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THE ROLE OF INFLAMMATION IN THE PATHOGENESIS AND MANAGEMENT OF ARTHRITIS: AN INTEGRATIVE REVIEW

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Abstract

Arthritis represents a set of inflammatory disorders causing pain, joint stiffness, and limited mobility, affecting millions globally. This review examines inflammation's role in the progression and management of common arthritis types—rheumatoid arthritis (RA), osteoarthritis (OA), psoriatic arthritis (PsA), and gout. It explores inflammatory mechanisms involving cytokines, chemokines, and immune dysregulation, which lead to joint and systemic damage. Conventional treatments like NSAIDs, corticosteroids, and DMARDs are analyzed, noting their benefits and limitations. The review also addresses biological therapies targeting IL-1, TNF-α, and IL-6, alongside growing interest in anti-inflammatory medicinal plants as potential long-term management options. Highlighting research gaps, it calls for optimized, personalized treatment protocols and suggests future directions integrating biologics, plant-based therapies, and early diagnostics. This synthesis enhances understanding of arthritis pathophysiology and highlights avenues for advancing therapeutic approaches to improve patient outcomes.

INTRODUCTION

Arthritis is an inflammatory condition affecting multiple body systems. It is mostly characterized by pain in the musculoskeletal system accompanied by stiff, swollen joints, resulting in severe functional impairment and negatively impacting overall quality of life (Ellis, 2019). Arthritis inflammation is a term that refers to the swelling and pain of the joints caused by various types of arthritis, such as rheumatoid arthritis, gout, and psoriatic arthritis. Arthritis inflammation can affect the function and quality of life of patients and may also lead to complications such as joint damage, infection, and cardiovascular disease (Shrivastava & Pandey, 2013). The causes of arthritis inflammation are not fully understood, but they may involve genetic, environmental, and immunological factors. Some of the common triggers of arthritis inflammation are infections,

injuries, stress, obesity, and aging (Makkar, Behl, Bungau, Kumar, & Arora, 2020).

Arthritis and inflammation are common health conditions that affect millions of people worldwide. According to recent studies, chronic inflammation has been linked to a variety of age-related diseases, “including Alzheimer's disease, Type 2 diabetes, arthritis, cancer, atherosclerosis, and hypertension” (Cannata et al., 2020; Rea et al., 2018; Trzeciak, Herbet, & Dudka, 2021).

The purpose of this review is to provide a comprehensive analysis of the inflammatory mechanisms underlying various forms of arthritis, highlighting their impact on disease progression and patient quality of life. It aims to synthesize current research on the role of inflammatory mediators in arthritis and explore both conventional and alternative therapeutic strategies. By doing so, this review seeks to identify gaps in existing knowledge and point towards potential avenues for future research and treatment advancements.

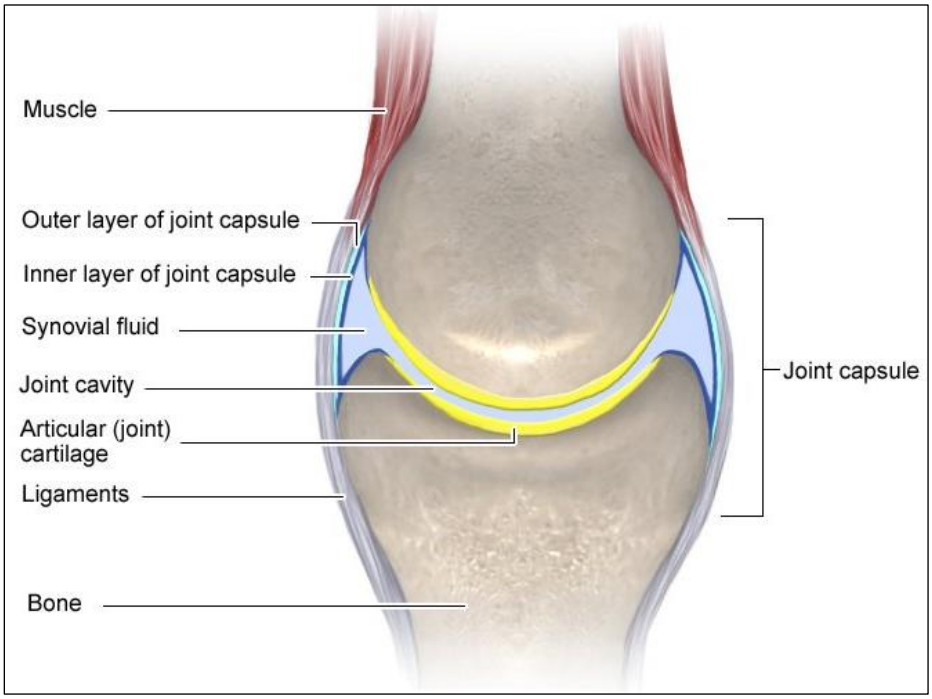

Figure 2. 1 Normal Joint Structure (https://www.informedhealth.org/how-do-joints-work.html)

2. Methodology

In order to develop a comprehensive review of arthritis and its underlying inflammatory mechanisms, a systematic approach was followed to gather relevant data and research articles. Various academic databases, including Google Scholar, PubMed, and Science Direct, were used to search for peer-reviewed journal articles, clinical trials, and review papers published within the last two decades. The search terms included "arthritis," "inflammation in arthritis," "rheumatoid arthritis," "osteoarthritis," "psoriatic arthritis," "gout," "inflammatory mediators in arthritis," and "arthritis treatment strategies." Articles were selected based on their relevance to the key themes of the review, which focused on the pathophysiology of arthritis, the role of inflammatory mediators, and current treatment approaches. Inclusion criteria involved studies with significant contributions to understanding inflammatory processes, advancements in therapeutic interventions, and evidence-based data on arthritis management. Articles excluded from the review were those with limited relevance, outdated findings, or a lack of substantial empirical evidence.

Data synthesis involved categorizing the articles into themes addressing the prevalence and burden of arthritis, the mechanisms of inflammation, and therapeutic strategies, including both conventional and alternative approaches. This method ensured a well-rounded perspective on the multifaceted aspects of arthritis and its management.

3. Prevalence of arthritis

With a population of over 200 million, Pakistan has significant healthcare requirements. According to reports, Pakistan has a 58–148/1000 prevalence of rheumatic illnesses. According to recent estimates, Pakistan has at least 15 million people with rheumatic disorders (Khan et al., 2020). Between 0.5 -1 percent of people globally have RA. In the UK, there is a reported prevalence of 0.81 percent. In India 0.75 percent (Khaliq, Khan, & Malik, 2020).

