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CONTEMPORANEOUS STATE OF ARTHRITIS: CONVENIENT THERAPIES, IDENTIFICATION OF ITS TYPES AND MANAGEMENT

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ABSTRACT

"Arthritis" is a combinatorial word that comes from a combination of Latin and Greek. "Arthron" means joint in Greek, and "itis" means inflammation in Latin. As a result, arthritis is typically thought of as a condition brought on by inflamed joints; however, it is a group of medical issues that are collectively referred to as "Arthritis." Any condition affecting the joints is commonly referred to as arthritis. Joint stiffness and discomfort are typical symptoms. Redness, warmth, swelling, and a reduction in the affected joints' range of motion are possible additional symptoms. Other organs may also be impacted by some forms of arthritis. Onset may occur gradually or abruptly. A few typical kinds Psoriatic arthritis, juvenile idiopathic arthritis, osteoarthritis, gout, ankylosing spondylitis Thumb arthritis, septic arthritis, rheumatoid arthritis, and reactive arthritis Among the risk factors for arthritis are: Family history: If your parents or siblings have arthritis, you may be at a higher risk of getting the condition yourself. Age: As people age, their risk of developing various forms of arthritis, such as gout, rheumatoid arthritis, and osteoarthritis, rises. Your sex: Gout is a kind of arthritis that primarily affects males, although rheumatoid arthritis is more common in women than in men. Prior joint injury: Individuals who have previously suffered a joint injury, possibly during athletic activities, are at an increased risk of developing arthritis in that joint in the future. Obesity: Being overweight strains your joints, especially your spine, hips, and knees. Arthritis is more likely to occur in obese people. Arthritis affects 9.36% of Indian adults 45 and older. The female prevalence was 11.03 per cent, while the male prevalence was 7.49 per cent. Women are more likely than men to develop arthritis, with an odds ratio of 1.59 (95 per cent CI: 1.50, 1.69). Prevention and treatment of arthritis may involve rest, physical therapy or occupational therapy, hot or cold compresses, joint protection, exercise, medication, and occasionally surgery to repair damaged joints. We evaluate the current status, potential treatments, and underlying causes of arthritis in this article.

KEYWORDS: Arthritis, Etiology, Pathophysiology, Epidemiology Diagnosis, Treatment.

INTRODUCTION

One or more swollen and painful joints are signs of arthritis. The primary signs of arthritis are stiffness and joint pain, which usually worsen as people age. Some causes of arthritis include: OA, one of the most prevalent types of arthritis, which is brought on by normal wear and tear. A joint injury or infection may make this natural decomposition of cartilage tissue worse. The cartilage in your joints is a flexible yet tough connective tissue. Acute or chronic joint inflammation is referred to as arthritis. A wide range of symptoms, such as pain, stiffness, reduced range of motion, and joint abnormalities, can be attributed to arthritis. Arthritis comes in various forms, and each one requires a distinct approach to treatment. The word "arthritis" comes from the Greek "disease of the joints." Acute or chronic joint inflammation that frequently coexists with pain and structural damage is its definition. Arthralgia, which is pain that is localized to a joint, independent of its cause (which may or may not be attributable to joint inflammation), is not the same as arthritis. Both Neanderthals and ancient Egyptians suffered from arthritis, but Dr. John K. Spencer did not use the name "osteoarthritis" until 1886. There are about 100 forms of arthritis known to exist, with osteoarthritis and degenerative arthritis—non-inflammatory arthritis—being the most prevalent. Inflammation can be brought on by autoimmune processes (such as rheumatoid arthritis, psoriatic arthritis, ankylosing spondylitis, etc.), infections (such as Lyme's arthritis and septic arthritis), or crystal deposition-induced inflammation (such as gout, pseudogout, and basic calcium phosphate disease). Other autoimmune connective tissue disorders such as systemic lupus erythematosus, Sjogren syndrome, scleroderma, myositis, inflammatory bowel disease, celiac disease, etc., can also coexist with inflammatory arthritis.[1,2]

