1) **Obligatory Criteria**: (A) Presence of aching, pain, or stiffness in three anatomical areas for at least 3 months. (B) Absence of causes to explain the condition,

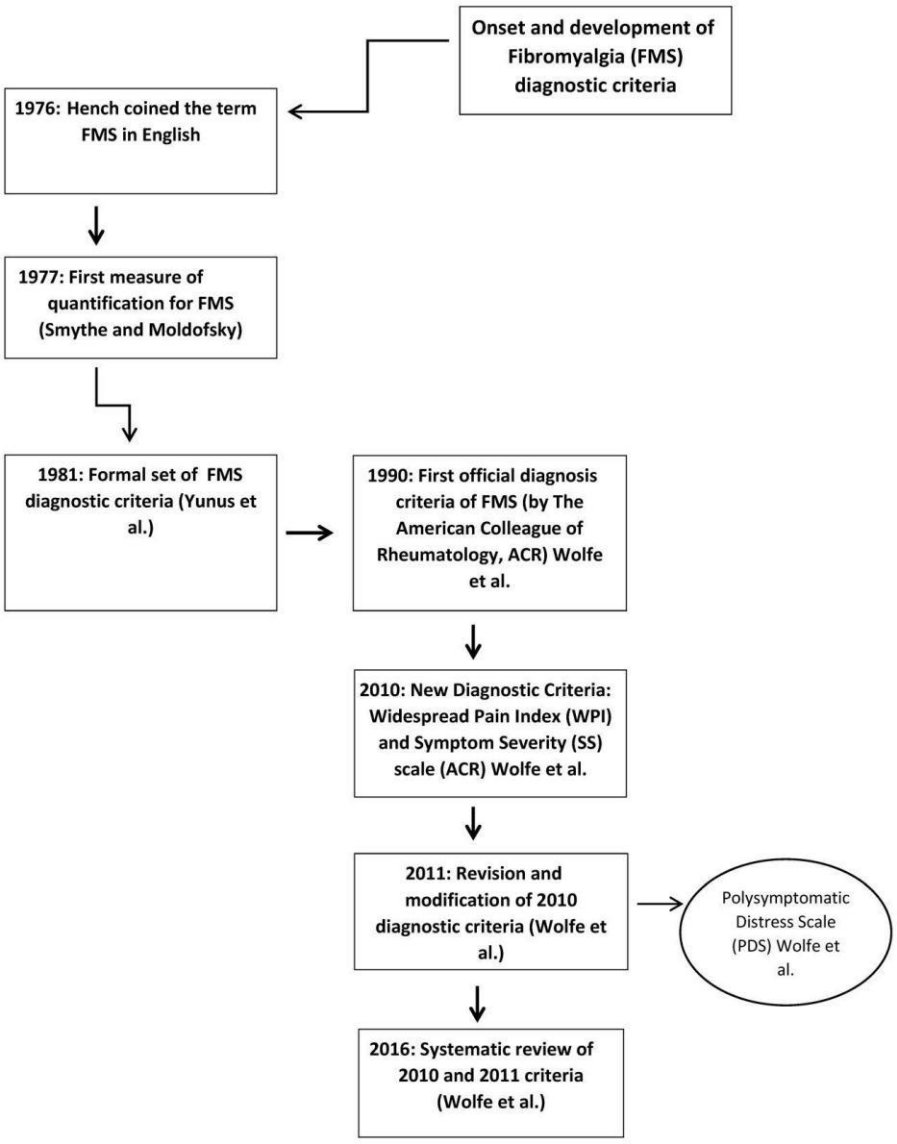
(2) **Major Criteria**: presence of at least five typical and consistent tender points.

(3) **Minor Criteria**: (A) modulation of symptoms by physical activity (aggravated due to physical inactivity and relieved with moderate physical activity), (B) modulation of symptoms by weather (i.e., worsening of symptoms due to cold, humid weather, and relief of them by heat) or time factors (i.e., worsening of symptoms in the morning and the evening), (C) aggravation of symptoms by anxiety or stress, (D) poor sleep, (E) general fatigue or tiredness, (F) anxiety, (G) chronic headache, (H) irritable bowel syndrome, (I) subjective

swelling, and (J) numbness (symptoms can be also relieved by reduction of stress and vacations).

All primary FMS patients must satisfy the two obligatory criteria, as well as the major criterion plus at least three minor criteria. If the patient has only 3 or 4 tender points, then five minor criteria must be met. For cases wherein a secondary condition (e.g., inflammation) produces the syndrome, the term secondary fibrositis was proposed.

In 1987, the American Medical Association accepted Fibromyalgia, as a disease. As a result of this recognition, the American College of Rheumatology (ACR) created a committee to establish the diagnostic criteria for Fibromyalgia. In spite of their limitations, the 1990 ACR criteria brought official recognition to Fibromyalgia. In 1992, Fibromyalgia was included in the International Classification of Diseases (ICD), received international recognition as a source of disability and funding from research and government institutions, and gained academic recognition. In 1992, the World Health Organization (WHO) also recognized Fibromyalgia, as a disease, and it was classified as a non-joint type of rheumatism under the code M.79.7 of the ICD. Therefore, the 1990 ACR diagnostic criteria helped legitimize the syndrome.



In 2010, the ACR proposed a new version of the diagnostic criteria based exclusively on the use of two scales: The Widespread Pain Index (WPI) and the Symptom Severity (SS) Scale. The 2010 ACR criteria have been validated in Iranian, Japanese, French, Turkish, and Spanish populations. The 2010 ACR criteria have also been criticized due to the absence of a tender point count. Whereas Wolfe et al. showed that SS scores correlate better with WPI scores (0.73) than with tender point count.

Criteria Needed for a Fibromyalgia Diagnosis

1. Pain and symptoms over the past week, based on the total of number of painful areas out of 19 parts of the body.
2. Level of severity of these symptoms:
   a. Fatigue
   b. Waking unrefreshed
   c. Cognitive (memory or thought) problems, plus number of other general physical symptoms
3. Symptoms lasting at least three months at a similar level
4. No other health problem that would explain the pain and other symptoms.

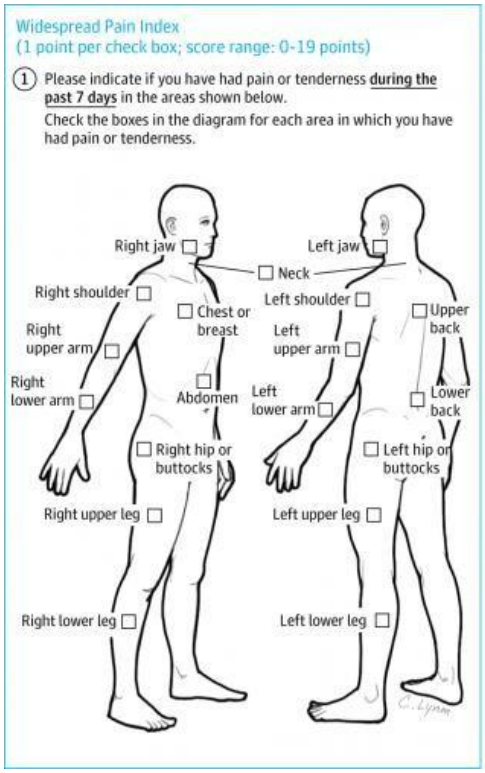
A patient meets the diagnostic criteria for Fibromyalgia if the following 3 conditions are met:

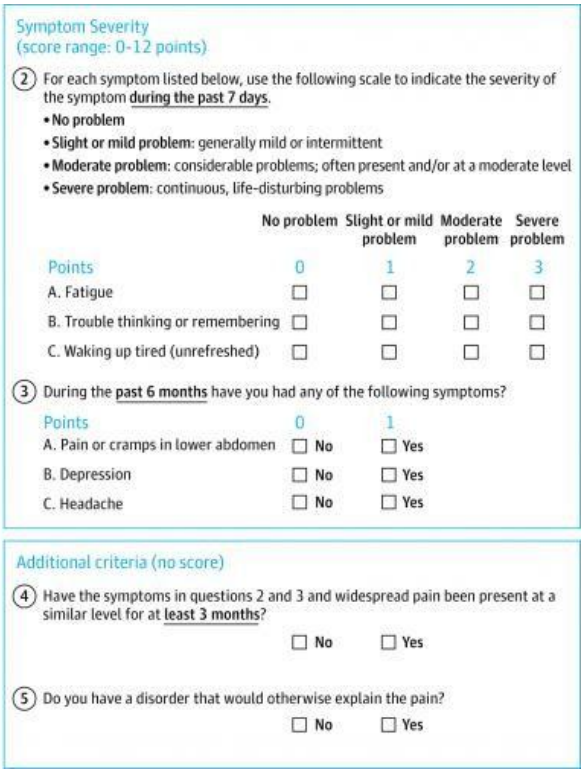
1a. The WPI score (Part 1) is greater than or equal to 7 and the SS score (Parts 2a and 2b) is greater than or equal to 5.

OR

1b. The WPI score (Part 1) is from 3 to 6 and the SS score (Parts 2a and 2b) is greater than or equal to 9.

2. Symptoms have been present at a similar level for at least 3 months.
3. The patient does not have a disorder that would otherwise explain the pain.





2016 Revised Criteria:

A patient satisfies modified 2016 fibromyalgia criteria if the following 3 conditions are met:

(1) Widespread pain index (WPI) ≥ 7 and symptom severity scale (SSS) score ≥ 5 OR WPI of 4–6 and SSS score ≥ 9.

(2) Generalized pain, defined as pain in at least 4 of 5 regions, must be present. Jaw, chest, and abdominal pain are not included in generalized pain definition.

(3) (3) Symptoms have been generally present for at least 3 months.

(4) A diagnosis of fibromyalgia is valid irrespective of other diagnoses. A diagnosis of fibromyalgia does not exclude the presence of other clinically important illnesses.

The Analgesic, Anaesthetic, and Addiction Clinical Trial Translations Innovations Opportunities and Networks (ACTTION) public-private partnership with the U.S. Food and Drug Administration (FDA) and the American Pain Society (APS) initiated the ACTTION-APS Pain Taxonomy (AAPT) to develop a diagnostic system that would be clinically useful and consistent across chronic pain disorders. Future studies will assess the criteria for feasibility, reliability, and validity.

Clinical Presentation:

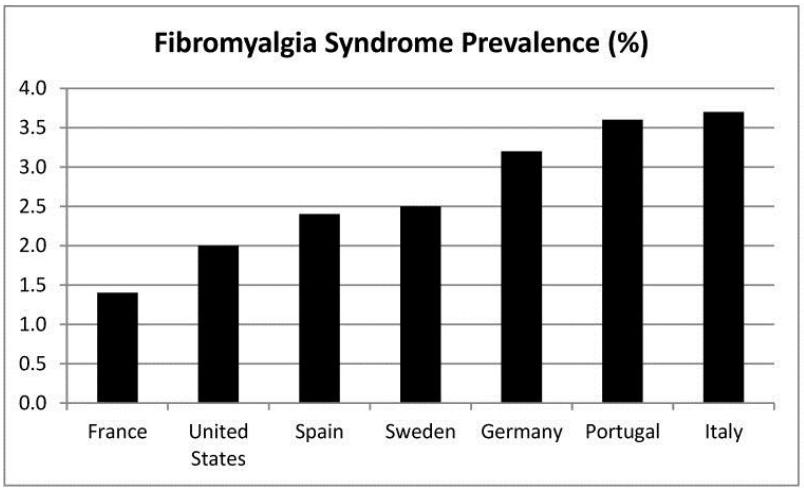


Figure: Clin. Med. 2020, 9, 1219; doi:10.3390/jcm9041219.

Pain, especially Generalised Body Pain is the major symptom, which bring them to Doctors. Some of them have following symptoms as well-

- Tenderness to touch or pressure affecting muscles and sometimes joints or even the skin
- Severe fatigue
- Sleep problems (waking up unrefreshed)
- Problems with memory or thinking clearly
- Depression or anxiety
- Migraine or tension headaches
- Digestive problems: irritable bowel syndrome or gastroesophageal reflux disease.
- Irritable or overactive bladder

- Pelvic pain
- Temporo-mandibular disorder - often called TMJ (a set of symptoms including face or jaw pain, jaw clicking, and ringing in the ears).

Fibromyalgia is a diagnosis of exclusion and patients must be thoroughly evaluated for the presence of other disorders that could be the cause of symptoms before a diagnosis of fibromyalgia is made. The clinical assessment may reveal objective evidence for a discrete or comorbid illness, such as the following:

- Hypothyroidism
- Rheumatoid arthritis
- Systemic lupus erythematous
- Polymyalgia rheumatica
- Other inflammatory/autoimmune disorders
- Serious cardiac conditions in those with chest pain, dyspnoea, and palpitations.

Pathophysiology:

Fibromyalgia is currently understood to be a disorder of central pain processing or a syndrome of central sensitivity. Research has provided evidence for altered functional connectivity and chemistry in the pain-processing system of the brain. Clauw, describes the syndrome as a diffuse problem of sensory –volume control such that patients have a lower threshold of pain and of other stimuli, such as heat, noise, and strong odours. Clauw also suggests that patients may have hypersensitivity because of neurobiologic changes that affect the perception of pain or because of expectancy or hyper vigilance, which may be related to psychological factors.

A number of abnormalities in pain processing have been demonstrated in fibromyalgia. Among them are the following:

- Excess excitatory (pro-nociceptive) neurotransmitters (eg, substance P, glutamate levels in the insula)
- Low levels of inhibitory neurotransmitters (eg, serotonin and norepinephrine) in descending anti-nociceptive pathways in the spinal cord
- Maintained enhancement of temporal summation of second pain
- Altered endogenous opioid analgesic activity in several brain regions known to play a role in pain modulation
- Dopamine dysregulation

Treatment:-

There is no cure for fibromyalgia, this is the first comment you will get when you review the literature. This statement itself will make the patient anxious. However, symptoms can be treated with both non-drug and medication based treatments. Many times the best outcomes are achieved by using multiple types of treatments.

Non-Drug Therapies: People with fibromyalgia should use non-drug treatments. Research shows that the most effective treatment for fibromyalgia is physical exercise. Physical exercise should be used in addition to any drug treatment. Patients benefit most from regular

aerobic exercises. Other body-based therapies, including Tai Chi and yoga, can ease fibromyalgia symptoms. Although you may be in pain, low impact physical exercise will not be harmful.