According to the WHO (World Health Organization) report, 18 million people were living with rheumatoid arthritis worldwide as of 2019. Rheumatoid arthritis affects around 70% of women, and 55% of those affected are 55 years of age or older. Thirteen million of the overall number of people with rheumatoid arthritis have moderate to severe levels of the disease, meaning they may benefit from rehabilitation. The prevalence of RA is believed to be between 0.5 and 1% worldwide, placing it behind gout and osteoarthritis as the leading causes of disability (Siddiqui et al., 2021). Rheumatoid arthritis (RA) has been more prevalent globally in the last several decades, and this trend is expected to continue. To lessen the overall burden of RA, then greater emphasis should be placed on early detection and treatment of the disease. In 2020, 595 million people worldwide were estimated to have osteoarthritis, with a 95% confidence interval of 535–656. This represents 7.6% (95% UI 6.8-8.4) of the global population and shows a significant increase in total cases during 1990 of 132.2% (95% CI 130.3-134.1) (Steinmetz et al., 2023). Rheumatoid arthritis is the most frequent type of arthritis in pre- and post-menopausal women in Pakistan, accounting for 47.3% of cases among female patients in the study. With an estimated 527 million cases globally, according to the Global Burden of Disease Study 2019, osteoarthritis (OA) is most communal kind joint inflammation. Pain and impairment are the outcomes of this long-term medical illness. About 60.6% of all prevalent instances in 2019 were knee OA, with hand OA, other kinds of OA, and hip OA coming up at 23.7%, 10.2%, and 5.5%, respectively. A major contributing factor to the increasing global prevalence and impact of the disease on people, healthcare systems, and society is the aging population combined with the obesity epidemic (Küçükdeveci, 2023).

4. Inflammation

Inflammation is a natural immune response to injury or infection, characterized by redness, swelling, heat, and pain. It was previously known that persistent inflammation is a pathological state that can cause cancers and Geriatric ailments, such as CV, autoimmune diseases as well as DM. Inflammatory processes induce Redox imbalance as well as reduce intracellular anti-oxidative capability, leading to the production of highly reactive species that can Interact with cellular membrane lipids as well as proteins, leading to irreversible dysfunction. Reactive oxygen species may induce genetic mutations as well as DNA harm, potentially contributing to the development of cancer as well as age-associated conditions (Rea et al., 2018). However, recent research has also found a strong association between inflammation and arthritis. Arthritis-related stress can impact the CNS (central nervous system) as well as cause central sensitization, leading to long-lasting pain and affective distress. Cytokines and chemokines play a role in joint inflammation as well as central sensitization. Evidence of these interactions has been reviewed in 05 types of arthritis: psoriatic arthritis, RA, ankylosing spondylitis, osteoarthritis of the hips and knees, as well as gout. Central sensitization in these conditions can result in chronic pain as well as psychosomatic anguish. (Harth & Nielson, 2019). Several researches have presented that chronic inflammation shows significant role in the

evolvement as well as progression of aging-associated ailments. In fact, chronic inflammation has been implicated as a leading cause of cancer and severe aging (Motta, Barone, Sica, & Selmi, 2023). Moreover, the concept of "inflammaging" has emerged, referring to chronic, low-grade inflammation that contributes to biological aging as well as increases the risk for age-related diseases and mortality among older adults. Additionally, researchers have identified that aging itself is closely associated with chronic low-grade inflammation, a phenomenon termed "inflammaging." Inflammaging has been recognized a risk factor for several age-related diseases and mortality among older adults (Fulop et al., 2021; Motta et al., 2023). In conclusion, chronic inflammation is a significant factor in the development and progression of various age-related diseases like arthritis. Multiple sources highlight the importance of controlling chronic inflammation in preventing and treating age-related diseases (Hossain, Kopec, Atiquzzaman, & Karim, 2022).

RA is an inflammatory ailment, while osteoarthritis (OA) is not. However, certain inflammatory cytokines are observed to be involved in Course of OA. Because of the intricate pathophysiology of arthritis, managing the condition requires a multifaceted strategy. Traditional anti-arthritic therapies have been utilized to treat arthritis disorders, but the documented negative consequences on human health have sometimes eclipsed their effectiveness and acceptability. Investigating complementary as well as alternative therapeutic options, like traditional medicinal plants as well as their phytochemicals, which have strong anti-inflammatory properties and less side effects on human health, is therefore of utmost importance (Gandhi et al., 2022). The causes of arthritis inflammation are not fully understood, but they may involve genetic, environmental, and immunological factors. Some of the common triggers of arthritis inflammation are infections, injuries, stress, obesity, and aging (Boissier, Biton, Semerano, Decker, & Bessis, 2020). The diagnosis of arthritis inflammation is based on the clinical history, physical examination, laboratory tests, and imaging studies of the affected joints (Boissier et al., 2020). The treatment of arthritis inflammation aims to reduce pain, improve mobility, prevent joint deformity, and slow down disease progression. The treatment options include medications, such as anti-inflammatory drugs, analgesics, corticosteroids, and disease-modifying anti-rheumatic drugs; physical therapy, such as exercises, massage, and heat or cold therapy; and surgery, such as joint replacement, fusion, or repair (Lin, Anzaghe, & Schülke, 2020).

4.1 Inflammatory Arthritis

Inflammatory arthritis encompasses a group of conditions characterized by joint pain, swelling, warmth, and tenderness, often accompanied by morning stiffness. The latest research has shed light on the diverse nature of inflammation in people with rheumatoid arthritis (RA), leading to the identification of subtypes of inflammation (Arumalla et al., 2023).

In addition to RA, other types of inflammatory arthritis have been the subject of recent research. Axial spondyloarthritis (axSpA) and axial psoriatic arthritis have been highlighted, with a focus on their distinct characteristics and impact on affected individuals. Understanding the specific features of these types of inflammatory arthritis is essential for accurate diagnosis and targeted treatment strategies (Kerola et al., 2023).

Rheumatoid arthritis, a chronic, systemic, autoimmune inflammatory ailment, remains a significant area of research focus. Recent studies have delved into the complexities of this condition, emphasizing its impact on the joints and periarticular soft tissues. This research contributes to a deeper understanding of the pathophysiology of rheumatoid arthritis as well as informs the development of innovative treatment approaches (McGonagle, Gibbon, & Emery, 1998; Niki, Matsumoto, Otani, Tomatsu, & Toyama, 2007).

4.2 Role of Inflammatory mediators

Inflammatory mediators play a significant role in the development of rheumatoid arthritis (RA) (Mariani et al., 2023). Some of the inflammatory mediators associated with RA include

Interleukin-1 (IL-1) that Mediates bone resorption and cartilage destruction (Shen et al., 2023). Reactive oxygen species, Prostaglandins (PG), Lipoxins, Leukotrienes, Platelet-activating factor as well as Nitric oxide, play a significant role in the development of RA (Makkar et al., 2023). While Role of Histamine, Kinins, Anaphylatoxins in RA is not well understood. Additionally, Prostanoids, particularly PG E2 play an important role in the pain associated with rheumatoid arthritis and may be partly responsible for joint swelling (Negi, Agarwal, Garg, Ali, & Kulshrestha, 2023).

Inflammasome inhibitors are a class of drugs that target the inflammasome, which is a protein complex that activates inflammatory cytokines such as interleukin-1 beta (IL-1β) and interleukin-18 (IL-18) (Landy, Carol, Ring, & Canna, 2023). Inflammasomes are involved in various inflammatory and autoimmune diseases, including rheumatoid arthritis (Jiang et al., 2023).

Chronic inflammatory arthritis causes joint damage by stimulating osteoclasts, which are cells that break down bone and cartilage. Osteoclasts are formed by cytokines, "such as RANKL, MCSF, TNF, IL-1 and IL-6", that are produced in the inflamed synovial membrane. Blocking osteoclast formation can prevent joint damage in animal models and human patients with rheumatoid arthritis (Nandakumar, Fang, Wingbro Ågren, & Bejmo, 2023; Schett, 2008).