ARTHRITIS CLASSIFICATION

Osteoarthritis (OA): This condition causes cartilage to gradually deteriorate because it loses its elasticity and becomes stiff. It can no longer function as a suitable shock absorber as a result. Ligaments experience stretching as a result of slow erosion, which starts the pain. The result is that the bones begin to rub against one another, causing further pain and suffering. The symptoms start slowly but get worse over time, leading to inflammation and more joint damage. Both during and after the joint movement, patients experience pain. The joints hurt when you try to move quickly, which commonly happens when you wake up in the morning. Generally speaking, the onset of stiffness is a typical sign. As people age, the condition worsens. *Gout:* It is also classified as a type of arthritis that affects the bone joints, especially the distant ones like the toes. Men are more likely to have the disease, but as women reach menopause, the prevalence levels out, indicating that estrogen plays a significant role in preventing it. The inflammatory gouty condition is a painful and incapacitating disease that is brought on by the deposition of uric acid/urate crystals in the joints, such as the toes, fingers, or ankles. Faulty purine metabolism leading to hyperuricemia or mal-excretion of uric acid/urate because of impaired kidney filtration are the main causes of the rise in serum uric acid levels, which causes their deposit while causing excruciating pain suffering and inflammation. *RA: Rheumatoid arthritis* is a well-known inflammatory condition. In RA, the autoimmune onslaught causes inflammation in the synovium, which later results in stiffness, oedema, discomfort, and deformity. Women between the ages of 40 and 60 are three times more likely than males to suffer from RA, though children are rarely the victims. Joints such as the arms, fingers, wrists, knees, or legs are impacted. Inflammation causes the joints to swell, which results in stiffness, particularly in the morning. Patients experience discomfort that appears red or swollen where it is touched. Patients frequently notice weight loss and fatigue. In addition to affecting the wrists, ankles, and feet, the disease can also affect the elbows, knees, hips, necks, and shoulders, moving from minor to larger joints at the same time. Another name for *ankylosing spondylitis (AS)* is an inflammatory autoimmune disease that affects the joints in the spine or the space between the spine and the pelvis. The

excruciating pain that gets worse over time is caused by the inflammatory joints. Because of the bone fusion, the spine becomes rigid during that process. Although the precise cause has not been determined, speculation is that it is genetic. It typically begins between the ages of 20 and 40 and affects more men than women. When physical activity begins during the day, the pain and stiffness that are intense in the morning or at night start to lessen. The condition first affects the sacroiliac joints, which are the spine and pelvis, and then spreads to other areas. About 1.5 million people in the US alone suffer from *lupus arthritis (LA)*, a systemic autoimmune disease. About 35% of those with lupus have LA, and nearly 90% of them experience joint and muscle discomfort. The illness causes morning stiffness along with joint discomfort and swelling. Fluid might occasionally build up at the swollen joints. In addition to causing severe harm, LA causes discomfort and deformity but does not harm the neck or spine. The majority of the affected areas are distant parts of the body, such as the hands, fingers, wrists, knees, feet, toes, and elbows. LA has a symmetrical impact; for instance, it attacks the same joints on both sides of the body. Children are the patients with juvenile *arthritis (JA)*. The symptoms include weight loss, rashes on the arms and legs, reduced appetite, and sporadic nighttime fever. Often, the patients are limping and have knee, finger, or wrist pain. Because of the swelling, the joints seem bigger. Among the discomforts include hip or neck pain and stiffness. It is also observed that anaemia develops frequently. *Infectious arthritis (IA)*: IA is brought on by a bacterial, fungal, or viral infection within the synovium. The virus affects the joints after spreading through the bloodstream. Patients who already have arthritis are more likely to develop it, which exacerbates their symptoms. It could be the reason why people with arthritis frequently develop infections, which makes their condition worse. The symptoms, which frequently begin with an accident, include joint pain, oedema, and inflammation followed by a persistent fever. Attack targets include the knee, ankle, shoulder, wrist, elbow, finger, and so forth. However, IA only affects one joint. *Fibromyalgia:* The illness is characterized as a condition that arises from exhaustion and physical pain. Many people think that compared to normal perception, it exaggerates the pain sensation. The disorder is frequently brought on by physical trauma, psychological stress, or infection. According to the survey, women suffer more harm than males. A sizable portion of fibromyalgia patients also exhibit anxiety, sadness, irritable bowel syndrome, and tension headaches.