Cognitive behavioural therapy (CBT) is a type of therapy focused on understanding how thoughts and behaviours affect pain and other symptoms. CBT and related treatments, such as mindfulness, can help patients learn symptom reduction skills that lessen pain. Mindfulness is a non-spiritual meditation practice that cultivates present moment awareness. Mindfulness based stress reduction has been shown to significantly improve symptoms of fibromyalgia.

Other complementary and alternative therapies (sometimes called CAM or integrative medicine), such as acupuncture, chiropractic and massage therapy (Myofascial Release), can be useful to manage fibromyalgia symptoms.

It is important to address risk factors and triggers for fibromyalgia including sleep disorders, such as sleep apnea, and mood problems such as stress, anxiety, panic disorder, and depression. This may require involvement of other specialists such as a Sleep Medicine Doctor, Psychiatrist, and Physiotherapist.

Medications: The U.S. Food and Drug Administration (FDA) has approved three drugs for the treatment of fibromyalgia. They include two drugs that change some of the brain chemicals (serotonin and norepinephrine) that help control pain levels: Duloxetine and Milnacipran. Older drugs that affect these same brain chemicals also may be used to treat fibromyalgia. These include Amitriptyline and Cyclobenzaprine. Other antidepressant drugs can be helpful in some patients.

The other drug approved for fibromyalgia is Pregabalin. Pregabalin and another drug, Gabapentin, work by blocking the over activity of nerve cells involved in pain transmission. These medicines may cause dizziness, sleepiness, swelling and weight gain.

It is strongly recommended to avoid opioid narcotic medications for treating fibromyalgia. The reason for this is that research evidence shows these drugs are not of help to most people with fibromyalgia, and will cause greater pain sensitivity or make pain persist. Tramadol, may be used to treat fibromyalgia pain if short-term use of an opioid narcotic is needed. Over-the-counter medicines such as Acetaminophen or Nonsteroidal anti- inflammatory drugs, like Ibuprofen or Naproxen are not effective for fibromyalgia pain. Yet, these drugs may be useful to treat the pain triggers of fibromyalgia. Thus, they are most useful in people who have other causes for pain such as arthritis in addition to fibromyalgia.

For sleep problems, some of the medicines that treat pain also improve sleep. These include Cyclobenzaprine, Amitriptyline, Gabapentin or Pregabalin. It is not recommended that patients with fibromyalgia take sleeping medicines like Zolpidem or Benzodiazepine medications.

Living with fibromyalgia:

Even with the many treatment options, patient self-care is vital to improving symptoms and daily function. Along with medical treatment, healthy lifestyle behaviours and Postural Correction can reduce pain, increase sleep quality, lessen fatigue and help you cope better with fibromyalgia. With proper treatment and self-care, you can get better and live a more normal life.

- Make time to relax each day. Deep-breathing exercises and meditation will help reduce the stress that can bring on symptoms.

- Set a regular sleep pattern. Go to bed and wake up at the same time each day. Getting enough sleep lets your body repair itself, physically and mentally. Also, avoid daytime napping and limit caffeine intake, which can disrupt sleep. Nicotine is a stimulant, so those fibromyalgia patients with sleep problems should stop smoking.
- Exercise often. This is a very important part of fibromyalgia treatment. While difficult at first, regular exercise often reduces pain symptoms and fatigue. Patients should follow the saying, –Start low, go slow. Slowly add daily fitness into your routine. For instance, take the stairs instead of the elevator, or park further away from the store. As your symptoms decrease with drug treatments, start increasing your activity. Add in some walking, swimming, water aerobics and/or stretching exercises, and begin to do things that you stopped doing because of your pain and other symptoms. It takes time to create a comfortable routine. Just get moving, stay active and don't give up!
- Educate yourself. Nationally recognized organizations like the Arthritis Foundation and the National Fibromyalgia Association (of the USA) are great resources for information.
- Look forward, not backward. Focus on what you need to do to get better, not what caused your illness.

References:

1. Medline
2. Arthritis Care & Research;Vol. 62, No. 5, May 2010, pp 600–610;DOI;10.1002/acr.20140
3. Am Fam Physician. 1995; 52(3):843-51, 853-4
4. J. Clin. Med. 2020, 9, 1219; doi:10.3390/jcm9041219.
5. Biomedicines. 2017; 5(2) (ISSN: 2227-9059).
6. Baillou G. Liber de Rheumatismo et Pleuritide Dorsali. Thevart MJ; Paris, France: 1642.
7. Psychogenic rheumatism; the musculoskeletal expression of psychoneurosis. BOLAND EW. Ann Rheum Dis. 1947 Dec; 6(4):195-203.
8. Hench P.K. Nonarticular rheumatism, 22nd rheumatism review: Review of the American and English literature for the years 1973 and 1974. Arthritis Rheumatol. 1976;19:1081–1089.
9. Yunus M., Masi A.T., Calabro J.J., Miller K.A., Feigenbaum S.L. Primary fibromyalgia (fibrositis): Clinical study of 50 patients with matched normal controls. Semin. Arthritis Rheum. 1981;11:151–171.
10. Wolfe F, Häuser W. Ann Med. 2011 Nov; 43(7):495-502.
11. Wolfe F, Smythe HA, Yunus MB, Bennett RM, Bombardier C, Goldenberg DL, Tugwell P, Campbell SM, Abeles M, Clark P. Arthritis Rheum. 1990 Feb; 33(2):160-72.

Hemophilic Arthropathy: A rehab perspective…..

Dr. Shigy Francis, Consultant, Lissie hospital, Ernakulam

Hemophilia is a rare X-linked congenital bleeding disorder characterized by a deficiency of coagulation factor VIII (FVIII), called Hemophilia A, or factor IX (FIX), called Hemophilia B. The factor deficiencies are the result of pathogenic variants in the F8 and F9 clotting factor genes. The best estimates of the prevalence of Hemophilia, based on the most reliable national patient registry data available and recent World Federation of Hemophilia (WFH) annual global surveys, indicate that the expected number of males with Hemophilia worldwide is 1,125,000, the majority of whom are undiagnosed, including an estimated 418,000 males with severe hemophilia.[1]

Hemophilic arthropathy can result from a single bleed or recurrent bleeds. It generally evolves gradually from hemarthrosis to chronic synovitis and extended erosions of the articular surface, culminating in the final stage of joint destruction, chronic Hemophilic arthropathy[2], which often manifests during the second decade of life, particularly if prophylactic therapy is unavailable or inadequate.

Joint bleed most commonly occurs in the ankle, knee, and elbow joints, and frequently the hip, shoulder, and wrist joints and in particular muscles (iliopsoas and gastrocnemius)[3,4]. Spontaneous bleeding may occur depending on the severity of the disease or breakthrough bleeding may occur depending on the prophylactic treatment approach. Recurrent joint bleeds cause progressive joint damage as a result of blood accumulation in the joint cavity and synovial inflammation. This leads to complications such as chronic synovitis and Hemophilic arthropathy.

Inadequate treatment of intramuscular bleeds can lead to muscle contractures, especially in bi-articular muscles (e.g., gastrocnemius and psoas muscles), often within the first decades of life[3,4]. Other more serious complications such as compartment syndrome and pseudotumours may also develop.

Chronic Synovitis:

Following acute hemarthrosis, the synovium becomes inflamed, hyperemic, and friable. This acute synovitis can take several weeks to resolve. Failure to manage acute synovitis results in recurrent hemarthroses and subclinical bleeds[3,4]; the synovium becomes chronically inflamed and hypertrophic, and the joint becomes prone to further bleeding. A vicious cycle of bleeding, loss of joint motion, and inflammation can ensue which ultimately leads to irreversible cartilage[5,6] and bone damage and impaired joint function. If this process exceeds 3 months, it is defined as chronic synovitis.

Physical examination includes joint circumference, muscle strength, joint effusion, joint angle, pain according to a visual analogue scale and should be conducted at all routine follow-ups. However, in many cases, the synovium never returns to its original state.

Even though ultrasound evaluation is advised Magnetic resonance imaging (MRI), is currently the gold standard for Chronic Synovitis.

Treatment of chronic synovitis

The goal of chronic synovitis treatment is to suppress synovial activation and to reduce inflammation to preserve joint integrity and function[7,8].

Nonoperative options:

- Prophylaxis for 6-8 weeks (for those not on regular prophylaxis) to control bleeding
- Physiotherapy to improve muscle strength and joint function
- Selective Cox-2 inhibitors to reduce pain and inflammation

Physiotherapy with individualized goals and exercises based on the patient's functional level should start slowly with increasing progression of weight-bearing activities.

Bracing may be appropriate to stabilize the affected joint and limit movement in order to prevent recurrent bleeding and synovial impingement during movement. Isometric exercise even within bracing is advised to prevent muscle weakness due to immobilisation.

Synovectomy/ synoviorthesis

Synovectomy should be considered if chronic synovitis persists with frequent recurrent bleeding not controlled by other means[9,10].

1. Chemical or radioisotope intra-articular injection (synoviorthesis)
2. Arthroscopic synovectomy
3. Open surgical synovectomy

Nonsurgical synovectomy should always be the first procedure of choice for all patients. Radiosynovectomy is indicated for synovitis (confirmed clinically or by point-of-care ultrasound) causing 2 or more bleeds in a particular joint over the last 6 months despite adequate treatment. Radioisotope synovectomy using a pure beta emitter (phosphorus-32, yttrium-90, rhenium-186, or rhenium-188) is highly effective, has few side effects, and can be accomplished in a single outpatient procedure.[11] Choice and dose of radioisotope depend on the joint to be injected, the condition of its synovium, and available radioisotopes. Prophylaxis should be administered prior to radiosynovectomy; one dose of CFC is usually sufficient for a single injection of the radioisotope. Where possible, simultaneous administration of intra-articular steroids is recommended[12]. The joint should then be rested for at least 24-48 hours in a splint or other immobilization device, after which rehabilitation can commence. Rehabilitation after radiosynovectomy is less intensive than after surgical synovectomy, but it is still required to help patients regain strength, proprioception, and functional use of the joint. An individualized rehabilitation program for at least 3 weeks may be appropriate. Intensive exercise and weight bearing should be avoided immediately following radiosynovectomy.

If radioisotopes are not available, chemical synoviorthesis with either Rifampicin or Oxytetracycline Chlorhydrate may be considered. Chemical synoviorthesis may be painful, and the sclerosant injection should be combined with an intra-articular local anesthetic to minimize pain, supplemented by oral analgesics (a combination of Acetaminophen/Paracetamol and an Opioid) as required.)

Surgical synovectomy

Arthroscopic synovectomy is suggested over open synovectomy[13,14]. If surgical synovectomy (either open or arthroscopic) is necessary, ensure longer prophylaxis coverage with CFCs or other appropriate haemostatic agents sufficient for the procedure and postoperative rehabilitation.

Hemophilic arthropathy

Hemophilic arthropathy can result from a single bleed or recurrent bleeds. It generally evolves gradually from hemarthrosis to chronic synovitis and extended erosions of the articular surface, culminating in the final stage of joint destruction, chronic hemophilic arthropathy[15], which often manifests during the second decade of life, particularly if prophylactic therapy is unavailable or inadequate.

As the arthropathy worsens, range of motion and swelling of the joint often subside due to progressive fibrosis of the synovium and capsule. As the joint becomes ankylosed (stiffened), pain may diminish or disappear.

The appropriate radiographic technique for assessing chronic hemophilic arthropathy

depends on the stage of progression. MRI is useful to assess early arthropathy and will show early soft tissue and osteochondral changes[16]. Ultrasound imaging is useful for assessing soft tissue and peripheral cartilage pathology in early hemophilic arthropathy. Plain radiographs are insensitive to early change and are used to assess late arthropathic changes[17,18].

Treatment of chronic hemophilic arthropathy

The goals of treatment are to reduce the incidence of hemarthroses, improve joint function, relieve pain, and help the patient continue or resume normal activities of daily living. Treatment options for chronic hemophilic arthropathy depend on many factors including:

- The stage of the condition
- The patient‘s symptoms
- The patient‘s age
- The impacts on the patient‘s lifestyle and functional abilities
- The resources available

Physiotherapy aimed at preserving muscle strength and functional ability is an essential component of treatment of chronic hemophilic arthropathy. The intensity of therapy should be increased gradually and adapted according to prophylaxis coverage.

Intermittent prophylaxis coverage may be necessary if breakthrough bleeding occurs during therapy.

Different rehabilitation methods like exercise therapy, manual therapy, electrotherapy, and hydrotherapy have been used to complement physical therapy. Ice is used before and after therapy for pain relief and bleeding.

For the prevention and treatment of chronic hemophilic arthropathy, the World Federation of Hemophilia recommends a combination of regular replacement therapy to reduce frequency of bleeding and exercise aimed at preserving muscle strength and functional ability.

Other conservative management techniques include:

- Serial casting to correct deformities
- Traction devices
- Bracing and orthotics to support painful and unstable joints
- Walking aids or mobility aids to decrease stress on weight-bearing joints
- Adaptations to the home, school, or work environment to allow participation in community activities and employment, and to facilitate activities of daily living.

Surgical interventions

If nonsurgical measures fail to provide satisfactory pain relief and improved function, surgical intervention may be necessary.

Surgical procedures, depending on the specific condition, may include:

- Synovectomy and joint debridement[19]
- Arthroscopy to release intra-articular adhesions and correct impingement, especially in the ankle or elbow joint[20]
- Extra-articular soft tissue release to treat contractures
- Osteotomy to correct angular deformity
- External fixators to assist in deformity correction
- Prosthetic joint replacement (knee, hip, shoulder, elbow, or ankle) [21]
- Radial head excision for select patients with radiocapitellar arthropathy
- Arthrodesis for painful ankle joint arthropathy

Adequate resources, including prophylaxis (e.g., sufficient supply of CFCs) and postoperative rehabilitation, must be available to support and increase the likelihood of success of any surgical procedure.

Physiatrist plays an important role in educating people with hemophilia and their caregivers on preventive measures, facilitating complete functional recovery after each bleed, and counselling individuals about preserving musculoskeletal health. The prevention and treatment of musculoskeletal

complications in people with hemophilia are important to their health, autonomy, and quality of life. Rehabilitation is important for functional improvement and recovery after musculoskeletal bleeds and for those with established hemophilic arthropathy.

