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Metabolic Syndrome and Inflammatory Diseases: A Comprehensive Review of Mechanisms and Management

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Abstract:

Rheumatic inflammatory diseases not only affect joints and other body systems, but also lead to increased morbidity and death. Because of the etiology and pathophysiology of the disease, chronic inflammation, and the pharmacological treatments used, cardiovascular causes rank among the most common mortality factors in those with these diseases. While rheumatoid arthritis, systemic lupus erythematosus, psoriatic arthritis, and gout show different pathophysiology and symptoms, persistent inflammation is their common pathophysiological feature. Researchers have recently discovered links between several of these diseases and the metabolic syndrome. For several reasons, including its connection with the beginning of cardiovascular disease, the development of a pre-inflammatory condition, treatment choice, and related monitoring, the research of metabolic syndrome in inflammatory rheumatic diseases is important. First discussing the relevance of metabolic syndrome in rheumatic diseases, this review article then goes into great length on every disorder separately. This paper concludes, based on a review of past studies, that abdominal obesity in rheumatoid arthritis and lupus patients, as well as abdominal obesity and high blood pressure in psoriatic arthritis and gout patients, are key parts of metabolic syndrome that need more focus.

Keywords: Metabolic Syndrome, Rheumatoid arthritis, Psoriatic arthritis, Systemic lupus erythematosus, gout

Introduction

Apart from influencing internal organs and joints, rheumatic inflammatory diseases also correlate with higher morbidity and mortality (1, 2). Due to the causes and effects of the disease, ongoing inflammation, and the medications used, heart-related issues are among the most common reasons for death in people with these diseases. Although rheumatoid arthritis, systemic lupus erythematosus, psoriatic arthritis, and gout show different pathophysiology and symptoms, persistent inflammation remains their common pathophysiological characteristic (4). Researchers have recently discovered links between several of these diseases and the metabolic

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syndrome. Researching metabolic syndrome in inflammatory rheumatic diseases is important for several reasons, including its connection to the onset of cardiovascular disease, the development of a pre-inflammatory condition, treatment choices, and related monitoring. This review article first explores the relevance of metabolic syndrome (5). With their increasing incidence, cardiovascular diseases and stroke rank among the main causes of death worldwide (6). Age, sex, hypertension, diabetes, smoking, and hyperlipidemia are the recognized risk factors for cardiovascular disease; yet, chronic inflammation has lately been identified as another risk factor (7). Inflammatory rheumatic disorders correlate with an elevated risk of cardiovascular mortality, maybe attributable to chronic inflammation, pharmacological interventions, or alterations in physical activity resulting from pain or mobility restrictions. The reasons for the increased frequency of atherosclerosis risk factors and metabolic syndrome in people with rheumatic diseases are yet unknown (8). Organ involvement, including renal failure, drug-induced nephropathy, and prolonged glucocorticoid therapy, is addressed in general terms within rheumatic illnesses, followed by a detailed examination of each condition individually (9). This study concludes, through a review of prior research, that abdominal obesity in rheumatoid arthritis and lupus patients, abdominal obesity and hypertension in psoriatic arthritis patients, and hypertriglyceridemia and hypertension in gout are significant elements of metabolic syndrome that necessitate increased focus. Calcineurin inhibitors and nonsteroidal anti-inflammatory medications may contribute to the onset of hypertension, poor glucose tolerance, and obesity (10). Metabolic syndrome (Met S) is a widespread inflammatory condition marked by a group of heart disease risk factors, such as high cholesterol, obesity, high blood sugar, and high blood pressure, and its occurrence is increasing around the world. This condition is a significant predictor of type 2 diabetes, stroke, and cardiovascular disease (11). Metabolic syndrome can cause the body to become resistant to insulin by increasing the release of inflammatory substances, such as interleukin-6 and tumor necrosis factor (TNF-α). Individuals with metabolic syndrome exhibit elevated levels of CRP, IL-1b, IL-1 RA, P-selectin, adhesion molecules, and leptin (14). Visceral adiposity can stimulate the synthesis of IL-6, TNF-α, and adiponectin. Chronic rise of CRP in inflammatory rheumatic disorders correlates with heightened cardiovascular disease risk (15). Metabolic syndrome has been demonstrated to correlate with modest levels of chronic inflammation (16). The prevalence of metabolic syndrome in Europe is estimated to be 15.7% in men and 2.14% in women, 34% in America, and 10-20% in Asia (17). Multiple definitions of metabolic syndrome have been suggested, with the two most prevalent being the Adult Treatment Panel III (ATP) and the International Diabetes Federation (IDF) (18). As per the National Cholesterol Education Program (NCEP ATP III) criteria, an individual is classified as having metabolic syndrome if they exhibit three or more of the following characteristics: 1. Central obesity (waist circumference above 102 cm in males and 88 cm in females). 2. Triglyceride levels beyond 150 mg/dl. 3. High-density lipoprotein (HDL) cholesterol levels below 40 mg/dl in men and 50 mg/dl in women (19). An individual's blood pressure surpasses 130/85 mm Hg, or they are taking antihypertensive medication. A fasting blood glucose level exceeding 100 mg/dl or the management of diabetes (20) are also considered risk factors. Comprehending the metabolic syndrome and its elements in rheumatic diseases can facilitate the prioritization of patient assessments, enabling rheumatologists to not only provide prompt diagnosis and treatment of these diseases but also mitigate the cardiovascular complications associated with them through suitable diagnostic and therapeutic interventions and essential recommendations (21, 22). This article examines the frequency of metabolic syndrome and its components across four distinct diseases: rheumatoid arthritis, systemic lupus erythematosus, psoriatic arthritis, and gout.