Since the illness seems to run in families, there is a fair chance that genetic abnormalities are a contributing factor. People with RA or post-traumatic stress disorder frequently contribute to the development of the illness. *Psoriatic arthritis (PA)*: Psoriasis is a common inflammatory skin condition that affects 1-3 per cent of white Europeans. About 15% of psoriasis patients have PA that expresses HLA B27, a Human Leukocyte Antigen (HLA). The B locus in the Major Histocompatibility Complex (MHC) on chromosome 6 encodes the Class-I surface antigen HLA-B27, which presents antigenic peptide to T lymphocytes. The AS is closely related to HLA-B27 as well. Research indicates that psoriasis and PA are notably prevalent in people with Crohn's disease and inflammatory bowel illness. The pathophysiology of both PA and psoriasis involves genetic predisposition and both innate and adaptive immunity More than 100 disorders involving inflammation and injury to the joints, surrounding tissues, and other connective tissues are categorized as arthritis. Three prevalent forms of arthritis are psoriatic, rheumatoid, and osteoarthritis.[3-14]

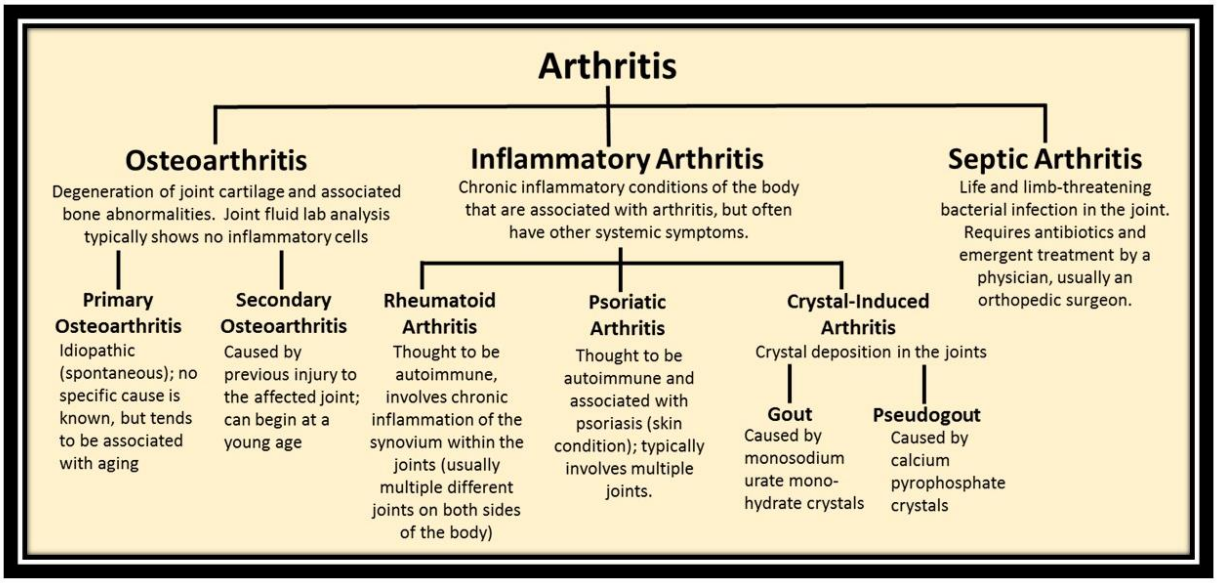

Fig. 1: Several Major Forms of Arthritis.[3]

PATHOPHYSIOLOGY

A degenerative cascade of gradual cartilage loss that results in bone deterioration is the hallmark of osteoarthritis. Osteophytes, subchondral plate thickening, and subchondral cysts are characteristic findings. Joint collage is broken down by proteolytic enzymes like matrix metalloproteinases, serine proteases, and cysteine proteinases that are triggered by interleukin-6, monokines, interferon-induced protein-10, and macrophage chemotactic protein. The cartilaginous matrix becomes thinner and eventually disintegrates as the surrounding articular cartilage calcifies. Additionally, a decline in chondrocyte function is linked to ageing, increasing the risk of osteoarthritic degeneration. Generally speaking, rheumatoid arthritis symptoms are worse than those of osteoarthritis. An autoimmune reaction to an external trigger results in rheumatoid arthritis, a persistent, systemic inflammatory disease. Endothelial cell activation and synovial cell hyperplasia occur before cartilage and eventually bone deterioration. After exposure to an antigenic pathogen, the disease develops as a result of the abnormal synthesis of inflammatory mediators, including interleukins 1, 6, and 8, tumour necrosis alpha, and others. Gout's monosodium urate salts form needle-shaped crystals when they precipitate. Cooler bodily areas and acidic environments are more likely to experience this crystallization. The usual acute flare-up of gouty arthritis is caused by an inflammatory response mediated by IL-1 when these accumulated intraarticular uric acid crystals become unstable. In pseudogout, the process is different because calcium pyrophosphate dihydrate is created when chondrocyte-derived inorganic pyrophosphate interacts with calcium. Joint areas that are prone to osteoarthritic alterations are where this crystal is deposited. Osteophytes, subchondral cysts, and bone and cartilage fragmentation are all examples of pseudoout crystal injury. Hyperparathyroidism, hemochromatosis, and hypomagnesemia are metabolic diseases that increase the risk of calcium pyrophosphate accumulation. An inflammatory reaction to a monobacterial infection is usually the cause of septic arthritis. Cytokines, chemokines, and proteases are released when bacteria enter the synovial fluid, breaking down cartilage and causing the synovial membrane to swell. Another harmful function of bacterial toxins is to damage the joint space itself. The most prevalent pathogen in adults is Staphylococcus aureus, while streptococci types are also prevalent. Gram-negative bacterial infections are more frequently observed in the elderly and very young, as well as in those who have had trauma, intravenous drug use, or immunosuppression.[15-18]