References:

1. Iorio A, Stonebraker JS, Chambost H, et al. Establishing the prevalence and prevalence at birth of hemophilia in males: a meta-analytic approach using national registries. Ann Intern Med. 2019; 171 (8): 540 - 546.
2. Poenaru DV, Patrascu JM, Andor BC, Popa I. Orthopaedic and surgical features in the management of patients with haemophilia. Eur J Orthop Surg Traumatol. 2014; 24 (5): 685 – 692
3. Llinas A. Haemophilic arthropathy. Haemophilia. 2010; 16 (Suppl 5): 121.
4. Rodriguez-Merchan EC. Musculoskeletal complications of hemophilia. HSS J. 2010; 6 (1): 37 - 42.
5. Rodriguez-Merchan EC. Pathogenesis, early diagnosis, and prophylaxis for chronic hemophilic synovitis. Clin Orthop Relat Res. 1997; 343: 6 - 11.
6. Jansen NW, Roosendaal G, Lafeber FP. Understanding haemophilic arthropathy: an exploration of current open issues. Br J Haematol. 2008; 143 (5): 632 – 640
7. Rodriguez-Merchan EC. Aspects of current management: orthopaedic surgery in haemophilia. Haemophilia. 2012; 18 (1): 8 - 16.
8. Seuser A, Berdel P, Oldenburg J. Rehabilitation of synovitis in patients with haemophilia. Haemophilia. 2007; 13 (Suppl 3): 26 - 31.
9. Llinas A. The role of synovectomy in the management of a target joint. Haemophilia. 2008; 14 (Suppl 3): 177 - 180.
10. Yoon KH, Bae DK, Kim HS, Song SJ. Arthroscopic synovectomy in haemophilic arthropathy of the knee. Int Orthop. 2005; 29 (5): 296 - 300.
11. Van Kasteren ME, Novakova IR, Boerbooms AM, Lemmens JA. Long term follow up of radiosynovectomy with yttrium- 90 silicate in haemophilic haemarthrosis. Ann Rheum Dis. 1993; 52 (7): 548 - 550.
12. European Association of Nuclear Medicine. EANM Procedure Guidelines for Radiosynovectomy. European Association of Nuclear Medicine; 2002. http://www.eanm.org/publi catio ns/guide lines/ gl_radio_synov ectomy.pdf. Accessed May 3, 2020.
13. Silva M, Luck JV Jr. Radial head excision and synovectomy in patients with hemophilia: surgical technique. J Bone Joint Surg Am. 2008; 90 (Suppl 2 Pt 2): 254 - 261.
14. Verma N, Valentino LA, Chawla A. Arthroscopic synovectomy in haemophilia: indications, technique and results. Haemophilia. 2007; 13 (Suppl 3): 38 – 44
15. Poenaru DV, Patrascu JM, Andor BC, Popa I. Orthopaedic and surgical features in the management of patients with haemophilia. Eur J Orthop Surg Traumatol. 2014; 24 (5): 685 - 692.
16. Doria AS, Lundin B, Miller S, et al. Reliability and construct validity of the compatible MRI scoring system for evaluation of elbows in haemophilic children. Haemophilia. 2008; 14 (2): 303 - 314.
17. Arnold WD, Hilgartner MW. Hemophilic arthropathy: current concepts of pathogenesis and management. J Bone Joint Surg Am. 1977; 59 (3): 287 - 305.
18. Pettersson H, Ahlberg A, Nilsson IM. A radiologic classification of hemophilic arthropathy. Clin Orthop Relat Res. 1980; 149: 153 – 159
19. Silva M, Luck JV Jr. Radial head excision and synovectomy in patients with hemophilia: surgical technique. J Bone Joint Surg Am. 2008; 90 (Suppl 2 Pt 2): 254 - 261.
20. Wiedel JD. Arthroscopic synovectomy: state of the art. Haemophilia. 2002; 8 (3): 372 - 374.
21. Goddard NJ, Mann HA, Lee CA. Total knee replacement in patients with end-stage haemophilic arthropathy: 25- year results. J Bone Joint Surg Br. 2010; 92 (8): 1085 – 1089.

Yttrium Synovectomy for chronic synovitis in hemophilia

Dr.Nittu Devassy Panjikaran,
Assistant Professor, Department of PMR,
Amrita Institute of Medical Sciences

Hemarthrosis is a common complication in patients with Hemophilia. Recurrent hemarthrosis leads to synovial hypertrophy and proliferation due to the hemosiderin released into the joint by the lysed RBCs. Hemarthropathy changes start as early as by the age of 2 years in severe hemophilia and if not treated will develop into hemophilic arthropathy in the second decade of life.[1,2,3] The patients who underwent synovectomy have an improved quality of life in their physical health, family and social life [4]

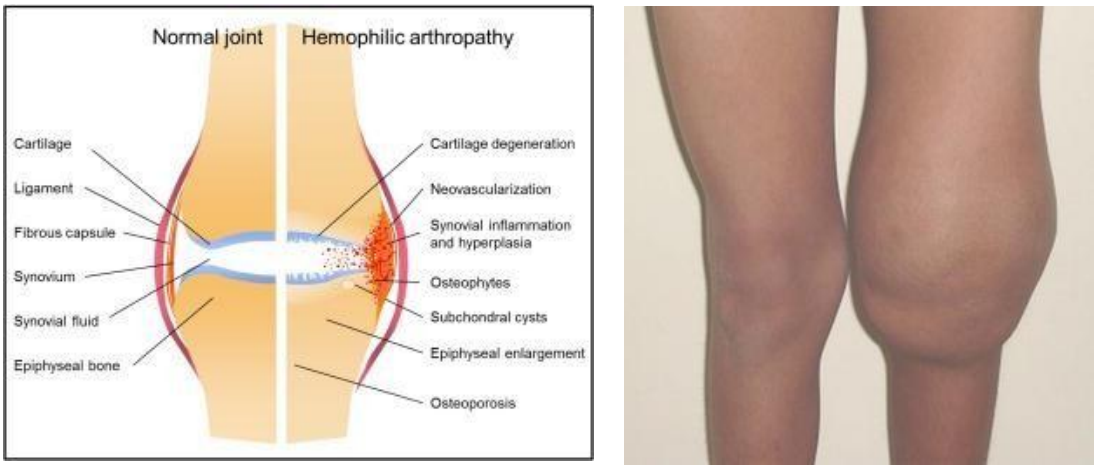


Fig 1: Chronic synovitis in hemophillia

Surgical synovectomy is a radical option to delay progression to hemophilic arthropathy but it cannot be repeated. Radioactive synovectomy or Radiosynoviorthesis in comparison is a day care procedure which can be repeated every 6 months. It is a well established treatment for chronic synovitis in patients with hemophilia [5, 6]. It is less invasive, requires minimal factor coverage, fewer infections and minimal pain and does not require as extensive rehabilitation as the prior[7].

Radiosynoviorthesis reduces the bleed rate in a joint and the articular pain by ablating the hypertrophied synovium. The most commonly used radio-isotopes are Yttrium -90, Rhenium-186 and Phosphorous-32(P-32). Being beta emitters, patients don‘t need to be isolated post-procedure, unlike those receiving gamma radiation.

Yttrium 90 has a penetration depth of up to 11 mm with the majority of radiation delivered at 2 mm distance. Being a comparatively larger molecule (particle size is 0.1 µm) chance of lymphatic spread is negligible which reduces the risk of radiation to the lymph nodes[8]. Yttrium silicate has the least leakage compared to yttrium ferric hydroxide and yttrium citrate[9]. This makes it best for large joints like the knee. Rhenium-186 on the other hand has a soft tissue range of 1.1mm and hence useful for smaller joints as elbow and ankle. Both generate free radicals resulting in apoptosis and finally ablation of the hypertrophied synovium. The advantage is that they don‘t damage the articular surface[10,11].

Once the drug arrives and is removed from its travel container, it starts degrading. The half life of yttrium-90 lasts only for 64 hours and the procedure is done as soon as possible not later than 2.7 days[12]. Half an hour prior to the procedure the deficient factor has to be replaced appropriately. An adsorbent sheet is used under the patient's joint as a precautionary measure. The drug is brought in a lead container and has a glass guard as a radioactive shielded receptacle. The procedure is done under ultrasound guidance especially in the case of small joints like ankle and elbow. The affected joint is aspirated pre-injection to avoid dilution of the drug and the yttrium will be injected followed by steroid[1]. The joint will be immobilized for the first 48 hours so that the drug remains in the joint till the radioactivity fully decays [13, 14,15].

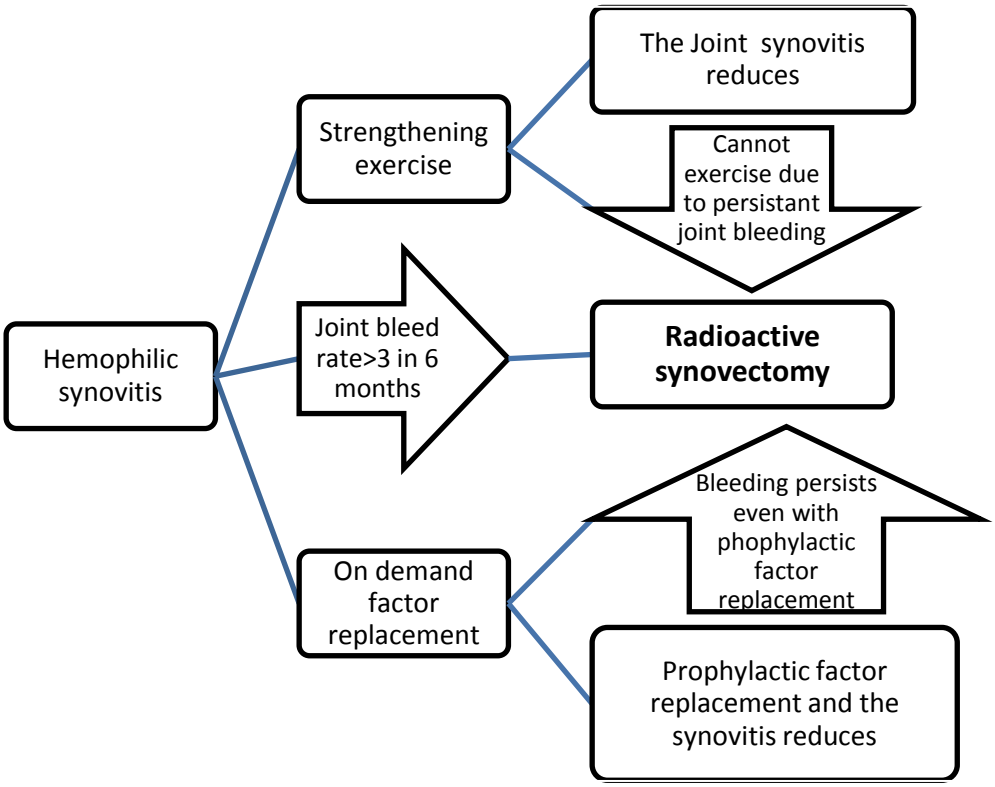


Figure 2. Hemophilic synovitis treatment flow chart.

The drug is procured from France and dispatched and divided into the required doses for each patient by the Nuclear medicine team. The quantity of the drug is calibrated in the factory with an intention to give 5-7 mCi (micro curie) to the knee and 2-3 mCi to the elbow and ankle joint[16].

Patient selection is of primary importance. One should explain that this treatment option reduces bleed rates in the target joint (any joint which bleeds >3 times in 6 months). It might not stop completely. Do not do the procedure in case of joint deformities and proximal muscle weakness as the joint will continue to be unstable after the procedure and will rebleed.

A study by et al Heim showed children as young as four years of age with severe hemophilia developed early hemarthropathy changes and recurrent bleeds which eventually lead to enlarged epiphysis and limb length discrepancy. Therefore, age is not a limiting factor and we should do it at the earliest if the benefit outweighs the risk[1]. The pediatric to adolescent age group (5-20 years) will have the best outcomes as they will have comparatively less cartilage destruction[17].

There are a few mandatory tests that should be done like the pre-op serology and inhibitor assay. Repeated deficient factor replacements can also lead to the development of inhibitors which are antibodies against the factor to be replaced. If the Inhibitor is positive, normal factor replacement will not stop the bleeding and the patient will have to be given FEIBA (Factor eight inhibitor bypass activity).

The clinical evaluation includes measures like Hemophilia Joint Health score (HJHS) [Table 1], and Functional Independence measure in Hemophilia (FISH) for activities of daily living. The annual joint bleed rate (AJBR) and the Annual Bleed rate (ABR) will show the severity of the disease. The school or work absenteeism is something that should be taken into account as theses patients won't be able to travel immediately after a bleed. As not all families know how to take factor replacements on their own the number of hospital visits are also an important factor to take into account to see how much the quality of life is affected by the disease.

Hemophilia Joint Health Score 2.1 - Summary Score Sheet

	Left Elbow	Right Elbow	Left Knee	Right Knee	Left Ankle	Right Ankle
Swelling	☐ NE	☐ NE	☐ NE	☐ NE	☐ NE	☐ NE
Duration (swelling)	☐ NE	☐ NE	☐ NE	☐ NE	☐ NE	☐ NE
Muscle Atrophy	☐ NE	☐ NE	☐ NE	☐ NE	☐ NE	☐ NE
Crepitus on motion	☐ NE	☐ NE	☐ NE	☐ NE	☐ NE	☐ NE
Flexion Loss	☐ NE	☐ NE	☐ NE	☐ NE	☐ NE	☐ NE
Extension Loss	☐ NE	☐ NE	☐ NE	☐ NE	☐ NE	☐ NE
Joint Pain	☐ NE	☐ NE	☐ NE	☐ NE	☐ NE	☐ NE
Strength	☐ NE	☐ NE	☐ NE	☐ NE	☐ NE	☐ NE
Joint Total						

Sum of Joint Totals +
Global Gait Score
(☐ NE included in Gait items)
HJHS Total Score =

NE = Non-Evaluable

Figure 3: Hemophillia Joint health score

The radiological assessment consists of Xray of the affected joint. The X-ray can be used to see the severity of the hemarthropathy using the Petterson scale (see figure 3) but is not a good outcome tool to evaluate synovitis[7]. An important differential diagnosis that can be ruled out in patients with persistent bleeding after a trivial fall is fractures.