Inflammasome inhibitors work by targeting the upstream and downstream molecules in the Inflammasome signaling pathway. For example, some Inflammasome inhibitors block the activation of caspase-1, which is a key enzyme that cleaves pro-IL-1β and pro-IL-18 into their active forms. Other inflammasome inhibitors target the NOD-like receptors (NLRs), which are the cytosolic sensors that recognize pathogen-associated molecular patterns (PAMPs) or danger-associated molecular patterns (DAMPs) and activate the inflammasome. Some inflammasome inhibitors also inhibit the production or secretion of IL-1β and IL-18 by blocking their transcription, translation, or release (Bhaskar et al., 2023).

Inflammasome inhibitors have shown promise as novel therapies for various inflammatory and autoimmune diseases, including RA, gout, type 2 diabetes, as well as cancer. However, the clinical efficacy and safety of inflammasome inhibitors are still under investigation, and more research is needed to optimize their design, dosing, and delivery (N. Li et al., 2023).

5. Types of Arthritis

Arthritis disorders are described by inflammation of the joints as well as connective tissues, which results in reduced joint function and is associated with significant morbidity and death (Figus, Piga, Azzolin, McConnell, & Iagnocco, 2021). There are more than 100 distinct types of arthritic disorders, which cumulatively impose a significant financial and health burden on communities (Hsieh et al., 2020). "Rheumatoid arthritis (RA), osteoarthritis (OA), lupus, psoriatic arthritis, ankylosing spondylitis (AS), as well as gout are common arthritic illnesses" (Park, Mendy, & Vieira, 2018).

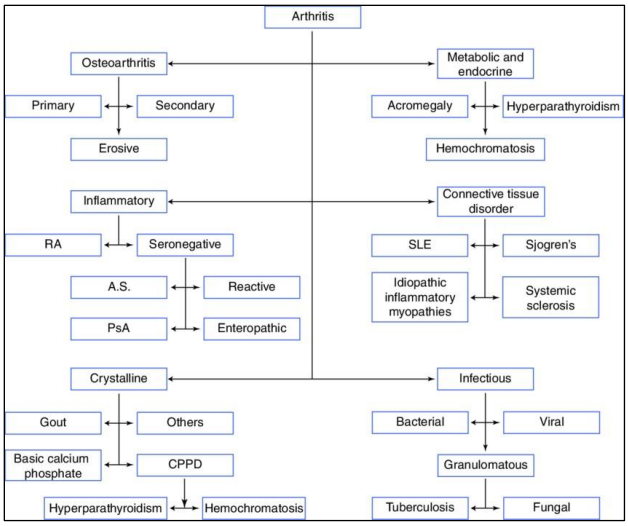

Figure 2. 2 Different types of arthritis (https://www.researchgate.net/figure/Classification-of-arthritis_fig1_285613106)

5.1 Rheumatoid arthritis

Rheumatoid arthritis (RA) is an autoimmune disease marked by inflammation in synovial tissue, cartilage, as well as bone, occasionally extending to non-joint regions (Smith & Berman, 2022). In the recent past, it is evident that genetic as well as epigenetic factors play a role in RA, however, the environment, including, particularly the microbiome, cigarette smoke, and dust exposure can also contribute, which also constitutes an "internal" environment (Scherer, Häupl, & Burmester, 2020). There appears to be a significant interaction between Primitive immune system as well as Antigen-specific immune system components. Impairments in the cellular as well as humoral immune response responses result in the formation of Abs "autoantibodies", most remarkably RF "rheumatoid factors" and antibodies against post-translationally modified proteins "Anti-modified protein antibodies (AMPA)" that include antibodies against various modifications such as "citrullination (ACPA), carbamylation , and acetylation (AAPA)", as well as T and B-lymphocyte migration into the synovium (Weyand & Goronzy, 2021). There is also a strong induction of the nonspecific defense mechanisms, dynamically activated monocyte/macrophage cells in the affected tissue regions. RA clinical as well as morphological features are the outcome of several unique phenomena: inflammation manifests as joint pain, swelling, and subsequent cartilage and bone degeneration, as well as systemic manifestations induced by arachidonic acid metabolites and different inflammatory cytokines (Scherer et al., 2020).

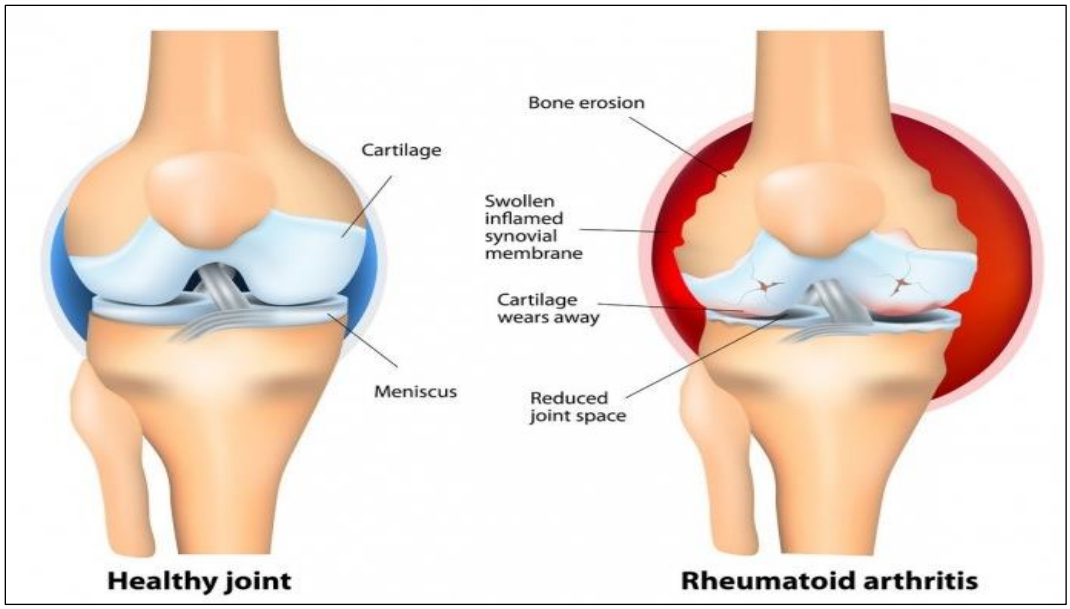

Figure 2. 3 Rheumatoid arthritis (Inflammatory arthritis) (https://www.disabled-world.com/health/autoimmunediseases/rheumatoid-arthritis/#google_vignette)

5.2 Osteoarthritis

The most prevalent form of arthritis in the world is osteoarthritis (OA) (Allen, Thoma, & Golightly, 2022). This condition results in subchondral sclerosis, osteophytes growing within the joints, and a weakening of the muscles surrounding the joints (Lockwood, 2021). These processes have a significant impact on the person as well as society at large by contributing to the experience of pain, functional restrictions, and disability (Scheuing, Reginato, Deeb, & Kasman, 2023). Three main symptoms of OA include stiffness in the joints, discomfort, and limited range of motion. Osteoarthritis usually affects one or more joints, although it can also affect the hands, feet, hips, knees, and spine (Yu, Huang, Lu, Tong, & Chen, 2022). In 2021, OA affected more than 500 million people worldwide, with over 22% of adults aged 40 and older being diagnosed. Patients with advanced OA often need joint replacement surgery because there are no viable long-term therapeutic remedies available. The global growth rate for this surgical operation is 10% each year, with OA being the cause of 95% of cases. Even though they are widely used, the prosthetic joints used in these surgeries are short-lived and may not work out well (Yao et al., 2023). According to projections, OA will rank as the fourth most common cause of disability worldwide. It also plays a major role in driving up medical and healthcare expenditures, as well as indirect costs related to early retirement and job loss.