AETIOLOGY

Different types of arthritis have different etiologies. The main risk factors for osteoarthritis include obesity, joint injuries, female sex, and ageing. Mutations in the genes encoding types II, IV, V, and VI collagens are among the genetic factors that have been described. In contrast, rheumatoid arthritis (RA) is a systemic inflammatory disease that is autoimmune. Inflammation in RA is caused by the interaction of multiple environmental variables (smoking) and genetic factors (HLADRB1 and others), which activate and malfunction the immune system. Prolonged hyperuricemia in gout causes uric acid buildup in the joints, which in turn causes inflammation. Less than 10% of gout is caused by hyperuricemia, which can be caused by several genetic abnormalities. Most gout sufferers are under-excretors, meaning they are unable to eliminate all of the uric acid generated by their body's endogenous or exogenous purine metabolism. Other risk factors for hyperuricemia and gout include male sex, ageing, chronic renal disease, alcoholism, and certain medications like diuretics. Patients with pre-existing risk factors, such as immunodeficiency, ageing, diabetes mellitus, prosthetic joints, rheumatoid arthritis, and intravenous drug misuse, are more susceptible to septic arthritis, an acute form of arthritis that is uncommon in the general population. One of the most prevalent clinical characteristics in individuals with systemic lupus erythematosus (SLE) is arthritis, which is also commonly observed in patients with other autoimmune disorders. Inflammatory bowel disease, psoriasis, celiac disease, Sjogren syndrome, systemic sclerosis, dermatomyositis, mixed connective tissue disease (MCTD), and other conditions are often linked to arthritis.[19-21]

EPIDEMIOLOGY

There are a lot of studies being done on the prevalence of different types of arthritis in the eastern and western hemispheres. There is currently no clear explanation for why environmental stimuli or genetic predisposition may be involved. According to research by Helmick et al., over 21% of adults in the US alone, or around 46.4 million people, have been diagnosed with arthritis. Additionally, according to data from 2008, 1.3 million Americans suffer from RA, which is a little lower than the 2.1 million estimated in 1995. According to the study, there are between 0.6 and 2.4 million AS sufferers, while 161,000 to 322,000 LA sufferers. The number for JA is approximately 294,000. Additionally, the study reveals that around 27 million Americans had clinical OA, up from 21 million in 1995. About 5.0 million people suffer from fibromyalgia, while approximately 3.0 million people have gout (up from 2.1 million in 1995). On imaging, more than one-third of Americans have arthritis, and as the population's average age rises, this percentage will inevitably rise. Osteoarthritis is the most common type of arthritis. Osteoarthritis of the knee affects 19% to 30% of persons over 45, hand osteoarthritis affects 27%, and hip osteoarthritis affects 27%. In their lifetime, osteoarthritis is predicted to affect 40% of men and 47% of women; if a person's body mass index is higher than 30, the incidence rises to 60%. The most prevalent inflammatory arthritis in the US is gout, which affects over 8 million people and has a prevalence of 3.9%, with over 9% of people over 60 having it. Gout affects almost 45 out of every 100,000 people. Notably, over the past few decades, there has been a more than twofold increase in the incidence and frequency of gout. Between 4% and 7% of adults have pseudogout, and more than half of those who have it have knee arthritis. About 1% of Caucasians have rheumatoid arthritis, and women are more likely than men to have it (lifetime risk of 3.6% for women vs. 1.7% for men). Early adulthood is usually when the disease first manifests, and in women over 65, the disease prevalence is 5%. The most common cause of septic arthritis is bacterial seeding of an already-arthritic joint through hematogenous dissemination, usually from a urinary tract or skin infection. The prevalence of septic arthritis is 0.7% in rheumatoid arthritis patients and 0.01% in the general population. In 1990, there were about 23.46 million people with OA in India; by 2019, that number had risen to 62.35 million. Between 1990 and 2019, the age-