Joint Scoring: Pettersson Score
A Radiological Evaluation

Radiological Change	Finding	Score
Osteoporosis	Absent/Present	0/1
Enlargement of epiphysis	Absent/Present	0/1
Irregularity of subchondral surface	Absent/Slight/Pronounced	0/1/2
Narrowing of joint spaces	Absent/<50%/>50%	0/1/2
Subchondral cyst formation	Absent/1 cyst/>1 cyst	0/1/2
Erosions at joint margins	Absent/Present	0/1
Incongruence between joint surfaces	Absent/Slight/Pronounced	0/1/2
Deformity	Absent/Slight/Pronounced	0/1/2

Pettersson H, et al. *Clin Orthop Relat Res*. 1980;(149):153-159.

7

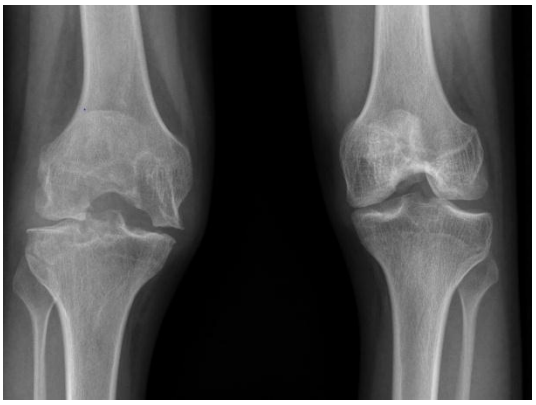


Fig 4:,A. Pettersson score, B. Xray of a 20 year old severe Hemophilia A

Hemophilic early arthropathy detection with ultrasound (HEAD-US) [14] is an ultrasound assessment of the 6 joints (bilateral knee, ankle, elbow) for 2 minutes each. It assesses the synovial thickness, subclinical bleeds, effusion and blood clots in the joints which are signs of disease activity. Myositis ossificans and pseudotumours are complications that an ultrasound can detect (Fig 4).

Figure 5 .Point of care ultrasound (8-12 Hz) used for screening hemophilic synovitis.

The three phase ^{99m} Tc- methylene diphosphonate (MDP) bone scan scintigraphy scan will radiologically detect the amount of synovitis prior to the procedure. It can be repeated in 3 months to see if the synovial hypertrophy is reduced following the radiosynoviorthesis.

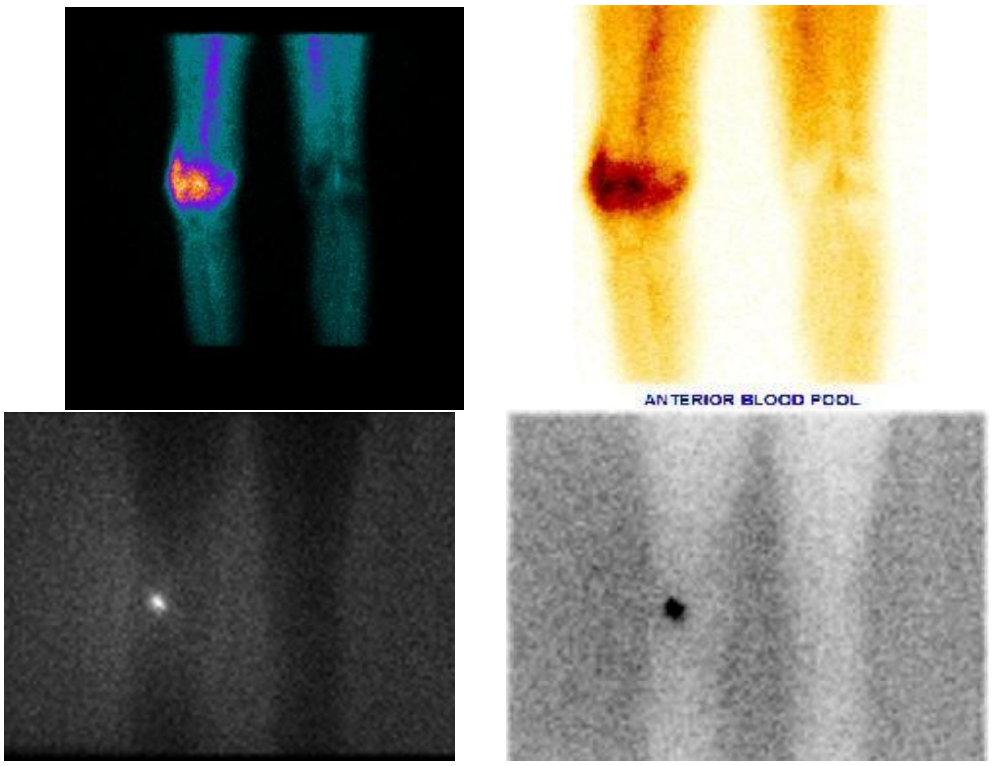


Fig 6. (A).The first picture shows the vascularity of the synovial hypertrophy
(B.) Shows the arterial blood pool
(C.) shows the radioactivity of yttrium in the joint post injection.
(D.) Post injection localization of the drug in the joint capsule and no activity in the extraarticularly or in the puncture channel in the three phase ^{99m} Tc-methylene diphosphonate (MDP) bone scan scintigraphy

Yttrium radiosynoviorthesis is effective in other inflammatory diseases (Table 2). Heim et al did a study which showed a minority had to eventually undergo surgeries such as joint replacement, synovectomies and arthroscopies but they are delayed by 10 years after the yttrium exposure in a prospective study. The 11 year follow up showed good results which is 80% had reduction in the bleeding frequency but only 15 % had no more bleeds at all. In few patients the initial reduction in bleeding reduced dramatically but increased gradually after a few years [10].

Indications for Yttrium Synoviorthesis
1. Hemophilic synovitis
2. Rheumatoid arthritis
3. Pigmented villonodular synovitis
4. Familial mediteranian fever
5. Psoriatic arthritis

Table 1: Indications for Radiosynoviorthesis

In the present scenario patients have a huge factor shortage due to COVID affecting the transportation of factor from Delhi. Radiosynoviorthesis help to reduce the significant economic burden to patients and improves their quality of life. A study done in CMC Vellore on Yttrium over the last 15 years showed that the patients bleed rate reduced dramatically. Specifically, the retrospective study shows that in the 144 joints, 37.4% of the joints had no further bleeds, 56% had <3 episodes per month, and 6% of the joints had (no difference)[19].This is important in countries like India where factor is not easily available for majority of the patients[20].

This is a field physiatrists can grow into as the joint correction and strengthening prior and after the injection is mandatory. Dr. Judy Ann John from CMC Vellore & I hope there will be more physiatrists helping patients with Hemophilia.

References

1. Knobe, K., & Berntorp, E. (2011). Haemophilia and joint disease: pathophysiology, evaluation, and management. *Journal of comorbidity, 1*, 51–59.
2. World Federation of Hemophilia. *Guidelines for the management of hemophilia.* Montreal: World Federation of Hemophilia; 2005.
3. Blanchette P, Rivard G, Israels S, Robinson S, Ali K, Walker I, Stain AM, Blanchette V; Association of Hemophilia Clinic Directors of Canada and Canadian Association of Nurses in Hemophilia Care. A survey of factor prophylaxis in the Canadian haemophilia A population. Haemophilia. 2004 Nov;10(6):679-83.
4. Hoda M Hassab, Hayam Abdel Ghany, Hany Rizk; Quality of Life and Clinical Assessment of Joint Health in Children with Hemophilic Arthropathy. *Blood* 2016; 128 (22): 4972.
5. Rodriguez – merchant EC,Magallon M,Galindo E,Lopez-Carbarcos C. Hemophilic synovitis of the knee and elbow.Clini Orthop 1997;343:47-53
6. Dawson TM,Ryan PF,Street AM et al. Yttrium Synovectomy in Hemophilic arthropathy.Br J Rheumatology 1994;33-351-6
7. Kamaleshwaran Koramadai Karuppusamy, Rajan David, Use of yttrium-90 hydroxyapatite radiosynovectomy as a primary modality of treatment in diffuse pigmented villonodular synovitis of the knee joint: A first case report, Indian journal of Nuclear Medicine,2015, volume 30,Issue Number 1, Page 47-59
8. Heim M, Goshen E, Amit Y, Martinowitz U. Synoviorthesis with radioactive Yttrium in haemophilia: Israel experience. Haemophilia. 2001 Jul;7 Suppl 2:36-9.
9. Gumpel JM, Beer JC, Crawley JC, Farran HE. Yttrium 90 in persistent synovitis of the knee - a single centre comparison. The retention and extra-articular spread of four 90-U radiocolloids. Br J Radiol. 1975 May;48(569):377-81.
10. Falcon –de-Vargas A,Fernandez- Palazzi F. Cytogenic studies in patients with hemophilic srthrosis treated by 198 AU,186 RE and 90 Y radioactive synoviorthesis.J Pediatr Orthop 2000;9:52-4
11. Sholter D,Davis P.Radiochemical synovectomy.Scand J Rheumatology 1997;26:337-41
12. Eanm procedure guidelines for Radiosynovectomy eanm.org/publications/guidelines/gl_radio_synovectomy.pdf
13. Erken EHW.yttrium -90 in the management of Hemophilic arthritis.Ort Traum Oggi 1990;10:33-9
14. Erken EHW, Schepers A, Sweet MBE, Lurie A. Use of Radioactive colloid(^{90}Y) in the treatment of Hemophilic arthropathy.J Bone Joint Surg(Br) 1979;61B: 257.
15. Gumpel JM .90 Y colloids in chronic synovitis of the knee . Rheum Rehab 1979; 18:38-41.
16. Editorial.medical synovectomy .BMJ 1974;2 (29):682.
17. Erken EH. Radiocolloids in the management of hemophilic arthropathy in children and adolescents. Clin Orthop Relat Res. 1991 Mar;(264):129-35. PMID: 1997228
18. Li J, Guo XJ, Ding XL, Lyu BM, Xiao J, Sun QL, Li DS, Zhang WF, Zhou JC, Li CP, Yang RC. [Clinical application and optimization of HEAD-US quantitative ultrasound assessment scale for hemophilic arthropathy]. Zhonghua Xue Ye Xue Za Zhi. 2018 Feb 14;39(2):132-136. Chinese. doi: 10.3760/cma.j.issn.0253-2727.2018.02.011. PMID: 29562448; PMCID: PMC7342577.
19. Sunny Saumya Sara, Hephzibah Julie, Shanthly Nylla, Oommen Regi, Mathew David, Abraham Aby Yttrium-90 synovectomy in hemophilic arthropathy: An institutional experience for 15 years .Indian Journal of Nuclear Medicine.Year : 2020 / Volume: 35 /Issue Number: 2 / Page: 143-146
20. Nidra I. Rodriguez, W. Keith Hoots,Advances in Hemophilia: Experimental Aspects and Therapy,Pediatric Clinics of North America Volume 55, Issue 2,2008,Pages 357-376,ISSN 0031-3955

Evidence Based Medicine (how we got here..)

Dr. Bineesh Balakrishnan, Asst professor Dept of PMR, Sree Narayana Medical College

"Most histories of medicine are strikingly odd. They provide a clear account of what people believed they were doing, but almost none at all of whether they were right."- Druin Burch

We now live in the era of Evidence Based Medicine. Was Medicine always evidence based?? When we use modern science(or even what is common sense to us now) to judge the efficacy of historical treatments, it is obvious that most interventions were useless or worse like..

The Ostrich egg poultices applied by ancient Egyptian physiscians to heal head fractures!.

The Egyptian Keeper of the Royal Rectum, prepared enemas for the royal family, in an attempt to keep them healthy![1].

The practice of bloodletting which was for too long considered to be a restorative process, right from the ancient Greeks to George Washington's doctors![1].

Convictions & time..

Nietzsche once stated that –"Convictions are bigger enemies of the truth than lies". When we look at how unethical & unscientific Medicine was in the past, the above statement is raised to the level of an axiom…This whole article contains ample evidence for the same...Let's see...

Hippocrates believed that sneezing arises from the head, owing to the brain being heated, or the cavity in the head being filled with humours[2].

Galen, the second century physician to the Roman emperors has influenced generations of Physicians. His writings were considered to be indisputable sources of authority for more than a 1000 years. *Galen claimed that he had revealed 'the true path of medicine' & yet he never conducted anything even close to a modern experiment. His healing potions 'healed all the patients in a short interval of time, unless the case was deemed incurable'!!(a success rate of 100%!!!).*

Galen also believed that blood was the dominant humour, and therefore an excess of this humour was treated by bloodletting and purging!!

Besides these famous figures many others propounded theories which were supported only by their convictions.

The U.S President falls ill...

When George Washington fell ill with sore throat, cold & chills his physicians resorted to the following treatment regime:-

- Bleeding him relentlessly, removing nearly 82 ounces of blood in less than 16 hours[3]
- Dosing him with mercury to cause diarrhoea
- Inducing vomiting
- Raising blisters by applying hot cups to the old President's skin[1]

Sadly, a physician in Aristotle's Athens, or Nero's Rome, or Medieval Paris, or Elizabethan London would have nodded at all the components of the above mentioned odious treatment regime. It's possible that the treatments helped but not enough to overcome the disease that took Washington's life, or that they didn't help at all, or that the treatment regime hastened his death. Ignorance & professional hubris remained the defining features of medicine at that point in time. As surgeon & historian, Ira Rutkow observed, physicians back then who furiously debated the merits of various

treatments & theories were akin to 'blind men arguing on the colours of the rainbow'[1].

James Lind & Scurvy....

The cure for this plague of certainty was tantalizingly close to discovery in 1747.
Scurvy was very common among British sailors in the 18th Century. One British expedition to raid Spanish holdings in the 1740s lost 65% of the sailors to scurvy. Long sea voyages resulted in more people dying from scurvy than from enemy action. In 1747, Dr.James Lind, the Surgeon's mate on the H.M.S Salisbury, carried out one of the first controlled clinical trials recorded in medical history. The essence of what he did is described here.

He took 12 men suffering from similar symptoms of scurvy, divided them into 6 pairs & treated them with remedies suggested in popular medical literature:-

1. A quart of cider a day
2. 25 drops of elixir of vitriol, three times a day
3. Half a pint of seawater a day
4. A nutmeg-sized paste of garlic, mustard seed, horse radish, balsam of Peru, & gum myrrh three times a day
5. Two spoonfuls of vinegar three times a day
6. Two oranges & one lemon a day

The sailors who took citrus fruits recovered in a week, & even helped in taking care of the other sailors[4]. Unfortunately nothing much changed & here's why..