Imaging-based estimates of the frequency of hand osteoarthritis (OA) vary widely, from 27% to more than 80% (Motta et al., 2023). According to an analysis of data from the Framingham Cohort, the prevalence of OA with at least one hand joint exhibiting symptoms was found to be 26.2% in women and 13.2% in men aged 70 or older (Y. Zhang et al., 2002). Although less common than hand OA, knee involvement is more common in women. In the Framingham Study, individuals over 45 years old had a radiographic knee OA prevalence of 19.2%, which rose to 43.7% in people over 80 years old. In comparison, hip OA is less frequent. Moreover, OA has a major impact on resource usage and healthcare expenses. The estimated average yearly direct patient costs in the US ranged from $1,442 to $21,335 (adjusted to US$ equivalent of 2015) (Jin et al., 2023). Work-related indirect expenditures need to be considered as well. Workers with knee OA had

greater rates of disability or sick leave, according to a population-based study conducted in Sweden (Hubertsson, Turkiewicz, Petersson, & Englund, 2017).

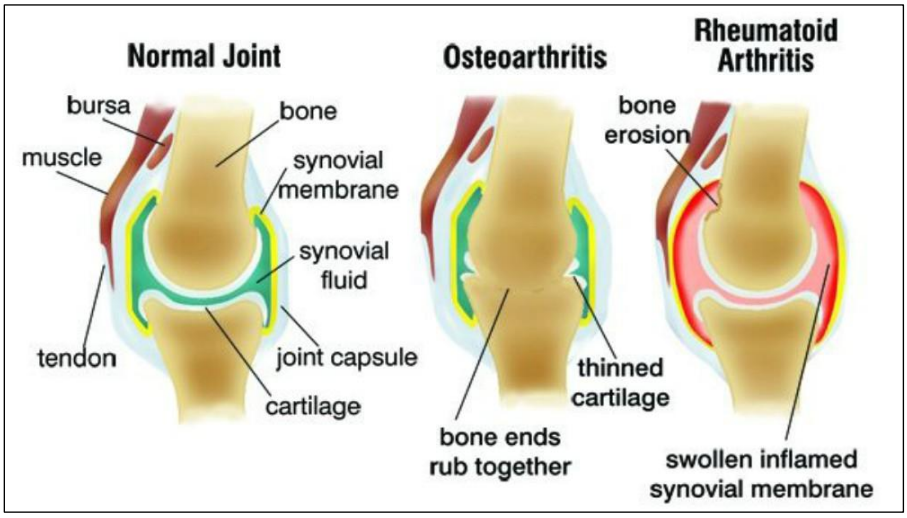

Figure 2. 4 Osteoarthritis (degenerative arthritis) (https://tristatearthritis.com/arthritis/what-is-osteoarthritis-rheumatoid-arthritis-and-gout)

5.3 Psoriatic arthritis

Psoriatic arthritis (PsA) is a musculoskeletal disorder that is chronically inflammatory and associated with psoriasis (Stober, 2021). About thirty percent of people who have psoriasis end up going on to acquire PsA in their lifetime.

"Peripheral arthritis, spondylitis, dactylitis, or inflammation of the entire hand, as well as enthesitis, or inflammation where tendons, ligaments, or joint capsules adhere to the bone", are among the musculoskeletal symptoms associated with PsA (Kishimoto et al., 2021). Psoriasis vulgaris, also known as plaque psoriasis, is the most prevalent kind of psoriasis linked with PsA. Nail disease is another manifestation of Psoriasis. In addition to the musculoskeletal and cutaneous features, PsA patients frequently struggle with fatigue, diminished work capacity, sleep disruptions, physical function limitations, and social participation (Aouad, Moysidou, Rakotozafiarison, Fautrel, & Gossec, 2021). Along with Associated features like uveitis and IBD, PsA is intertwined with multiple concurrent ailment "such as depression as well as anxiety, obesity, and metabolic disorders (diabetes, hypertension, hyperlipidemia, fatty liver disease, cardiovascular issues)" (Perez-Chada & Merola, 2020). Young adults in their prime years of productivity are frequently affected by psoriatic arthritis (PsA), which has been connected with a decline in quality of life (QOL) (Conaghan et al., 2020). Delays in diagnosis and the difficulty in finding appropriate therapy can exacerbate joint deterioration and, in many cases, result in permanent impairment (Saketkoo et al., 2021). Since doctors' judgment is a principal component in diagnosis of PsA in typical clinical settings, clinicians must become familiar with the fundamental clinical features of PsA.

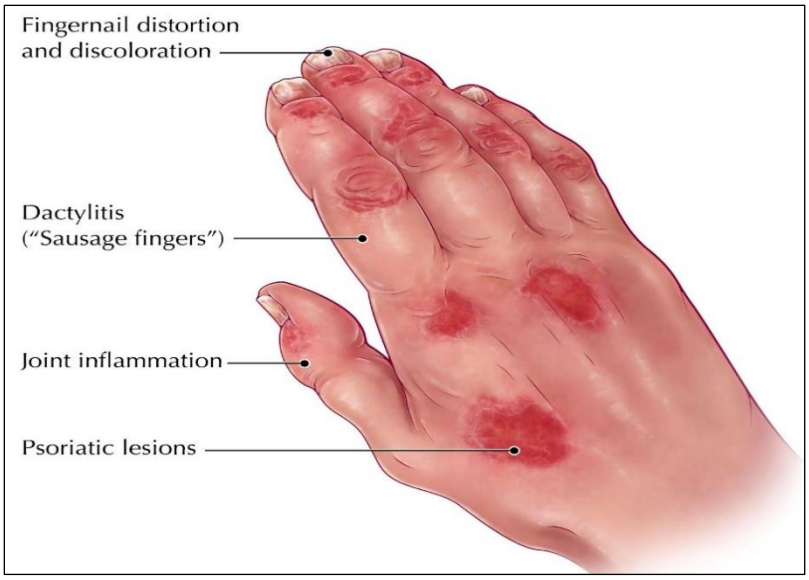

Figure 2. 5 Psoriatic arthritis (Inflammatory arthritis) (Psoriatic Arthritis Illustration)

5.4 Gouty arthritis

Gout is caused by MSU "monosodium urate" crystals deposited in synovia and other body tissues. Unpredictable pain, redness, swelling in the joints, and joint degeneration from repeated bouts are all common symptoms of gout attacks (Choi & Stone, 2023). Apart from the inherent clinical manifestations, gout attacks frequently result in numerous grave consequences, including hyperuricemia, chronic renal disease, early mortality, and numerous metabolic disorders (Singh & Gaffo, 2020). The incidences of gout are rising annually due to people's increased consumption of purine-containing foods. In Western nations, gout currently affects 2.7% to 6.7% of the population, with an incidence rate of 0.58 to 2.89 cases per 1000 people annually. More importantly, there may be underdiagnoses, which means that these numbers may underestimate the true prevalence of gout (Dehlin, Jacobsson, & Roddy, 2020). The development of gout therapy medications has been aided by the quick development of medical technology and the effectiveness of several gout animal models. Unfortunately, there isn't a reliable treatment for gout anywhere in the globe right now (Singh et al., 2017). The rate of hospital stays and outpatient visits has increased dramatically due to the rising frequency of gout and the lack of effective gout care options, which has raised overall economic expenses. Conversely, individuals have low Well-being as a result of chronic medication and activity constraints brought on by gout development. These days, gout is a serious health dilemma that costs a lot of money as well as causes a lot of misery (Dehlin et al., 2020).