standardized prevalence of OA rose from 4,895 (95% UI:4,420–5,447) per 100,000 people to 5313 (95% UI:4,799–5,898). From 1990 to 2019, age-standardized DALYs grew from 164 (95%UI:83–325) to 180 (95%UI:91–361) per 100,000 people, whereas DALYs owing to OA increased from 0.79 million (95%UI:0.40–1.55) to 2.12 million (95% UI:1.07–4.23). In India, OA accounted for 1.48% (95%UI:0.88–2.78) of all YLDs in 2019, making it the 20th most prevalent cause. In 1990, it was the 23rd most common cause (1.25% (95%UI:0.74–2.34)). The most prevalent type of OA was knee OA, which was followed by hand OA. For both OA and knee OA, the prevalence, incidence, and DALYs were consistently higher in women than in men.[22-31]

DIAGNOSIS

The diagnosis and severity rating of an arthritic condition might be aided by laboratory and radiographic assessment. ESR and CRP, two indicators of inflammation, are elevated in inflammatory arthritides. Chronic disease-related anaemia is prevalent. Non-articular pain, such as that caused by fibromyalgia, myofascial pain syndrome, neuropathy, tendinitis, and complex regional pain syndrome, must be distinguished from arthritis. This distinction can be made with the aid of a physical examination, laboratory testing, and radiographic imaging.

Before starting treatment, it's critical to obtain a definitive diagnosis because there are over 100 distinct forms of arthritis. Leukopenia and thrombocytopenia are observed in RA and SLE-associated arthritis, while leukocytosis is seen in septic arthritis, gout, pseudogout, and adult-onset Still disease. Patients with gout may have high serum uric acid, however, this should not be the only diagnostic criterion used. To help with diagnosis, serologies such as rheumatoid factor, anti-citrullinated peptide antibodies, anti-nuclear antibodies, and more specialized autoantibodies should be carried out when properly indicated. The first imaging modality will be plain radiography. Common symptoms of osteoarthritis include effusion, osteophytes, and narrowing of the joint space. In inflammatory arthritis, periarticular osteopenia appears on radiographs first, followed by joint space constriction, erosions, and secondary osteoarthritis. Erosive osteoarthritis is characterized by central "gull-wing" erosions, whereas RA is characterized by periarticular erosions. Seronegative spondyloarthritis, particularly psoriatic arthritis and ankylosing spondylitis, can exhibit entheseal calcifications. Early in axial spondyloarthropathies, X-rays are normal; but, as the condition progresses, they may reveal erosions and fusion of the sacroiliac joint and bamboo spine. Conversely, axial osteoarthritis manifests on X-ray as joint space narrowing, osteophytes, and disc bulges. As can be observed in the menisci, the wrist's triangular fibrocartilage, or the cartilages of the hands' second and third MCP joints, pseudogout displays the radiographic hallmarks of chondrocalcinosis, which is calcification of the cartilages. Early in the course of gout, X-rays are normal; but, as the condition progresses, they may reveal hard tophi, periarticular osteopenia, and the typical juxta-articular erosions, sometimes known as rat-bite erosions, with overhanging edges. More imaging may be considered if radiographs are not diagnostic. With a far higher sensitivity than X-rays, magnetic resonance imaging (MRI) can help determine whether synovitis, erosions, or sacroiliitis are present. Additionally, MRI can help assess malignancies and soft tissue or ligamentous injuries that are otherwise challenging to detect. Although a CT scan is typically used when an MRI is not possible, it can help identify bone abnormalities and detect chondrocalcinosis. An emerging technique called musculoskeletal ultrasound is very useful, particularly when evaluating peripheral arthritis. It can help determine whether synovitis, effusion, erosion, or structural defects like rotator cuff or meniscus teat are present or absent. It can also help with ultrasound-guided aspiration and injection. Because of its high sensitivity and low specificity, nuclear medicine joint scans are rarely recommended. However, they can help determine if inflammation is present or not, particularly when assessing infections in prosthetic joints. Another cutting-edge radiographic technique that is highly