Dr. Lind's 'A Treatise of the Scurvy' containing a celebrated review of literature on the disease appeared sometime in 1753. In this 450 page document, the groundbreaking experiment aboard the H.M.S Salisbury was given only 4 pages. Thus this document didn't do much for the afflicted sailors or for the furtherance of scientific medical experiments. Like author Druin Burch noted:" He (Lind) failed so completely to make sense of his own experiment that even he was left unconvinced of the exceptional benefits of lemons & limes"[1,4].

In 1795, a year after Dr.Lind's death, the Royal Navy, following advice from its own medical staff made the issue of lemon juice compulsory on ships...

Small pox vaccination...

One question, much debated in the 18th century, was whether smallpox inoculation was a good thing. In England inoculation was generally favoured, in France it was opposed. Various calculations were made as to the death rate from smallpox which was considered to be around one in ten, excluding fatalities of those under 2 years old. Other calculations were 1 in 12 and 1 in 7. This was compared to the death rate from inoculation which James Jurin, secretary of the Royal Society, calculated at 1 in 91. The Swiss mathematician, Daniel Bernoulli calculated that inoculation increased the average life expectancy by two years. A further problem was that people inoculated with smallpox could spread it to others and this was not taken into account in calculating death rates from inoculation. If people who were inoculated could be isolated for a period, then the figure might not be too high, but then if people who got smallpox naturally were isolated that would reduce the death rate from normal smallpox. An additional problem was that the rate of smallpox infection varied considerably from large cities where nearly everyone would, sooner or later get smallpox and the small towns and villages where most people in the 18th century lived, and many people could live their lives without getting smallpox. Modern estimates of the death rate from inoculation are as high as 12%, not much better than the death rate from normal smallpox infection[5].

But since then vaccine research has burgeoned into a multi-billion dollar industry. The Covid vaccine race for example is worth 750 billion dollars. Vaccine companies are competing to provide vaccines that meet scientific standards & have few (if any) adverse effects…

J'ai tort !!(that's 'I'm wrong' in French!)

French physician Pierre Charles Alexandre Louis (1787-1872) routinely bled patients suffering from pneumonia. He decided to assess bloodletting's efficacy using the new –numerical method‖ (statistics) to see how many lives he was saving. Bloodletting was not considered archaic or harmful back then & in 1833 France imported 42 million leeches for medical use.

According to American physician J.J. Jackson (1836): –"If anything may be regarded as settled in the treatment of diseases, it is that bloodletting is useful in the class of diseases called inflammatory; and especially in inflammations of the thoracic viscera"[6].

Physicians' daily application of the therapy, followed by numerous recoveries, provided all the proof they needed that bloodletting was effective. To his dismay Louis's statistical analyses showed that bloodletting increased rather than decreased mortality. This physician considered his findings startling but eventually accepted them.

J.J Jackson, a physician from Massachusetts General Hospital doubted Louis's findings but found similar results with his own patients when he did a statistical comparison.

Thousands of years of direct clinical experience with phlebotomy accomplished two things:

(1) It increased mortality among patients with systemic diseases such as pneumonia

(2) It convinced millions of physicians and their patients that it was saving lives.

Sir William Osler (1849–1919) listed several indications for bloodletting in his famous textbook *Principles and Practice of Medicine*. In the first edition in 1892, Osler wrote:

–During the first five decades of this century the profession bled too much, but during the last decades we have certainly bled too little. Pneumonia is one of the diseases in which a timely venesection may save life. To be of service it should be done early… the abstraction of from twenty to thirty ounces of blood is in every way beneficial[7].

At a meeting held in London at the Royal Society of Medicine in April 1927 on the subject of venesection, it was agreed that the procedure should not be employed on chronic anaemias and low blood pressure, especially in the elderly. The indications upon which most of the group appeared to agree were polycythaemia and certain cases of high blood pressure among the chronic group, and acute conditions associated with lividity and right heart dilatation. It was also agreed that it was beneficial in sunstroke. At this meeting, the distinguished physician Sir William Hale-White (1857–1949) commented that if the discussion had taken place eighty or ninety years previously several hundred people would have been bled in the afternoon in London, since 'every person who looked after his health in those days was bled every spring and autumn'. Hale-White reminded the audience that in 1840 a doctor was charged with malpractice for failing to bleed a patient with pneumonia, but subsequently there had been a great change in the frequency of bloodletting. Another speaker at this meeting, a well-known physician, Sir William Wilcox, considered venesection indicated in five classes of cases: right-sided dilation of the heart; convulsions; heatstroke; polycythaemia; and violent asphyxia from drowning or hanging. Even in relatively modern times, therefore, the indications for bloodletting were considered a suitable topic for discussion at a medical meeting in London[8].

The Placebo…

The word *placebo* first appeared in medical literature in the early 1800s. *Hooper's Medical Dictionary* of 1811 defined it as –'an epithet given to any medicine more to please than benefit the patient'.

But it wasn't until 1863 that United States physician Austin Flint conducted the first medical experiment comparing a dummy remedy to an active treatment. He treated 13 patients suffering from rheumatism with an

herbal extract instead of an established remedy.

–This was given regularly, and became well known in my wards as the 'placeboic remedy' for rheumatism,‖ Flint wrote in his 1886 book *A Treatise on the Principles and Practice of Medicine*. –The favourable progress of the cases was such as to secure for the remedy generally the entire confidence of the patients[9].

"Is the application of the numerical method to the subject matter of medicine a trivial & time wasting ingenuity as some hold, or is it an important stage in the development of our art, as others proclaim it?" – The Lancet, 1921

The Streptomycin trial by the M.R.C …

The first randomized curative trial was designed to assess the efficacy of Streptomycin in treating Pulmonary Tuberculosis, in 1946, by the M.R.C of U.K. The committee behind this trial was chaired by Sir Geoffrey Marshall, &the statistician was Sir Bradford Hill. The trial began in 1947. As the amount of Streptomycin available for the trial was limited, it was ethically acceptable for the control subjects to be untreated (something considered unethical these days). A key advantage of Dr.Hill's randomization scheme over alternation procedure, was 'allocation concealment' at the time patients were enrolled in the trial. Another key feature was that blinding was used so that the objective measures such as X-ray interpretation was done by experts who didn't know the patient's treatment assignment[10]. The trial showed that Streptomycin was effective in treating Pulmonary Tuberculosis. Dr Hill instituted randomization – a new statistical process which has been described in detail in the landmark BMJ paper of 1948[11]. Though the trial was a groundbreaking one, Sir Hill was anxious that physicians will be unwilling to give up the doctrine of anecdotal evidence…

Smoking & Cancer...

After World War II the great majority of the adult population smoked and lung cancer deaths were rapidly increasing. If you've watched the movie- 'The King's Speech', you must have noted the scene where one doctor states that smoking relaxes the throat & has no ill effects. As always human beings justify their convictions, & don't want them shattered, but then time has schemes of it's own!!

Bradford Hill (who was pivotal in the Streptomycin trial), Edward Kennaway, Percy Stock and Dr Richard Doll were asked to investigate whether smoking was a cause of the increasing number of lung cancer deaths. Smoking was only one possible explanation, others such as increased air pollution especially from motor vehicles were considered to be as likely or more likely the cause of increased lung cancer deaths, than smoking. The asphalting of roads was considered to be another possible cause of the escalating lung cancer deaths. Given that most adults smoked it was difficult to find a suitable control group of non-smokers. The investigation was conducted by creating a detailed questionnaire which patients suspected of having lung cancer completed. The questionnaire was also completed by patients who had other cancers and also by patients in hospital for reasons other than cancer to act as two control groups. It was found that 99.7% of the lung cancer patients smoked against 95.8% of the control group patients. This was not a great difference but it was also found that 4.9% of the lung cancer patients smoked 50 cigarettes a day as opposed to only 2% of the control group patients. The lung cancer rate amongst those smoking 50 cigarettes a day was over double for lung cancer patients than for the control group. The more people smoked the greater their chances of getting lung cancer.

The study conducted by Doll and Bradford Hill had looked at lung cancer patients and looked back in time at their smoking habits. They then decided to do a study of healthy people investigating their smoking habits and then observing how their health developed in the future. Doll and Bradford Hill decided to

do the study on doctors, 40,000 of whom filled in and returned their questionnaire. Two and a half years later enough doctors had died for Doll and Bradford Hill to be able to show that the more the doctors smoked the greater the likelihood they had died of lung cancer. It was eventually found that doctors smoking 25 cigarettes per day were 25 times as likely to develop lung cancer compared to non smokers[5].

Archibald Cochrane's Ordeal…

When the dermatologist saw spots on the back of the patient's hand, it aroused suspicion & a bit of the skin was biopsied. The histopathological report stated that it was basal cell carcinoma. The patient, Archibald Cochrane (being a Physician), knew that these cancers seldom spread. The cancerous tissue was surgically removed, but Cochrane decided to consult a reputed Cancer Surgeon to get a second opinion. The surgeon found a lump in his axilla & advised excision of the lump, Cochrane agreed to undergo the procedure. After the surgery, when the anaesthesia wore off, the patient was dismayed to find his whole chest wrapped in bandages. The surgeon walked with a grim expression, into the patient's room & told him:" I must tell you the truth. Your axilla is full of cancerous tissue. I have done my best to excise it & have removed your Pectoralis Minor but I may not well have saved your life."[1]

Cochrane sank into abysmal despair. –"For a moment the world seemed to end.", he later wrote –"After a short period of surprise & shock I turned as far as I could on my side & sobbed unashamedly. I do not remember much about the rest of the day…..The next morning, with a clear mind I worked out a simple plan of how I would spend my remaining time..A curious feeling of peace came over me when I had completed the plan & I feel asleep." In the days that followed friends & family came over to offer him whatever comfort they could, & as is usual, Cochrane found all this rather awkward[1].

This dismal moment happened sometime in 1956, but Archie Cochrane did not die, but went onto be a revered figure in medicine. He did not have cancerous growth in his axilla to begin with, as was confirmed by the histopathological report. Cochrane was more startled with his reprieve than with his death sentence. He now realized that if he had doubted the Cancer surgeon's convictions, & waited for the pathologist's report this ordeal could have been averted. It's human nature to make up our minds quickly & then be slow (at times really slow) to change them. As has been repeatedly stated in this article, if we don't examine the mistakes we've made, & change our minds quickly, we will keep repeating our mistakes, leading to stagnation, which as shown before can go on for years, lifetimes or longer!!

Archie Cochrane observed that in the 1950s & 1960s the N.H.S was promoting healthcare that lacked scientific validation & was least interested in changing the status quo. The practitioners were depending on their judgement & didn't feel the need for scientific validation for diagnosing or treating diseases. Cochrane abhorred this attitude calling it ' The God Complex'!

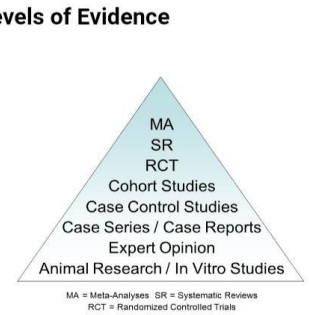
When hospitals started cardiac care units to treat patients recovering from heart attacks, Cochrane proposed a randomized trial to determine whether the new units delivered better results than the old treatment which was to send patients home for monitoring & bed rest. Physicians believed that it was obvious that cardiac care units were superior & denying patients optimal care would be unethical. But Cochrane didn't back down. His past term as prison camp physician treating fellow P.O.Ws during World War II had given him ample experience to stand up to authorities. Cochrane got his trial: some patients, randomly selected, were sent to the cardiac care units while others were sent home to follow the old treatment regime of bed rest & monitoring. Before the trial was over, he met with a group of the cardiologists who had tried to stop his experiment. He told them that the preliminary results showed that the difference in outcomes between the two treatments was not statistically significant,

but it appeared that patients might do slightly better in the cardiac care units. They severely reprimanded him. "Archie" they said, "we always thought you were unethical. You must stop the trial at once." Then Cochrane revealed that he had played a little trick. He had reversed the results, home care had done slightly better than the cardiac care units. He had silenced his critics, & the N.H.S was convinced that clinical trials were essential to decide which treatment regime was superior. As mentioned before, the groundbreaking trial of Sir Hill had also hinted that controlled trials were the way to go, as far as modern medicine was concerned.

The Cochrane Collaboration was established in 1993 to produce & disseminate reliable & up-to-date information to support decision making in health care[12]. Over 470,700 records were available and searchable from the combined Cochrane Library database collection, by 2005 alone[12].

Dr. David Sackett's trailblazing work….

The EBM movement was boosted to a great extent in 1981, when a group of clinical epidemiologists at McMaster University (Hamilton, Ontario, Canada), led by David Sackett, published the first of a series of articles in the Canadian Medical Association Journal advising physicians how to appraise the medical literature[13].The actual term ‗evidence-based medicine' was first coined by Gordon Guyatt, the Program Director of Internal Medicine at McMaster University from 1990 to 1997, who was one of Sackett's mentees in 1991[14]. In 1967, at the young age of 32, Sackett founded the first Department of Clinical Epidemiology in the world at the newly-minted medical school at McMaster University. There were other departments of epidemiology before, but they dealt with public health issues and statistics beyond the reach of the average clinician. David Sackett demystified all this by applying the methodologies from Public Health and biostatistics to individual patients at the bedside.He published two books- 'Clinical Epidemiology' & 'Evidence-Based Medicine: How to Practice and Teach EBM'[15]. The levels of evidence were first outlined by Sackett in his article published in the late 1980s. The diagram below shows the levels of evidence as they are in today's practice.



(Source of the image:- https://guides.library.stonybrook.edu/evidence-based-medicine/levels_of_evidence)

According to Sackett, the key components to EBM are:

(1) Consideration of the patient's expectations (wishes);

(2) Our clinical skills

(3) The best evidence available to us.