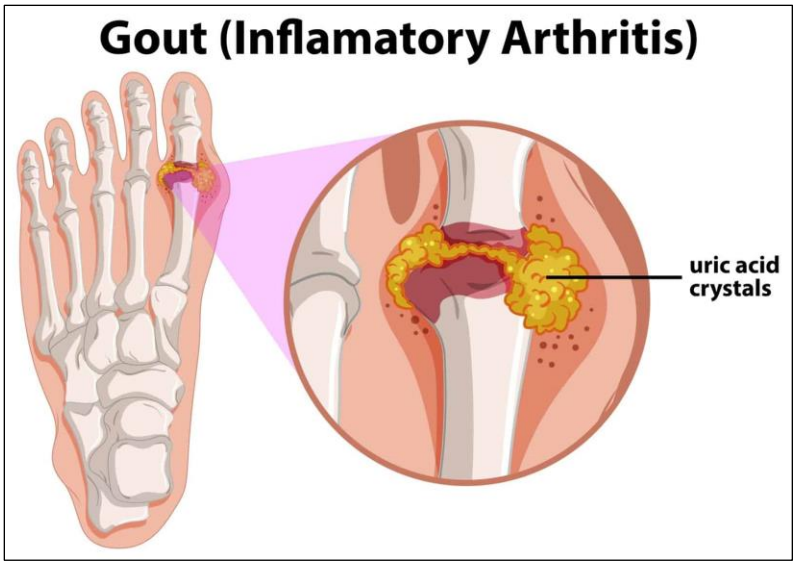

Figure 2. 6 Gout arthritis (crystal deposition-induced arthritis) (https://www.arthritisvirginia.com/arthritis-rheumatology/gout/)

6. Causes/Pathogenesis

The pathogenesis of arthritis is a complex process that involves multiple genetic, environmental, immunological, and other factors.

6.1 Rheumatoid arthritis

RA is the predominant long-lasting inflammatory arthritis, impacting roughly 1 percent of individual (Babaahmadi et al., 2023). RA is a multifaceted disease that is influenced by genetic, environmental, and stochastic factors (Petrovská, Prajzlerová, Vencovský, Šenolt, & Filková, 2021; Romão & Fonseca, 2021) . According to scientific studies, genetic factors account for approximately 50% of the risk for RA (Petrovská et al., 2021). The classification of RA into seropositive and seronegative subtypes segregated by the presence / absence of RF and anti-citrullinated protein antibodies (ACPAs) indicates specific risk factors associated with each subtype (Padyukov, 2022). The ACPAs-positive subtype is strongly correlated with risk alleles of tyrosine phosphatase non-receptor type 22 (PTPN22), human leukocyte antigen D-related (HLA-DR) alleles, and genes related to tumor necrosis factor-receptor associated factor 1 and complement component 5 (TRAF1/C5) (Padyukov, 2022). Interferon regulatory factor 5 (IRF-5), on the other hand, is specifically related with the ACPAs-negative subtype. There has been an increasing knowledge in the context of epigenetics in the genesis of RA during the last decade. Epigenetic processes cause inheritable changes in gene expression while leaving the deoxyribonucleic acid (DNA) sequence unchanged (Klein, Karouzakis, & Gay, 2018; Nemtsova et al., 2019) . This could explain the relatively low concordance rate seen among monozygotic twins (9-15%) and the disease's inadequate influence of genetic variables. A recent large epigenome-wide association study discovered different methylation patterns in monozygotic twin pairs with RA, offering information on the significance of epigenetic alterations (Webster et al., 2018). Furthermore, because external variables such as medications, smoking, and nutrition can cause epigenetic modifications, these systems may act as a link between interactions in the genome and the environment.

A key feature of rheumatoid arthritis is synovial hyperplasia, which plays a pivotal role in causing an invasive pannus to form. The presence of accumulating T cells existing in the synovial membrane led to the assumption of a "T-cell-dependent inflammatory" response induced by an unfamiliar antigen (Nygaard & Firestein, 2020). The typically thin synovial lining,

comprised of 1-3 cell layers, thickens significantly in rheumatoid arthritis (RA). This thickening is caused by macrophage-like cell invasion and enhanced multiplication of local synovial fibroblasts. The severity of cartilage erosion, which leads to the creation of an inflammatory pannus, is closely related to the level of synovial hyperplasia(Masoumi et al., 2021). This pannus adheres to and infiltrates joint cartilage, whereas osteoclast activity induces bone degradation. Synoviocytes in this location produce generous amounts of matrix-degrading enzymes such as gelatinase, as well as collagenase (Bian et al., 2023).

While the exact causes of RA remain unknown, it is certain that in the fully developed stage of the disease, activated cells derived from macrophages as well as fibroblasts play a dominant role in the destructive process. Understanding the signaling pathways that turn synovial cells into pannus tissue has revealed a complicated network of interacting cytokines. IL-6 as well as TNF-α are cytokines that alter the balance between the creation as well as breakdown of cartilage, bone matrix within the joint (Kondo, Kuroda, & Kobayashi, 2021).

6.2 Osteoarthritis

The etiology of osteoarthritis (OA) is multifaceted, resulting from the interaction of both systemic and local elements. Age is by far the most common risk factor for its start, with the elderly being the most affected (Hasan & Shuckett, 2010). Joint tissues change as increase age, making the joint more vulnerable to the slow onset and advancement of osteoarthritis (OA) over time. While articular cartilage is tough and able to resist recurrent stress from daily activities, it is nonetheless susceptible to injuries that can harm the cartilage as well as the subchondral bone. Such injury increases the likelihood of osteoarthritis progression when combined with intra-articular fractures. Directly and indirectly, obesity contributes to OA. Obese peoples have elevated body weight, as shown by a greater body mass index (BMI), causes significant overloading and damages weight-bearing joints (Toivanen et al., 2010). According to twin studies, genetic variables account for 39–65% of instances of OA in the general population (Ashkavand, Malekinejad, & Vishwanath, 2013). Certain rare mutations in type II, IX, or XI collagen—common components of articular cartilage—cause hereditary types of osteoarthritis (OA) that can cause premature OA onset, starting as early as puberty and presenting as a severe, debilitating form of arthritis affecting many joints. The anatomical shape of the joint also affects the development of OA; lower extremity alignment is a key determinant in the development of knee OA. In addition, cruciate ligament tears, Varus and valgus deformities, and a leg length difference of ≥1 cm are additional variables that increase the risk of OA development and progression in the knee (Yunus, Nordin, & Kamal, 2020).