accurate in aiding in the diagnosis of gout is the dual-energy CT scan. One of the most crucial diagnostics, particularly for the early diagnosis of arthritis, is the synovial fluid study. The synovial fluid will be subjected to Lyme's DNA PCR, bacterial/acid-fast bacilli/fungal cultures, cell counts and differentials, and crystal assessment under polarized light microscopy, as needed. Cell counts in inflammatory arthritis are often greater than 5,000 cells/mm3 and can reach 50,000 cells/mm3, but degenerative arthritis is typically linked to cell counts of less than 2,000 cells/mm3. Although this can also occur in the context of acute gout or pseudogout, a synovial fluid analysis with more than 90% neutrophils and/or 50,000 cells/mm3 cells will raise suspicions of septic arthritis. The needle-shaped crystals of gout are highly negatively birefringent. Rhomboid in shape, pseudogout crystals exhibit weekly positive birefringence. Basic calcium phosphate crystals require additional staining to demonstrate positive because they are invisible under polarized light microscopy. Although they are infrequently done, synovial biopsies may be considered, particularly in cases of monoarthritis where no further diagnosis has been made.[32-34]

SIGNIFICANT ARTHRITIS MANAGEMENT AND TREATMENT OPTIONS

Reducing pain and enhancing function should be the goals of osteoarthritic joint care. For the best care, pharmacological and non-pharmacological (or conservative) treatments are typically needed in combination. Specific exercises, physical therapy, bracing, acupuncture, and weight loss are examples of non-pharmacologic treatments. Oral and topical medicines are used in the pharmacologic treatment of osteoarthritis. Topical capsaicin, duloxetine, and oral and topical non-steroidal anti-inflammatory medicines (NSAIDs) are often used in pharmaceuticals. It is possible to inject corticosteroids straight into the joint. First-line treatment usually consists of topical NSAIDs, capsaicin, and other ointments; if these do not relieve symptoms or if the condition is more systemic, oral NSAIDs should be started. Duloxetine is helpful, particularly in treating osteoarthritis of the knees, and may be helpful for people who have medical contraindications to using NSAIDs. Injections of intra-articular corticosteroids may alleviate symptoms if pharmacological and non-pharmacological treatments are unsuccessful. Steer clear of opioids. In cases of refractory symptoms, surgery to replace the damaged joint or joints might be very beneficial. Limited function in the immediate postoperative period is not uncommon, and patients may develop post-operative problems. To maximize patient results, physical therapy is required after surgery. Early illness remission and halting radiographic progression are the main goals of pharmacological treatment for rheumatoid arthritis and seronegative spondyloarthropathies. Treatment with glucocorticoids and NSAIDs is less successful than the early use of biologics and disease-modifying anti-rheumatic medications (DMARDs). While the disease is still active, anti-inflammatories can be taken as an adjuvant to lessen inflammation. Symptom remission is followed by ongoing dose adjustments and regular symptom monitoring. Corticosteroids may be used to treat severe disease exacerbations. Flare-ups of gouty arthritis can be extremely painful and incapacitating. Anti-inflammatory drug use can offer significant relief and should ideally be started within 24 hours following an acute flare-up of gout. These consist of colchicine, NSAIDs such as indomethacin or high-dose naproxen, and oral corticosteroids. For patients with pauciarticular involvement, intra-articular corticosteroid injections may be helpful; however, if the patient is unable to take their meds orally, intramuscular or intravenous corticosteroids may be administered. Although they do not help treat an acute flare, medications that lower uric acid levels are advised for people who have recurring flares, chronic renal disease, nephrolithiasis, or tophi. The objective is to lower the intra-articular uric acid burden, which in turn lowers the blood uric acid burden, ultimately resulting in the alleviation of gout symptoms. Draining the afflicted joint and taking antibiotics are necessary for the treatment of septic arthritis. The joints of antibiotics are determined by the cultures and sensitivities of the joint fluid.[32,33,42-45,34-41]

DISCUSSION AND CONCLUSION

Our review articles contain an overview of arthritis, covering its various causes, epidemiology, pathophysiology, diagnostics, and alternate treatments. Although medications and other treatments can help treat arthritis, our research indicates that more clinical trials are necessary for some types of arthritis because they are more complex. More randomized controlled trials are needed to address arthritis. In the future, we hope to do a preliminary investigation into arthritis. Thanks to the cooperation of our colleagues, future counseling-based research in our state or country will assess the physical and mental health of patients and provide more accurate information about arthritis and its treatment.

ETHICAL STATEMENT

Be truthful and uphold a high standard of behaviour in all of our interactions and work-related endeavours. As we speak and act, let us be truthful.

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CONFLICT OF INTEREST

The authors attest that they are free of any known financial or personal conflicts of interest that would taint the findings of this study.

INFORMED CONSENT

Using websites, review articles, and other sources to produce research content.

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