In the past, decisions were made on observations and the dogma of the experts. Sackett did not think much of the experts. The truth, according to Sackett, can only be found in randomized trials when these are feasible and avoiding the influence of bias. In 1994, he left McMaster University and accepted a position as Foundation Director of the Centre for Evidence Based Medicine at Oxford University in the UK. Upon his retirement from Oxford, he returned to Canada, where he mentored young investigators at the Trout Research & Education Centre. Until his death he continued to lecture to students in Clinical Epidemiology at McMaster. He loved to interact with young people and share his ideas. He was a likable person who did not compete with his students and mentees, who are in fact his legacy. He was a legend in his own time[15].

How far we've come…

As is obvious from this article, we have come a long way, edging towards evidence based medicine. In this article, I have tried to mention the most important events that have shaped medicine & led us towards evidence based medicine. For those of you who are interested in reading more on this, do check out the references provided at the end (also check out pubmed, https://www.jameslindlibrary.org/https://www.cochranelibrary.com/https://www.clinicaltrials.gov/). Clinical trials are now the norm in deciding treatments, efficacy of drugs & vaccines in modern medical practice. In the last 21 years alone 370,361 trials have taken place in nearly 219 countries[16].

"A 21st century clinician who cannot critically read a study is as unprepared as one who cannot take a blood pressure or examine the cardiovascular system."- BMJ 2008:337:704-705

How to use E.B.M…

As modern medical practitioners it is essential to know how to use Evidence based medicine. This can be done by the 6 A technique:-

1. Ask a structured question
2. Acquire relevant evidence
3. Appraise the evidence
4. Evaluate *Applicability* of the evidence to patient care
5. Act by involoving the patient in the decision making process
6. Assess the patients before the beginning of the EBM cycle and also assess performance of the patients at the end of this process[17].

Hope everyone enjoyed reading this article as much as I enjoyed writing it….

References

1. Superforecasting – The Art & Science of Prediction by Philip Tetlock & Dan Gardner, Penguin Random House, U.K
2. Source Book of Medical History, L Clendening (ed), p. 19. Dover Publications, New York
3. https://washingtonpapers.org/resources/articles/illness/
4. https://www.bbc.com/news/uk-england-37320399
5. https://figshare.com/articles/journal_contribution/The_History_of_Medicine_pdf/4219722 (pdf available at this link)
6. A Morabia (1996). P. C. A. Louis and the Birth of Clinical Epidemiology. J Clin Epidemiol 49:1327-133
7. Osler W. Principles and practice of medicine. 1st ed. London & New York: Appleton & Co; 1892. p. 530.
8. Venesection. Br Med J 1927; 1:721–3. http://dx.doi.org/10.1136/bmj.1.3458.721-a
9. Legumes, lemons and streptomycin: A short history of the clinical trial (nih.gov)
10. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3149409/
11. MRC Streptomycin in Tuberculosis Trials Committee. Streptomycin treatment of pulmonary tuberculosis. BMJ. 1948;2:769–83.
12. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1175814/
13. Sackett D. How to read clinical journals: I. why to read them and how to start reading them critically. Can Med Assoc J. 1981;124(5):555-558.
14. The periodic health examination. Canadian Task Force on the Periodic Health Examination. Can Med Assoc J. 1979;121(9):1193-1254
15. A Brief History of Evidence-Based Medicine (EBM) and the Contributions of Dr David Sackett by Achilleas Thoma, MD, MSC, FRCS(C), FACS; and Felmont F. Eaves III, MD, FACS Aesthetic Surgery Journal 2015, Vol 35(8) NP261–NP263
16. https://clinicaltrials.gov/ct2/resources/trends
17. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5037940/

How do I write for the Invited Author submission

Dr. Ravi Sankaran, Associate Professor, Dept of PMR, Amrita Institute of Medical Sciences

Do you want to share your expertise with colleagues? Writing is one of the best ways to do this. Sadly MBBS, doesn‘t prepare us for scientific writing. Writing accomplishes a vital service. Clinical challenges faced today make us grow and may be worth writing about tomorrow. Connecting with the world around is critical to survival in modern times. Writing is a step towards this. This is an introduction to writing for the ‘Invited Author‘ section of our journal.

What is the ‘Invited Author‘ submission?

The article basically says, _In my opinion this is what you should do‘. Easy enough, we do this every day with family, patients, subordinates, etc. The only difference is instead of politics or the best smartphone, it is a clinical topic we discuss. This is not critiqued by peer review, only I read these. This isn‘t a license to write nonsense. You will be held accountable for your words. The only forum for disagreement though will be our conferences and whatsapp.

So how do we write for an ‘Invited Author‘ submission?

Let us define boundaries with examples. One extreme is the article that is pure opinion without science to back it. On the other, a new writer may gather a hundred papers and present all of them evenly. This exhaustive review would have no solid conclusions about when and for whom this information is applicable. Both are problematic. Optimally this submission type presents ideas and models, minimal referencing, and speculations on where things are moving. It would come from a basic science approach, the therapeutic indications and limitations, and from there review the literature concluding with the author‘s opinion on when this should be used and for whom. Revisions will be ordered if in-line citations are absent. This is a common trend amongst our authors. Most write a nice opinion paper but give no credit to facts that are clearly not their work. This is called plagiarism and is a crime. Sadly our MBBS gold medalists excel in this.

What comes next? The article will provoke or evoke responses from your colleagues. More importantly it is an excellent way to keep up with publications, ideas, trends, and models in a specific topic. The goal here is to develop a more critical approach to reading published papers. Point out the flaws in what is otherwise taken for granted. The goal is not to attack anyone, but to make your mind keener. These articles are precluded with a grain of salt disclaimer. (1)

FAQ

Checklist

1. Frame a research question.
2. Pick your audience- This is preset at Kerala PMR practitioners who read our journal.
3. Make an outline. Write in questions you need to answer to complete the paper.
4. Harvest available data- Pubmed search styles, Google, Clinical key, EBSCO. Get the relevant articles and

save them in a file labeled _literature review‘.

5. Read the articles, and highlight the parts you feel are useful. If you read in pdf you can copy the same lines with citation into a word document. Read what you‘ve copied and summarize/ rephrase the lines in your own words.
6. Insert these lines into the outline and read it. If it makes sense, move to the next paragraph. If not move the fragments around until they flow. Its best to have the full citation next to the relevant lines.

7. Once all the paragraphs are done read and see if the concepts flow.

8. If everything seems ok, make a separate copy and remove all the in-line citations.

9. Find a victim to read this. Preferably this person is from PMR or a discipline related to the topic. Get a group of victims to spread out the impact of your learning process, or you‘ll be the victim of Stockholm syndrome. Its fine if you only ask me to review. You may not be ok after I do though. 😊

10. Once they say everything is ok, make your Bibliography and submit.

That‘s easy, right? Things worth working for rarely are.

When do I write? Continuously make an effort, between patients, or during your free time. Most people start when they are totally free (rare) and when that happens they have nothing to work with or tire of the harvesting/ processing steps. Break it up into small tasks that build up to the end. Filling in the outline, paragraph by paragraph is do-able.

Some tricks to be interesting are: good word and sentence rhythm, carefully drawn word pictures, using pronouns to relate to the reader and buy-in and compelling leads and conclusions.

1. What is the Invited Review article? (Caveman A, The invited review ? or, my field, from my standpoint, written by me using only my data and my ideas, and citing only my publications. J Cell Sci. 2000;113(Pt 18):3125-3126.)

'Journal scan'

Dr.Nitin Menon, Noufal Ali, and Bineesh Balakrishnan

EULAR recommendations for the management of rheumatoid arthritis with synthetic and biological disease- modifying antirheumatic drugs: 2019 update.

Josef S Smole , Robert B M Landewé, Johannes W J Bijlsma, Gerd R Burmester, Maxime Dougados, Andreas Kerschbaumer, Iain B McInnes, Alexandre Sepriano, Ronald F van Vollenhoven, Maarten de Wit, Daniel Aletaha, Martin Aringer, John Askling, Alejandro Balsa

This recent update to existing EULAR guidelines have recommended that:

1. Initially start with Methotrexate (leflunamide, sulfasalazine) plus glucocorticoids.
2. If response is inadequate, stratify according to risk factors.
3. If poor prognostic factors are present (autoantibodies, high disease activity, early erosions, failure of two classical DMARDs) and biologic (b) DMARD or Janus Kinase Inhibitor (JKI) should be added to classical DMARDs.
4. If this fails, substitute bDMARD or JKI with any other drug from same class.
5. When remission is achieved, continue low dose DMARDs.

Ann Rheum Dis 2020;79:685–699.

Recent Advances in the Treatment of Rheumatoid Arthritis

Mahajan TD, Mikuls TR

Newer drugs are available now for management of rheumatoid arthritis which enable more tight control of the disease activity and allow for remission to be achieved in a larger group of patients. The newer drugs are – Biosimilars, which are structurally dissimilar but clinically similar to biologic DMARDs, Interleukin (IL) - 6 inhibitors like Sarilumab and targeted synthetic DMARDs or JAK inhibitors like tofaticinib. Tapering of DMARDs on achieving remission is being reported by many studies but it is recommended to continue both conventional and bDMARDs to maintain remission. Additional flare-ups while on remission require dose escalation with the same agents.

CurrOpinRheumatol 2018, 30:000–000

An updated algorithm recommendation for the management of knee osteoarthritis from the European Society for Clinical and Economic Aspects of Osteoporosis, Osteoarthritis and Musculoskeletal Disease (ESCEO)

Bruyere O, Honvo G, Veronese N et. al.

Osteoarthritis is a common condition leading to joint debility and chronic pain in the elderly population. 2014 guidelines were updated in this article to reflect newer issues with safety and to provide step-wise escalation in treatment of OA. Non-pharmacological treatments receiving Grade

1 recommendation are – education/information access, weight loss and targeted exercise programs. There is paucity on data about benefits of exercise on very elderly patients(age 75 and above) .Pharmacologic treatment recommendations (strong recommendations) are:-

Prescription synthetic glucosamine sulphate (GS) should only be used for long term background therapy (along with non-pharmacological measures) and all other over the counter glucosamine formulations should be discouraged.

1. Prescription chondroitin sulphate (CS) may be used as an alternative to glucosamine sulphate.
2. Topical NSAIDs should be used as add on analgesia before using oral NSAIDs if long term-measures do not lead to adequate control.
3. Total knee replacement may be recommended to patients with end-stage knee OA.

Seminars in Arthritis and Rheumatism. 2019;49:337-50

Advances in Nanomedicine for Treatment of Ankylosing Spondylitis

Yanhai Xi, Tingwang Jiang, BirendraChaurasiya, YanyanXhou, Jiangmin Yu, Jiankun Wen, Yan Shen, Xiaojian Ye, Thomas J Webster.

Conventional drugs including DMARDs are effective in ankylosing spondylitis but all have the twin drawbacks of long-term safety and increase in financial burden for continuation of treatment. Partly, the problem is caused by degradation of drug in the body, needing higher doses to achieve therapeutic effect. Nanotechnology based drug delivery systems can deliver both hydrophilic and hydrophobic drugs by enveloping them in synthetic coating of phospholipids which retard drug release and degradation by the body. So for example, if conventional NSAIDs are delivered in nanoform, a usual dose can last for much longer as compared to current drug delivery systems. Same principle applies to DMARDs as well. Drug preparation in hydrogel form can prevent rapid clearance of hyaluronic acid used in the treatment of OA. Nanodrug delivery systems can potentially revolutionise pharmacological management of joint conditions in the future.

International Journal of Nanomedicine 2019:14 8521–8542

Current and Emerging Pharmacotherapy for Fibromyalgia.

RoieTzadok, Jacob Ablin.

Fibromyalgia syndrome is a widespread entity affecting between 1-5% of people. Pregabalin, Duloxetine and Milnacipran are currently the only FDA approved treatments for this condition. However this review also covers the role of amitryptiline, escitalopram, cyclobenzaprine, lacosamide, naltrexone, tramadol, ketamine and cannabinoids in the management of fibromyalgia syndromes. The anticipated increase in widespread use of cannabinoids promises to revolutionise this otherwise difficult to treat condition.

JN. Pain Res Management. 2020.

Rheumatoid arthritis and depression: an inflammatory perspective

Louis Nerurkar, Stefan Siebert , Iain B McInnes , Jonathan Cavanagh

The coexistence of immune-mediated inflammatory diseases with depression has long been recognised. Data that illustrate the intimate associations between peripheral and brain immune responses raise the possibility of shared pathophysiological mechanisms. In this Review, the authors assess this evidence in relation to rheumatoid arthritis and depression, with a focus on innate immune and molecular responses to inflammation, and discuss the challenges of assessing causation in this population, acknowledging the difficulty of assessing the confounding and contributory effects of pain and fatigue. They also discuss how future clinical and preclinical research might improve diagnosis of depression in people with rheumatoid arthritis and shed light on mechanisms that could be substrates for therapeutic interventions.

Lancet Psychiatry. 2019 Feb;6(2):164-173.

Dietary Habits and Nutrition in Rheumatoid Arthritis: Can Diet Influence Disease Development and Clinical Manifestations?

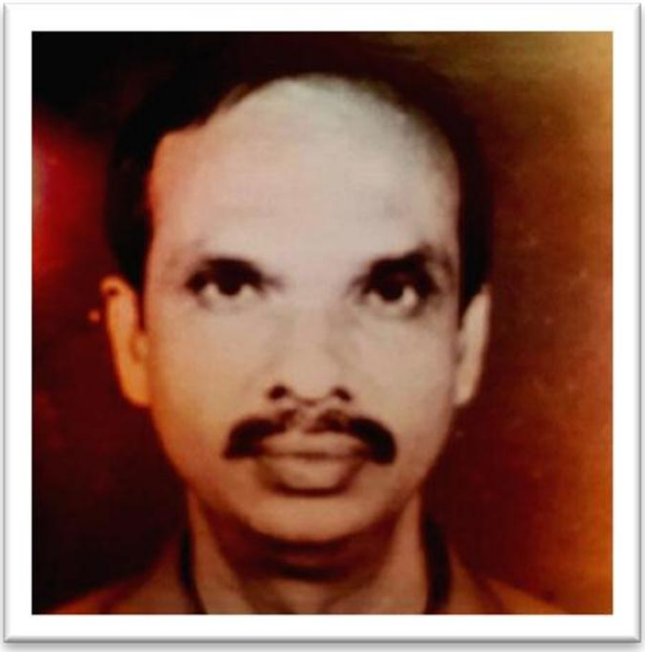
Chiara Gioia , Bruno Lucchino , Maria GraziaTarsitano , Cristina Iannuccelli , Manuela Di Franco

Genetic and environmental factors determine RA susceptibility. In recent years, an increasing number of studies suggested that diet has a central role in disease risk and progression. Several nutrients, such as polyunsaturated fatty acids, present anti-inflammatory and antioxidant properties, featuring a protective role for RA development, while others such as red meat and salt have a harmful effect. Gut microbiota alteration and body composition modifications are indirect mechanisms of how diet influences RA onset and progression. Possible protective effects of some dietary patterns and supplements, such as the Mediterranean Diet (MD), vitamin D and probiotics, could be a possible future adjunctive therapy to standard RA treatment. Therefore, a healthy lifestyle and nutrition have to be encouraged in patients with RA.