6.3 Psoriatic disease

Psoriatic disease (PsD) develops as a result of a complicated process that involves interactions between innate and adaptive immunological responses, local variables unique to ailment sites (joints, skin, and spine), genetic predisposition, as well as environmental triggers including biomechanical stress (Stober, 2021). Different clinical manifestations result from this interaction. Type 1 cells (CD4+ T helper 1 (Th1) or CD8+ cytotoxic T cells (T cytotoxic 1 (Tc1)) or type 17 cells (CD4+ T helper 17 (Th17) or CD8+ cytotoxic T cells (T cytotoxic 17 (Tc17)) are the result of innate immune system cells in the skin, being activated by environmental or mechanical stimuli in a genetically susceptible context (O'Brien-Gore, Gray, Durham, Taams, & Kirkham, 2021). Disease is influenced by the ratio of stromal cells to effector T cells at local locations, genetic predisposition, and the cytokine environment. The MHC (Major histocompatibility) class I alleles, haplotypes, as well as effector T cell subsets linked to four distinct psoriatic arthritis phenotypes have been identified: "cutaneous-predominant," "synovial predominant," "entheseal predominant with symmetrical axial disease," and "psoriatic arthritis mutilans (PAM)." Certain genetic variations and cellular mechanisms are associated with each

trait. The MHC class I alleles, haplotypes, and effector T cell subsets linked to four distinct psoriatic arthritis phenotypes have been identified: "cutaneous-predominant," "synovial predominant," "entheseal predominant with symmetrical axial disease," and "psoriatic arthritis mutilans (PAM)." Certain genetic variations and cellular mechanisms are associated with each trait (Rahmati, Tsoi, O'Rielly, Chandran, & Rahman, 2020).

6.4 Gout

Gout is not caused by a single factor but its pathophysiology consists of multiple interrelated stages. One of the main risk factors for gout is hyperuricemia, which is thought to be necessary for the crystallization of monosodium urate (MSU) (Wu et al., 2022). For most people, the primary cause of hyperuricemia is the under excretion of urate via the kidneys and the digestive system. Serum urate levels are related to a number of metabolic, environmental, and genetic factors that affect urate production or transport (Narang & Dalbeth, 2020). The production of MSU crystals is mostly dependent on urate super saturation, while other parameters like as pH, temperature, and connective tissue components also play a part. The Cryopyrin inflammasome and Nucleotide-binding oligomerization domain (NOD) receptors are crucial players in the inflammatory response triggered by MSU crystals, with interleukin-1β serving as the primary cytokine in triggering the inflammatory cascade (Narang & Dalbeth, 2020). Differences in the inflammatory response's regulatory mechanisms can affect a person's tendency to develop gout. Advanced gout is characterized by tophus production, which results in the development of structural joint damage in gout. Both MSU crystals and the inflammatory tissue component of the tophus contribute to this process.

7. Symptoms

Since inflammation of the joints is a common feature of all the illnesses that make up arthritis, there is a wide range of variation in the conditions' symptoms. However, arthritis is indicated by a few typical symptoms. A defining characteristic is persistent joint discomfort, which is frequently described as a dull aching or a sharp, stabbing sensation. It is common to experience stiffness, especially in the morning or after periods of inactivity, which makes it difficult to move joints freely (Somers, Wren, & Shelby, 2012). Common indicators of inflammation around afflicted joints include swelling and redness. As the illness worsens, a reduced range of motion and difficulties carrying out daily duties could result (Michaud et al., 2021). Rheumatoid arthritis and other inflammatory types of arthritis can induce systemic symptoms such as weight loss, fever, and exhaustion. RA is commonly characterized by morning stiffness that lasts longer than an hour and chronic pain in one or more joints that usually gets better with movement (Van Boheemen et al., 2023). With the growing recognition of RA as well as the advancement of novel treatment strategies, some people reduced disease activity or remission with prompt diagnosis and intervention. However, a significant portion of patients either do not get enough care or do not react well to it. The chronic inflammation caused by RA can lead to joint degradation in situations when early-stage treatment is inadequate. This can result in disability, reduced functional capability, early unemployment, and a shortened life expectancy. According to projections, people with RA will live 3–10 years less than the average people if RA is not effectively managed (Kim & Suh, 2020).

An asymmetric inflammatory distribution involving monoarticular to oligoarticular patterns is one of the distinguishing features of joint engagement in Psoriatic Arthritis (PsA), especially in its early phases. Sacroiliitis and inflammation in the distal tiny joints of the hands, as well as feet, are possible side effects of spondylitis. These particular characteristics help distinguish PsA from RA, which is characterized by a more symmetric and polyarticular distribution, proximal hand and foot engagement, more sensitive and swollen joints, and the lack of Sacroiliitis (Haroon, Gallagher, & FitzGerald, 2014). On the other hand, as opposed to PsA and RA, osteoarthritis (OA) is non-inflammatory

yet can still cause joint involvement. While joint pain and stiffness associated with OA usually increase with activity and improve with rest, patients with PsA and RA frequently have morning stiffness or worsening joint stiffness with inactivity. Distal interphalangeal joint involvement is common in both PsA and OA; however, OA is associated with related bone spurs, whereas PsA distal interphalangeal involvement frequently corresponds with psoriatic nail disease and joint inflammation (Sobolewski, Walecka, & Dopytalska, 2017).

Gout symptoms include swelling, discomfort, and soreness in a peripheral joint or bursa, most often the first metatarsophalangeal joint (56% to 78% of patients). There is a chance that other joints, such as the interphalangeal joints (6% to 25%), ankle (18% to 60%), upper limb (13% to 46%), and midfoot (25% to 50%), would also be affected (Clebak, Morrison, & Croad, 2020). A tophus is present when a transparent nodule beneath it drains a chalk-like substance, frequently with underlying vascularity (Yuan et al., 2023). The ear, the olecranon bursa, and the pulps of the fingers are common sites. Acute, inter critical, chronic, and asymptomatic hyperuricemia are the four stages that gout develops through. Most cases of acute gouty arthropathy are monoarticular, affecting over 50% of patients primarily in the lower limb, specifically in the podagra. In addition, ankles, knees, palms, elbows, bursae as well as tarsal joints, are frequently impacted. MSU crystals are often found at the entheses and in tendons. Acute gout attacks cause, patients afflicted joints, tendons, or bursa to suddenly become extremely painful, swollen, and red. Although this presentation could seem like an infection, the episode is self-limiting and usually goes away in a few days or a few weeks (Clebak et al., 2020).

During an acute attack, serum uric acid levels may be normal but inflammatory blood parameters may be high. The time in between acute gouty attack episodes, when there are no symptoms, is known as inter-critical gout (Weaver et al., 2021). Chronic arthropathy, which is characterized by persistent synovitis, tophus development as well as deposition, and ultimately erosions as well as joint degeneration, can result from recurrent acute gout bouts. About one-third of patients have tophaceous gout within five years if left untreated. Non-tender soft tissue masses called tophi can be found in various places, such as subcutaneous soft tissues, bursae, tendons, ligaments, and intra- or periarticular soft tissues. They are the outcome of a long-term granulomatous response to MSU crystals, with granulation tissue around a fibro vascular matrix that contains MSU crystals at its core. Tophaceous gout can cause osseous erosions and commonly affects the hands, wrists, extensor surfaces of the knees, and elbows. Tophi typically develop in mechanically stressed regions, like the vicinity of the podagra joint, the patellar as well as Achilles tendons, the prepatellar bursae and the olecranon. MSU deposition is frequently seen in the popliteus tendon, infrapatellar fat pad, peroneal tendons, and cruciate ligaments. In addition, tophi can develop in the tip of the nose, and the auricular appendages, resulting in a deformity and limited movement of the joints (Pradhan, Sinha, Sharma, & Sinha, 2020).

8. Diagnosis

Evaluating clinical aspects, including signs as well as symptoms, predictive tests, differential diagnosis, potential consequences, as well as extra-articular manifestations, is an essential part of managing rheumatoid arthritis (RA). It is critical to diagnose RA as soon as possible to distinguish it from other types of arthritis and autoimmune illnesses. Promptly implementing the appropriate treatment, reducing potential long-term consequences, and maintaining effective disease management all depend on this rapid detection (Radu & Bungau, 2021).

The ACR (American College of Rheumatology) as well as the EULAR (European League Against Rheumatism) developed the 2010 classification benchmarks for rheumatoid arthritis, which evaluate a several characteristics, like risk factors, the quantity, and type of affected joints, as well as the duration of symptoms. The main objective is to move the focus of RA management from the late-stage phase to early identification (Radner, Neogi, Smolen, & Aletaha, 2013). Based on

predetermined criteria, the categorization system assigns scores that are periodically reevaluated. A score of 1 is assigned to 2–10 big joints, 2–3 small joints (± large joints) to 3 and 4–10 tiny joints (± large joints) to 4. A score of 5 is assigned to more than 10 joints (containing at least 1 small joint) (Aletaha et al., 2010). People with a score of 6 or more than 6 are categorized to have RA.

Biomarker panel identification and improvement present a promising medical tool for prognostic, predictive, therapeutic, and diagnostic applications. The most recent classification includes four biomarkers (ESR, CRP, RF, and ACPA), each with particular limits (Banal, Dougados, Combescure, & Gossec, 2008). Additional diagnostic proteins, including antibodies against mutant citrullinated vimentin (anti-MCV), antibodies against carbamylated proteins (anti-CarP), and the 14-3-3 eta protein, have been revealed in recent investigations. These proteins are critical for the early diagnosis of RA. Correlation between biomarker detection and quantification with modern imaging technologies is necessary to achieve accurate diagnosis. Because of their much improved-accuracy over traditional radiography, magnetic resonance imaging (MRI), computed tomography (CT), and ultrasonography (US) are classified as imaging tools for early diagnosis in the ACR-EULAR 2010 (Aletaha et al., 2010). Conventional X-ray evaluations of joints frequently fail to identify early indications of deterioration and erosion.

Studies using radiographic assessment to diagnose and measure the severity of osteoarthritis (OA) frequently use the Kellgren and Lawrence score (Olsson, Akbarian, Lind, Razavian, & Gordon, 2021). According to the presumptive order of joint space loss, osteophytes, sclerosis, and cysts, this scoring method allocates overall severity ratings between 0 and 4 (Misir, Yildiz, Kizkapan, & Incesoy, 2020). Due to its increased sensitivity, magnetic resonance imaging (MRI) can identify diseases early and shows indications of degenerative changes in soft-tissue structures outside of cartilage, synovitis, bone marrow lesions, and matrix modifications in cartilage. Ligaments and the menisci of the knee are also included in this (Verma & Belapurkar, 2021).

Psoriatic arthritis (PsA) is highly variable and has a wide range of symptoms. Axial and peripheral PsA are the two primary forms that can coexist. Subgroups of peripheral symptoms, such as arthritis as well as extra-articular (enthesitis and dactylitis), can be further categorized. Imaging is frequently needed to determine the type of involvement, evaluate radiological damage, as well as track the evolution of PsA due to the intricacy of this disorder (Fassio, Matzneller, & Idolazzi, 2020). Although traditional radiography has long been the gold standard, interest in non-conventional imaging modalities such as MRI, as well as Ultrasound (US), has grown recently. These modalities have good sensitivities and specificities for identifying synovitis, making them useful for diagnosing and monitoring all forms of PsA. In the early or preclinical stages, in particular, they are useful in differentiating PsA from other musculoskeletal illnesses (Rida & Chandran, 2020). It's important to remember, nevertheless, that the US is ineffective in axial PsA diagnosis. In addition, additional imaging modalities, like high-resolution peripheral CT (HRpQ-CT) and computed tomography (CT) with an emphasis on dual-energy CT, show potential in assessing bone erosions and new bone growth (Antony & Tillett, 2021).

Gouty arthritis diagnosis requires a multimodal approach including imaging scans, laboratory testing, and clinical examination (B. Zhang, Yang, & Wang, 2020). Clinically, the first metatarsophalangeal joint (MTP), often referred to as podagra, is the focus of attention when severe pain, swelling, and redness suddenly appear in joints. Serum uric acid levels are measured as part of laboratory tests. Elevated levels can be a sign of gout, but normal levels can also rule out the diagnosis, particularly in the event of an acute attack (Deng et al., 2021). A definitive diagnostic marker is the presence of needle-shaped monosodium urate (MSU) crystals under a microscope, which makes joint fluid examination through synovial fluid aspiration essential (Christiansen, Østergaard, Slot, Fana, & Terslev, 2021). While more recent imaging

modalities like ultrasound (US) and MRI might detect distinctive features such as joint erosions and tophi in chronic instances, traditional radiography, particularly X-rays, plays pivotal roles. When it comes to identifying synovitis, helping with differential diagnosis, and assessing the course of the disease, US as well as MRI both show sensitivity and specificity; however, US is more useful for peripheral joints. The way the patient reacts to anti-inflammatory treatments, like colchicine or nonsteroidal anti-inflammatory drugs (NSAIDs), further supports the diagnosis. A full diagnosis of gouty arthritis is made through a combination of clinical examination, laboratory testing, joint fluid analysis, and imaging techniques, particularly MRI and US. The most often used diagnostic technique for conclusive confirmation is synovial fluid aspiration (Abdellatif, Ding, Khorshed, Shojania, & Nicolaou, 2020; S. Li et al., 2022).

It is necessary to evaluate specific features so that differentiate different forms of arthritis. Sometimes a biopsy is mandatory to distinguish between diseases that present similarly. Ankylosing spondylitis as well as psoriatic arthropathy exhibit different patterns of synovitis from RA, which has symmetric involvement of large as well as small joints, comprising the wrist as well as elbow. RA is characterized by more inflammation than osteoarthritis (Wasserman, 2011). In addition to being linked to systemic lupus erythematosus (SLE) and Sjögren's disease, rheumatoid factor (RF) is also present in RA (Rasmussen et al., 2016). Compared to RA, SLE is more common to have antinuclear antibodies. In rheumatoid arthritis, the most noticeable erosive variations on X-rays are seen. Dermatological presentations may indicate systemic sclerosis, psoriatic arthritis (PsA), or SLE (Gutierrez, Filippucci, Salaffi, Di Geso, & Grassi, 2011). The shoulders and hips are usually the first areas affected by polymyositis (PM). Inflammatory processes associated with spondylarthritis (SpA) frequently impact the back and eyes (Schoen, 2020). If a patient's symptoms haven't persisted for more than six weeks and they don't satisfy the ACR-EULAR criteria, they may be distrusted of having Lyme arthritis or viral infections such enterovirus/parvovirus (Schoen, 2020). Differentiating polyarthritis from osteoarthritis and fibromyalgia can be difficult, particularly if pain is the only symptom. Polyarthritis is characterized as arthritis affecting more than four joints (LUO, LUO, MO, YIN, & CHEN, 2023). Tests for blood and urine also aid in determining an accurate diagnosis.