Nutrients. 2020 May 18;12(5):1456.

OBITUARY

.



Senior Physiatrist & Retired DMO in Charge of Thrissur district, **Dr. M.L Rajagopalan,** passed away on the 13th of June 2021. He succumbed to Covid-19 after battling against it for a week at Aster Medicity. He had taken great efforts to promote health care & Physiatry. He had played a big role in setting up the IMA Blood bank, which has since then served tens of thousands of patients. He was also in charge of the PMR Department in District Hospital, Thrissur, during its early days. He is survived by his wife, son & daughter. Kerala Chapter of IAPMR bemoans this loss & prays for the bereaved family....

"How can the dead be truly dead when they still live in the souls of those who are left behind?"- Carson McCullers

Members in action

IAPMR National Executive Committee inductees from Kerala Chapter

It is a distinguished honour to the Kerala Chapter of IAPMR, that three of its eminent members have been inducted to the Executive Committee of IAPMR.

Dr.Muralidharan PC
(President of Kerala Chapter)

Dr. Selvan P
(Secretary of Kerala Chapter)

Dr. Arun A John
(Treasurer of Kerala Chapter)

The COVID PMR scenario

- Every PMR unit in the state is actively involved in the COVID duties, both ward and ICU.

- **Dr. Santhosh Raghavan**, GMC Alleppey, reports that all the faculty members of the Department are playing an active role in COVID management. He is the Officer-in-Charge of delegating COVID duties to Junior and Senior Residents in various COVID wards, Intensive Care Units and COVID triage & prepares the duty list of medical officers to supervise the COVID vaccination as well. Dr. Charvakan S, Assistant Professor, is the current Assistant Nodal Officer of Corona care. Dr. Sivaram, Assistant Professor, represents the Department in the Post- COVID Clinic. Senior Resident Dr. Bhavya K.P also regularly takes COVID duties.

E-Sanjeevani portal for PMR, was initiated by Dr. Selvan P. (Secretary, IAPMR, Kerala chapter and Dr. Muralidharan (President, IAPMR, Kerala chapter). This includes PMR personnel all over the state.

Dr. Sooraj Rajagopal, Associate professor, PMR dept., GMC, Kozhikode

- COVID nodal officer for IQRAA COVID Hospital, Kozhikode, IQRAA Dialysis centre , IQRAA COVID Hospital.
- KASP Nodal officer for NMCH AND PMSSY
- Co-Convenor for NMC Regional Centre For Medical Education Technologies
- Completed ACME medical Education course
- Co-author for 4 COVID -related papers

- Donated around 4 lakh rupees worth of medical and other supplies to GMCH COVID wards, casualty, IMCH and PMR Dept. Items included 130 NRBMasks,110 NIV masks, Pulse Oximeter s, 100 Anaesthesia circuits , induction cooker, Electronic BP apparatus, Storage bins, Backrests, Glucometers and strips, 1000 Paper plates and glasses, Surgical masks, Nitrile gloves etc..

FELLOWSHIP PROGRAMS PROPOSED BY KERALA CHAPTER

2. FNB in Musculoskeletal rehabilitation and Pain

3. FNB in Neurorehabilitation

4. FNB in Paediatric Rehabilitation

ANNUAL STATE CONFERENCE AUGUST 2021-VIRTUAL MODE

Tentative Date	: August 28th & 29th
Theme	: Cognitive Rehabilitation
Organising Chairman	: Dr. Muralidharan P.C
Organizing Secretary	: Dr. Bineesh Balakrishnan
Scientific Committee Chairperson	: Dr. Chitra G
Treasurer	: Dr. Soumya Thankappan




RETIREMENT

- **Prof. Dr. Abdul Gafoor** retired from active service as Professor and Head of PMR, GMC Trivandrum, after an illustrious career that spanned decades. He has taught and mentored PMR post-graduates all over the state.

NOTABLE ACTIVITIES

- **Prof. Dr. U. Nandakumar** is an active columnist in the media and has contributed to multiple newspapers, where he writes about PMR and creates awareness of PMR.

- For the first time in the history of haemophilia care in Kerala, **Dr. Nittu Devassy** of Amrita hospital pioneered and delivered Yttrium for radio-synoviorthesis

- **Dr. Roy R. Chandran**, Associate professor, PMR dept., GMC, Kozhikode
  - Article in Malayalam Manorama — AMITHA VANNAM {Obesity and Overweight}
  - Malayalam Manorama Aarogyam article May Edition – Exercises for persons over 50 yrs of age
  - May Cardiological Society of India Kerala chapter –Fitness conclave
  - Presented topic on prevention of injuries during fitness training
  - May 15 - Blood donation IMA - Award for the 30th time donor achievement
  - May 22 - IMA Trivandrum Aarogyam Aanandam programme- Presented topic – exercises during Covid
- **Dr. Sherwin Sherif**, GMC Kozhikode PMR, is currently the Team doctor for the National Football team at Qatar.
- **New postings**
  - **Dr. Mildrin** joined the health services.
  - **Dr. Sunand** has joined Wayanad PHC as a specialist doctor.
  - **Dr. Harsha P.S**., has joined Institute of Sports as Doctor in Lead position in Laxmibai National College for Physical Education (Sports Authority of India)

- **Dr. Mohammed Rashid**, doctor in lead position in Bangalore, National College of Physical Education (Sports Authority of India)
- **Dr. T.K. Vasudevan** joined as Professor and HOD on 1 March 2021at Sri Narayana Institute of Medical Sciences(SNIMS), Chalakka, North Paravur, Kochi. Senior Resident, Dr Soumya Thankappan presented talks on Rotator cuff disease, Pathological gait, Covid treatment & rehabilitation. On April 7th, Dr Vasudevan conducted a training session for interns on case taking documentation. In this succinct session everything from history taking to examination to documentation, communication skills were demonstrated.

- **Dr. Bineesh Balakrishnan**, Assistant Professor in the same institute (SNIMS), presented a talk- 'Introduction to PMR' in the Clinical Club Meeting (meeting of faculties from different departments of SNIMS). He also gave a talk on 'Sports Medicine', on an online platform, which was attended by other Physiatrists working in the private sector. He conducted an OSCE(Objective Structured Clinical Examination) session on 'Prostheses & Orthoses' for the DNB students (Orthopaedics) on behalf of Cochin Orthopaedic Society.

- **Dr Santosh Raghavan** reports
  -Recently the stroke clinic of our institution has won an award by **WSO,Geneva** . It was a combined effort of departments of Neurosurgery, Internal Medicine, P.M&R, Nephrology under the leadership of the department of Neurology .

മെഡിക്കൽ കോളജിലെ പക്ഷാഘാത ചികിത്സാപദ്ധതിക്ക് അംഗീകാരം

അമ്പലപ്പുഴ • മെഡിക്കൽ കോളജ് ആശുപത്രിയിൽ 2 വർഷമായി പ്രവർത്തിക്കുന്ന പക്ഷാഘാത ചികിത്സാ പദ്ധതി ലോക പക്ഷാഘാത സംഘടനയുടെ അവാർഡിന് അർഹമായി.

ന്യൂറോളജി വിഭാഗത്തിന്റെ നേതൃത്വത്തിൽ വകുപ്പ് തലവൻ മാരായ ഡോ.സി.വി. ഷാജി (ന്യൂറോളജി), ഡോ. ബിജു ഭദ്രൻ (ന്യൂറോ സർജറി), ഡോ. ബി. പത്മകുമാർ (ജനറൽ മെഡിസിൻ), ഡോ.ജോസി വർഗീസ് (റേഡിയോ ഡയഗ്നോസിസ്), ഡോ.സന്തോഷ് കെ. രാഘവൻ (ഫിസിക്കൽ മെഡിസിൻ ആൻഡ് റീഹാബിലിറ്റേഷൻ), ഡോ.എസ്. ഗോമതി (വൃക്കരോഗ വിഭാഗം) എന്നിവർ ചേർന്ന് നടത്തിയ ചികിത്സാ പദ്ധതിയാണിത്.

പക്ഷാഘാതം ഭേദമാക്കാനുള്ള വേറിട്ട ഈ ചികിത്സയിൽ തലച്ചോറിന്റെ രക്ത ഓട്ടത്തിന്റെ തടസ്സം നീക്കുന്നതു വഴി മണിക്കൂറിനുള്ളിൽ രോഗി പൂർണ രോഗവിമുക്തനാകും.

പക്ഷാഘാത രോഗ ശുശ്രൂഷയിൽ പ്രത്യേകം പരിശീലനം നേടിയ ഫെഡ് നഴ്സുമാരായ സുമ, മേഴ്സി, മധു എന്നിവർ ഇവിടെയുണ്ട് . അത്യാഹിത വിഭാഗം ചികിത്സ, സ്കാനിംഗ്,തീവ്രപരിചരണം, രോഗമോചനം, തുടർ ചികിത്സ എന്നീ ഘടകങ്ങൾ ഒരു കുടക്കീഴിൽ സമന്വയിപ്പിച്ചതിനാണ് മെഡിക്കൽ കോളജിന് സ്വർണ മെഡൽ ലഭിച്ചത്.

അടിയന്തിര പക്ഷാഘാത ചികിത്സയുടെ സമയപരിധി നാലര മണിക്കൂറിൽ നിന്ന് ഒരു മണിക്കൂറായി കുറയ്ക്കാനും സാധിച്ചു. 45,000 രൂപ വരുന്ന ചികിത്സ തികച്ചും സൗജന്യമാണ്. സംസ്ഥാന സർക്കാർ അഞ്ച് കോടിയുടെ കോംപ്രിഹൻസീവ് സ്ട്രോക്ക് യൂണിറ്റ് നിർമാണവും ആശുപത്രിയിൽ തുടങ്ങി.

Dr. Nitha J, had done an awareness talk on Rehabilitation in Parkinson‘s disease, as part of an educational video by SCTIMST on World Parkinson‘s day 11.04.2021

PMR Pain Clinic
Dr. Noufal Ali is organizing a PMR Pain Clinic on every Sunday at Meitra Hospital Kozhikode.

Kerala Journal of Physical Medicine and Rehabilitation: The current rule set

Dr. Ravi Sankaran, Associate Professor, Dept of PMR, Amrita Institute of Medical Sciences

This is a small introduction to the KJPMR and its board positions. The purpose of the journal is to improve the quality of PMR in Kerala. The journal panels are per NMC competencies. The Clinician section accepts topics like: Case reports, Cold call, Scales I use, Invited Author. The Lifelong learner section accepts topics like: Medical Education, quizzes, Things patients taught me, book review. Often this section handles itself. The Communicator section accepts topics like: historical narratives, members in action. The Leader and team member section accepts topics like: what have I done to improve (theme related), leadership articles. The Professional section accepts topics like: leadership, economics, political issues, Survey results. Current holder is Dr. Ravi Sankaran. The Editor ensures each coordinator gets their job done in a timely fashion. If a coordinator can‘t do their task and doesn‘t get a replacement in a timely fashion, this person is responsible for filling their duty and getting a replacement. The current holder will be performing until 2025 in the latest.

Panel coordinators main duty is to get authors. If there are no submission for the upcoming issue they will write the article, proofread and run through grammarly.com before sending to Editor-in-chief. Pre-requisites: To do this requires access to and basic knowledge of Microsoft word/ publisher, be willing to send reminders and updates. The current vacancies to fill are: Professional, Leader and team member

Rules

As decided by the current president, the same with treasurer and secretary are automatically board members. It is their duty to fund the journal, so they are absolved of other journal work when actively holding such positions. When (and if) the positions of Secretary and President change hands, if the current holders are not as such engaged, they will also gain responsibilities in panels. If any of the others newly inducted take up Chapter key roles (President, Treasurer, or Secretary) they will be exempted from panel work. This ensures every domain is covered regardless of who holds a position on the chapter committee. If a board member is appointed but later cannot perform their role, they will choose a replacement and inform the editor-in-chief. This information will be provided 45 days before the next release. The bottom line is, anyone holding a position in the board will carry their own weight. With such a system in place, when the Editor changes suitable replacements will be available.

KJPMR Board Members

Quiz

1. A diarthrodial joint is composed of....

   (i) The ends of bones (ii) Synovium (iii) Cartilage (iv) Joint capsule

   (a) I, ii (b) i, iv (c) i, ii, iv (d) i, ii, iii, iv

2. The most common joint disorder worldwide is?

(a) Rheumatoid arthritis (b) Psoriatic arthritis (c) Osteoarthritis (d) Crystal arthropathy

3. The most important risk factor for knee osteoarthritis is?

   (a) Obesity (b) Aging (c) Previous trauma (d) Female sex

4. In knee osteoarthritis, knee pain predominantly during usage indicates?

   (a) Inflammation (b) Intraosseous pressure (c) Faulty mechanical loading or enthesopathy (d) All of the above (e) None of the above

5. The____major symptoms &_____indicative signs are the central features of knee osteoarthritis.

   (a) 3, 3 (b) 3, 4 (c) 4, 5 (d) 5, 6

6. The 3 main factors pointing to a poor prognosis in knee OA include all except..?

   (i) Valgus deformity (ii) Male sex (iii) Female sex (iv) Varus deformity (v) Early onset

   (a) ii (b) i, ii (c) iii, iv (d) iv, v

7. The most important risk factor for developing hip osteoarthritis is. ?

   (a) Aging (b) Female sex (c) Abnormal joint morphology leading to abnormal loading (d) Genetic risk factors

8. The most commonly affected joint in hand osteoarthritis is ?

   (a) PIP (b) MCP (c) a & b (d) DIP

9. Vasculitis in Rheumatoid Arthritis can present in____different clinical ways.

   (a) 4 (b) 5 (c) 8 (d) 3

10. The peak age of onset in Ankylosing Spondylitis is..?

    (a) Between 40 & 50 years (b) Between 50 & 60 years (c) Between 20 & 30 years

    (d) Between 15 & 20 years

Key

1.(d)

A diarthrodial or synovial joint is composed of the ends of bones, synovium, cartilage, and a joint capsule, which encloses the joint. The joint capsule is surrounded by ligaments and other periarticular structures such as bursae, tendons, and muscles. The joint capsule contains a joint cavity, which is filled with a lubricating synovial fluid. Diarthrodial joints allow a large range of movement and are the most common joints in the extremities, such as *the shoulders, elbows, wrists, hips, knees, and ankles*.

2. (c)

OA, the most common joint disorder worldwide, can lead to disability and decreased quality of life. It is characterized by *cartilage degradation, subchondral bone alteration, meniscal degeneration, synovial inflammatory response, and accompanying periarticular bone response*. It often *affects knees, hips, hands, spine, and feet*. OA is known to be caused by multiple factors, including genetic predisposition, joint integrity, mechanical forces, and cellular biochemical processes. The prevalence of knee OA is estimated to be 12.5% worldwide. It is more common in women and older adults, and its prevalence increases with age.

3. (b)

Aging is the most important risk factor. The prevalence of symptomatic knee OA continues to increase with age. Various aging-related processes contribute to the development of OA, *including increased susceptibility to cell death, leading to the loss of chondrocytes, increased destruction of joint tissue, and defective repair of damaged matrix. Female gender* is also a risk factor. Women have a greater prevalence of knee OA compared with men. Women with radiographic knee OA are more likely to have symptomatic OA than are men.

Being overweight, the most common modifiable risk factor, promotes the development and progression of knee OA. It does so not only by increasing mechanical load but also by factors related to adipose tissues. The lifetime risk for knee OA will increases as body mass index (BMI) increases. *A person with a BMI greater than 30 has a threefold greater risk of developing early OA than a person of normal weight.* By contrast, weight loss can reduce the risk of developing knee OA. Moreover, *moderate exercise can be a protective factor.* Any factor that alters proper joint biomechanics can trigger the onset and accelerate the progression of the degenerative process. Subsequently, clinical symptoms may occur. *Any injuries affecting the knee ligaments* may also decrease joint stability and can contribute to joint degeneration.

Muscle weakness is an independent risk factor for knee OA and can also participate in the progression. Quadriceps weakness can be due not only to disuse but also arthrogenous inhibition of muscle contraction. The quadriceps can work as shock absorbers and stabilizers and hence protect the joint surfaces during loading and movement. For this reason quadriceps weakness should be specifically managed in the course of a rehabilitation program.

4. (c)

Pain at and around the knee joint is the most common chief complaint. It usually occurs after activity and is relieved by rest. Other symptoms include stiffness, joint swelling, deformity, crepitus, and impaired function. The different pain characteristics always indicate different underlying mechanisms. Pain predominantly during usage indicates faulty mechanical loading or enthesopathy; *pain at rest* indicates *inflammation*; and *pain at night* indicates *increased intraosseous pressure*. Persisting rest and night pain occurring in advanced OA is an indicator of severe damage. Disability due to knee OA usually results from pain, reduction of knee joint ROM, and poor control of movement.

5. (a)

The three major symptoms (*persistent knee pain, limited morning stiffness, and reduced function*) and three indicative signs (*crepitus, restricted movement, and bony enlargement*) are the central features of knee OA. Knee pain is, in fact, a consequence of structural deterioration and also contributes to its

progression. Persistent knee pain over 1 year can predict accelerated loss of cartilage, which increases the risk of progressing radiographic changes.

6. (b)

Knee OA progresses slowly. However, if untreated, it can eventually cause various degrees of disability, usually with difficulty in rising from a seated position, walking, climbing stairs, and performing housework. *Varus deformity of the knee joints, early onset, and female gender were the three main factors pointing to a poor prognosis*.

7. (c)

Hip OA is less common than knee OA. Hip joint symptoms do not obviously match radiographic findings. *The risk factors for hip OA can be divided into joint level and person level*. The former includes hip joint morphology, muscle function, and joint shape. The latter includes age, sex, weight, genetics, ethnicity, and occupation. *The most important risk factor for developing hip OA is the presence of abnormal hip joint morphology—for example, following trauma or congenital dislocation of the hip, leading to abnormal loading on the hip joint. As in the case of other joints, hip OA is strongly related to increasing age. For symptomatic hip OA, the difference between men and women is not significant*. The association of gender with OA appears weaker at the hip joint compared with other joints, where female gender is a vital risk factor.

8. (d)

Hand OA, *which commonly occurs in women*, is a highly prevalent joint disease with often slow progression. *The most frequently affected joint is the distal interphalangeal (DIP) joint*. The prevalence of hand OA is estimated to be higher than that reported for hip and knee OA. According to the various definitions of hand OA, the prevalence of radiographic hand OA ranges from 21% to 92% but that of symptomatic hand OA ranges from only 3% to 16%. *The most common symptom of hand OA is pain in the affected joint*. Other symptoms include stiffness, decreased hand muscle strength, and restricted hand movement. *The indicative signs include nodes at the DIP joints and proximal interphalangeal (PIP) joints, called Heberden and Bouchard nodes, respectively, and deformities such as squaring of thumb base. The presence of nodes is a hallmark of hand OA*. Involvement of first carpometacarpal joint is also common and often causes significant joint pain, impairment of hand function, and reduced grip strength.

9. (b)

Vasculitis is a serious condition; it can present in five different clinical ways: *distal arteritis, cutaneous ulceration, palpable purpura, arteritis of the viscera, and peripheral neuropathy (mononeuritis multiplex or distal sensory neuropathy).*

10. (c)

Seronegative spondyloarthropathy is a type of chronic inflammatory arthritis involving the axial structures; it is manifested by chronic back pain and progressive stiffness of the spine. It can also involve the shoulders, hips, and other peripheral joints. Its manifestations include AS, reactive arthritis, arthropathy of inflammatory bowel disease, psoriatic arthritis, undifferentiated spondyloarthropathy, and juvenile-onset AS. In addition to inflammation of the spine (including the sacroiliac joint), this disease is characterized by the absence of an RF, the tendency for familial aggregation, an association with the HLA-B27, inflammation around the entheses (the site of tendon insertion into bone), uveitis, urethritis, and psoriatic skin lesion. AS is the prototypic form and is frequently encountered by physiatrists. *The peak age of onset is between 20 and 30 years.*

Quiz 2

1. Patients with AS may experience arthritis outside of the spine, and peripheral arthritis occurs in approximately 35% to 50% of patients with AS over the course of the disease. The most frequently involved joint from among the below options is?

   (a) Hip (b) Knee (c) Wrist (d) Shoulder

2. Enthesitis occurs in approximately 40% to 70% of patients with AS at some time during the course of the disease. The most common location of enthesitis in patients with AS is…?

   (a) Calcaneal attachments of the plantar fascia (b) Shoulders (c) Calcaneal attachments of the Achilles tendon (d) Superior iliac crest

3. Moll & Wright have described__ clinical patterns of Psoriatic arthritis...?

   (a) 5 (b) 6 (c) 4 (d) 3

4. The most typical radiologic finding in Psoriatic arthritis is...?

   (a) Lysis of the terminal phalanges (b) The coexistence of erosive changes and new bone formation in the DIP joints (c) The presence of both joint lysis and ankylosis (d) –Pencil in cup‖ appearance

5. _____is the most common and among the first manifestations of SLE?

   (a) Cognitive impairment (b) Joint pain (c) Seizures (d) Stroke

6. The most commonly affected joint in septic arthritis is..?

   (a) Knee (b) Hip (c) Shoulder (d) DIP of hands

7. Because septic arthritis is rapidly destructive, broad-spectrum antibiotics are usually warranted until culture data are available._____is the initial antibiotic?

   (a) Amoxycillin (b) Amoxycillin + Clavulanic Acid (c) Gentamicin

   (d) Vancomycin

8. In hip & knee OA, the use of a cane on the less affected side can relive joint force up to_____?

   (a) 70% (b) 50% (c) 20% (d) 30%

9. _____ exercise is not recommended in the rehabilitation of acute inflammatory stage of Rheumatoid arthritis?

   (a) Isometric (b) Isotonic (c) AROM (d) AAROM

10. Which Physiotherapeutic modality was shown to be more effective than intra-articular Hyaluronic acid injections in Osteoarthritis of the knee (according to a study in Archives of PMR) ?

    (a) Ultrasound (b) IFCT (c) ESWT (d)TENS

Key

1. (d)

   Patients with AS may experience arthritis outside of the spine, and peripheral arthritis occurs in approximately 35% to 50% of patients with AS over the course of the disease. The *most commonly affected joints, in order of frequency*, are the *shoulders, hips, and knees*. Involvement of the ankles, sternoclavicular joints, and temporomandibular joints is also reported. Hip *involvement is present in 25% to 35%* of patients with AS and is associated with a high degree of physical disability and a *poor prognosis*.

2. (c)

   The most common location of enthesitis in patients with AS is *the calcaneal attachments of the Achilles tendon*. Other locations include the calcaneal attachments of the plantar fascia, shoulders, costochondral junctions, sternoclavicular and manubriosternal joints, and superior iliac crest. Patients with AS may have extra-articular comorbidities including anterior uveitis, inflammatory bowel disease, psoriasis skin lesions, aortic regurgitation, cardiac conduction disturbance, restrictive lung disease, apical pulmonary fibrosis, immunoglobulin nephropathy, or renal amyloidosis. Among these conditions, *anterior uveitis (or iritis) is the most common, occurring in 25% to 40% of patients with AS.*

3. (a)

   Moll and Wright have described *five clinical patterns* of psoriatic arthritis: (1) asymmetric oligoarthritis, (2) polyarthritis, (3) predominant DIP joint involvement, (4) destructive arthritis (arthritis mutilans), and (5) predominant spondyloarthritis. *Polyarthritis is the most common (63%), followed by oligoarthritis (13%),* and predominant DIP involvement (<5%). Although spinal involvement is found in 40% to 70% of patients with psoriatic arthritis, spinal involvement alone occurs in only 2% to 4% of patients with psoriatic arthritis. Some patients present with more than one pattern, and the classification is not fixed. The pattern of disease may fluctuate. Other musculoskeletal features include dactylitis (sausage digit, resulting from inflammation of the soft tissue and joints), enthesitis, tenosynovitis, nail lesions (pits and onycholysis), and pitting edema in the hands or feet.

4. (b)

   The most typical radiologic finding is the coexistence of erosive changes and new bone formation in the DIP joints. Other changes include fluffy periostitis with new bone formation at the site of enthesitis, lysis of the terminal phalanges, gross destruction of isolated joints, ‒pencil in cup‖ appearance, and the presence of both joint lysis and ankylosis. *MRI may be more sensitive than conventional radiography in detecting articular, periarticular, and soft tissue inflammation.*

5. (b)

   Joint pain is the most common and among the first manifestations of SLE. *It typically presents as symmetric polyarthritis involving the MCP, PIP, and knee joints. Any young female patient with arthritis should be evaluated for a possible diagnosis of SLE*. Unlike the case in RA, in SLE the joints are rarely erosive or deforming. *SLE may present with swan-neck deformities of the hand resulting from recurrent synovitis and inflammation of the joint capsule, tendons, and ligaments*. *Unlike RA, these deformities, known as Jaccoud arthropathy, are usually reducible*.

6. (a)

Septic arthritis, also known as infectious arthritis, is a bacterial, viral, mycobacterial, or fungal joint infection. Of these, *bacterial infection* is the most common. It usually occurs in older adults and very young children. Bacterial arthritis can lead to severe morbidity and mortality. Delayed or inadequate treatment can cause irreversible joint destruction, severe disability, and even death. *The most robust risk factor is preexisting joint disease. Other factors are RA, OA, gout, recent trauma, prior joint surgery, SLE, and receipt of TNF inhibitor therapy. Of these, RA is the most common factor and is related to worse outcomes*. Any conditions causing loss of skin integrity and those associated with compromised immunity present a risk for septic arthritis. *The most common symptom is acute onset of joint pain, erythema, heat, and restricted joint mobility. Fever is detected in only approximately 40% to 60% of patients with septic arthritis, indicating that an elevated body temperature is not a prerequisite for diagnosis*. Septic arthritis usually affects a single joint; in only 20% of cases, two or more joints are affected. The knee is the most commonly affected joint, followed by the shoulder, wrist, hip, and ankle, whereas *the hip joint is the most commonly affected joint in children. Infections of axial joints, such as the sternoclavicular or sacroiliac joints, are more common in patients with a history of intravenous drug abuse.*

7. (d)

   Vancomycin is the initial antibiotic. For *immunocompromised patients* or intravenous drug abusers, *vancomycin plus a third generation cephalosporin* should be administered. *Treatment is often continued up to 6 weeks, with intravenous administration of the antibiotic for the first 2 weeks and then a switch to oral administration.*

   Aggressive rehabilitation is crucial in preventing muscle wasting and joint contractures. Patients should be mobilized once their pain has decreased.

8. (c)
9. (b)

   For prevention of joint and soft tissue contracture, actively inflamed joints should be moved gently through the possible range by the patient (*active ROM*) or with assistance from another person (*active-assisted ROM*). *Three repetitions for each joint once or twice daily are suggested*. As joint inflammation subsides, the range may be increased gradually to the full range, possibly with assistance. *A PNF technique could be applied early on for selected muscle groups*. Passive stretching is usually not performed in an inflammatory joint; if performed, it should be done with extreme caution because it may worsen or prolong the inflammatory process or lead to subluxation or rupture of a joint. *For minimizing muscle atrophy, isometric exercise at submaximal effort is recommended in the inflammatory condition.* At first a few nonresistive repetitions should be performed with a gradual increase in repetition and resistance. *Isotonic exercise is not recommended in the acute inflammatory stage.*

10. (d)

    The purposes of electrical therapy are pain control and muscle stimulation. These modalities are commonly used in the treatment of rheumatic diseases. Electrical therapy has been shown to have a clinically beneficial effect on grip strength for patients who have RA with hand atrophy. Another study showed that TENS may be more effective than the intra-articular injection of HA in patients with OA of the knee.