9. Management

Researchers and healthcare professionals have conducted numerous clinical trials to evaluate various medicines for the treatment of arthritis. Some of these medications have been approved for ordinary clinical usage. NSAIDs (Nonsteroidal anti-inflammatory medicines) such as ibuprofen, etodolac, acetylsalicylate, and naproxen are commonly used to treat pain, swelling, as well as inflammation. NSAIDs work by decreasing the catalytic function of COX, which is intricate in the formation of PG "prostaglandins". NSAIDs decrease COX-2 production at inflammatory sites, whereas cyclooxygenase-1 inhibition in diverse tissues "platelets as well as the gastro duodenum mucosa" causes typical side effects such as bleeding and gastrointestinal ulcers (Paglia et al., 2021). Corticosteroids, including glucocorticoids, are another powerful class of anti-inflammatory medications that alter gene manifestation by engaging with glucocorticoid receptors, resulting in anti-inflammatory as well as immunosuppressive responses. They are, linked to side effects including ulcers, abdominal pain, nausea, diabetes, and osteoporosis (Hua, Buttgereit, & Combe, 2020).

Due to the side effects of NSAIDs as well as corticosteroids, a class of immunosuppressive as well as immunomodulatory medicines known as disease-modifying antirheumatic medications (DMARDs) has been created to prevent and treat rheumatoid arthritis (RA). Conventional synthetic (cs) DMARDs such as sulfasalazine, methotrexate, chloroquine, leflunomide, hydroxychloroquine, as well as gold salts are indicated as the initial therapy approach upon RA diagnosis. Methotrexate is very popular among patients due to its low cost and high

efficacy. Although the precise mechanisms of action of csDMARDs are unknown and may include several signal pathways, they are preferred due to their low cost. If csDMARDs do not work, further possibilities include "biological (b) DMARDs or targeted synthetic (ts) DMARDs". "bDMARDs', which include certolizumab, infliximab, adalimumab, canakinumab, tocilizumab, as well as secukinumab, are monoclonal antibodies with particular targets including IL-1, TNF-α, IL-17, and IL-6 (Lamers-Karnebeek, Jacobs, Radstake, van Riel, & Jansen, 2019; Rice, Ghandi-Das, & Negi, 2020). tsDMARDs also target specific entities; for example, tofacitinib, baricitinib, filgotinib, upadacitinib, and decernotinib all target Janus kinases (JAK) (Ho et al., 2019).

While DMARDs have shown significant effectiveness in treating RA, it is apparent that a substantial percentage of patients may experience treatment problems, such as nonresponse as well as limited efficacy (Conigliaro et al., 2023). Rheumatologists advise the use of combination therapy in RA patients to improve therapeutic outcomes (Gong et al., 2021). A combination of methotrexate and glucocorticoid, for example, has been proven to offer relief for around 25% of patients over 6-months period. If methotrexate plus glucocorticoid is ineffective, the addition of either biological DMARDs or targeted synthetic DMARDs to conventional synthetic DMARDs is advised. Methotrexate plus tocilizumab/rituximab/tofacitinib, as well as other combinations, may be used (Smolen et al., 2020). Aside from nonresponse, several DMARDs might cause undesirable clinical symptoms like mouth inflammation, rash, gastrointestinal upset, blood deficiency, lung infection, as well as nephritis, thereby exacerbating the condition (Gautam et al., 2016; Lee et al., 2020).

In the current state of knowledge, osteoarthritis (OA) does not have a cure. Clinical OA treatments have, for a long time, focused more on symptom relief for joint discomfort than on slowing the disease's progression. But in recent times, the emphasis on OA has turned to early prevention and stopping or delaying the disease's course before serious harm is done. As such, it is imperative to identify and understand possible biomarkers and treatment targets at different phases of osteoarthritis. Researchers and medical professionals have expended significant energy identifying critical molecules and signaling pathways that are essential for the onset and progression of osteoarthritis (OA). In the long run, these could be useful therapeutic targets to reduce or stop synovial joint deterioration.

Biologic treatments and traditional or conventional disease-modifying antirheumatic medications are offered for the treatment of psoriatic arthritis (PsA) (Ogdie, Coates, & Gladman, 2020). Biologics include inhibitors of TNF (TNFi), IL-17 (IL-17i), IL-12/23 (IL-12/23i), and newly developed oral targeted medications (PDEA-4) such an inhibitor of Janus kinase (JAK)/signal transducer and activator of transcription (STAT). Furthermore, some medications approved for psoriasis have not yet been licensed for PsA. These medications include three IL-23 inhibitors (rizankizumab, tildrakizumab, and guselkumab) and an IL-17 receptor blocker (brodalumab), both of which are anticipated to hit the market shortly (Kao, Lee, Ma, Ker, & Leung, 2023). The dynamic nature of evidence and the swift advancement of medicines present a formidable obstacle to the development of treatment guidelines.

Gout treatment medications include anti-inflammatory and analgesic treatments, as well as uric acid-lowering therapies. During a gout episode, reducing the inflammatory reaction is the main goal of treatment. Traditional analgesics and anti-inflammatory medications include glucocorticoids, NSAIDs, and colchicine. Colchicine is a well-known and efficient medication that is used early in acute gout attacks. It works by attaching itself to β-microtubulin and preventing the polymerization of cytoskeletal microtubules. By impeding the NLPR3 inflammasomes' ability to develop, this activity reduces the inflammatory reaction to gout (Quintana et al., 2023). Colchicine exhibits comparable effectiveness at dosages that are high and low. When the dosage reaches 6 ng/mL, it efficiently reduces early pain, while large doses

may have negative gastrointestinal consequences (Runjic et al., 2022). Typical NSAIDs, like naproxen and indomethacin, work as analgesics and anti-inflammatory agents by blocking cyclooxygenase, which lowers the synthesis of prostaglandins. To avoid further acute gout attacks, NSAIDs and uric acid-lowering medication are commonly used together in clinical practice (Grøvle, Hasvik, Holst, & Haugen, 2022; Hashmi, Hiraj, Saleem, Malik, & Mazari, 2022). Prednisone and triamcinolone acetonide are two examples of glucocorticoids that can be given systemically to treat gout-related inflammatory pain due to their strong anti-inflammatory properties (G. Li et al., 2019). Although glucocorticoids can be injected or used orally to quickly reduce inflammation, care must be taken because of the possibility of drug interactions and the potential harm to ligaments and cartilage from excessive or extended dosages (Sanmartí et al., 2020).

By targeting inflammatory mediators and lowering inflammation and discomfort, the typical pharmaceutical strategy to treat arthritis seeks to avoid joint degeneration (Drosos, Pelechas, & Voulgari, 2020). The current pharmacological medications significantly improve the quality of life and delay the progression of disease; nevertheless, long-term use of these therapies carries the risk of deleterious effects on the cardiovascular and gastrointestinal systems (Domper Arnal, Hijos-Mallada, & Lanas, 2022). As a result, traditional herbal medicines are becoming more and more popular as an alternative to standard therapy. The present trend in favor of natural approaches to managing arthritis is being pushed by the appeal of herbal remedies, which show fewer side effects and greater compatibility with long-term administration when compared to allopathic pharmaceuticals.

Conclusion

In conclusion, this integrative review highlights the critical role of inflammation in the pathogenesis and management of arthritis. The findings emphasize the need for a multifaceted treatment approach that combines conventional therapies with complementary, plant-based options to enhance patient outcomes. While conventional therapies such as NSAIDs, DMARDs, and biologics are effective, they are often associated with adverse effects that limit their long-term use. Plant-based therapies, rich in anti-inflammatory and antioxidant compounds, present promising alternatives with fewer side effects, offering potential for safer long-term management of arthritis. Future research should focus on optimizing these therapies and exploring innovative treatment modalities, including early diagnostic tools, to mitigate disease progression and improve the quality of life for arthritis patients.

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