Rheumatoid arthritis

An autoimmune disease linked with increased risk of cardiovascular death is rheumatoid arthritis (RA). Over half of deaths in those with rheumatoid arthritis (RA) are related to cardiovascular diseases (CVD). Those with rheumatoid arthritis show a mortality rate 1.5 to 1.6 times higher than that of the generaln (23). While the causes of death are similar to those of the general population, rheumatoid arthritis causes show earlier stages in those affected (24). Cardiovascular disease presents in this regard as well. Patients with rheumatoid arthritis have a 1.63 times higher risk of myocardial ischemia and fatal myocardial infarction than do healthy people. Forty to fifty percent of deaths in rheumatoid arthritis patients are related to cardiovascular diseases (25). In rheumatoid arthritis patients, several factors increase cardiovascular risk, including a sedentary lifestyle, disease-modifying treatments, and ongoing inflammation. With a range of 14.32% to 37.83% (26), the frequency of metabolic syndrome in rheumatoid arthritis patients is estimated to be 30.65%. Recent studies on metabolic syndrome in rheumatoid arthritis (RA) have clarified the complicated interactions among theses (27). One study looked at

rheumatoid arthritis and a raised risk of metabolic syndrome and found no appreciable correlation. Furthermore, a lack of any clear correlation between RA's activity level and the likelihood of metabolic syndrome was found. This implies that, in spite of RA's systemic inflammatory character, it might not independently increase the risk of metabolic syndrome, so contradicting some earliers (29). By contrast, another study found interesting results among male RA patients, who showed a lower incidence of hyperglycemia and dyslipidemia relative to a control group (30). This result suggests possible gender-specific variations in the expression of RA and related metabolic disorders, which calls for more investigation on the hormonal and biological processes behind theses (31). Another study concentrated especially on RA female patients' sensitivity to metabolic syndrome. Results indicated that thirty-two percent of the subjects had metabolic syndrome (33). The study did not, however, find a clear relationship between disease activity and metabolic syndrome occurrence. This draws attention to the possible influence on the development of metabolic syndrome in this population of other elements, including age, lifestyle, genes, or long-term medication usage. Notwithstanding the conflicting results, some general trends became clear (33). Researchers noted that patients with either active or chronic rheumatoid arthritis were more likely to have elevated blood glucose levels. Furthermore, linked to metabolic syndrome seemed to be disease activity and functional disability. These findings highlight the need to keep metabolic health under observation in RA patients since resolving metabolic problems could help to achieve better general results (34). All taken together, this body of studies emphasizes the need for customized strategies for controlling RA and its metabolic comorbidities. Future research should try to pinpoint the processes behind these correlations and investigate treatments aimed at both inflammation and metabolic health concurrently (35). Another autoimmune condition linked with cardiovascular morbidity is systemic lupus erythematosus (1). SLE is characterized by extensive organ involvement and a notable prevalence of metabolic syndrome, which is considered a pro-inflammatory disorder that exacerbates morbidity and mortality concerns (37). With women's cardiovascular morbidity risk 5–6 times higher than that of the general population, cardiovascular disease clearly contributes to death in SLE patients (38). Among those between the ages of 35 and 44, this risk could rise by as much as 50-fold. Although the first phase of the disease has improved survival rates, cardiovascular disease still ranks highest among SLE sufferers (39). Although SLE patients are young, many show cardiovascular risk factors, including dyslipidemia, hypertension, and high steroid use, which increases the risk of atherosclerosis (40). Since inflammation is a defining feature of atherosclerotic plaques, there is a significant correlation between systemic inflammation and sensitivity to cardiovascular disease (41). Oxidized low-density lipoprotein (OxLDL) plays an important role in the development of atherosclerosis by triggering immune responses and inflammation in atherosclerotic plaques. Such inflammation could thus compromise processes of endothelial integrity and repair. For SLE patients, autoantibodies, disease activity, clinical symptoms, drugs, and treatments aimed at dyslipidemia and hypertension help explain cardiovascular involvement (43). Glucocorticoids are complexly involved in the pathogenesis of metabolic syndrome in systemic lupus erythematosus. Because of their anti-inflammatory action, modest doses of glucocorticoids may improve vascular function (45). Still, at high doses especially with pulse corticosteroid treatment they could cause metabolic issues, including the start of metabolic syndrome. By lowering inflammatory levels, recent studies show that antimalarial medications including hydroxychloroquine may lower the risk of atherosclerosis in persons with systemic lupuss (46). Moreover, hydroxychloroquine has shown effectiveness in controlling diabetes and dyslipidemia, which highlights its potential ability to prevent cardiovascular events (47). Metabolic Syndrome and RAMobini et al. (2023) looked at how active rheumatoid arthritis (RA) affects metabolic syndrome and found that both active and long-term RA were connected to higher blood sugar levels, but they did not find a strong link between how long someone has been sick and metabolic syndrome. High-dose corticosteroid treatment in patients with systemic lupus erythematosus is associated with a clear increase in the risk of metabolic syndrome and cardiovascular disease (48). The effects of corticosteroids on glucose metabolism, lipid profiles, and blood pressure key components of metabolic syndrome probably explain the higher risk (49). Extended corticosteroid treatment may aggravate these effects, thus stressing the need for careful management and control of SLE patients getting this treatment (50). On the other hand, studies show that in patients with systemic lupus erythematosus (1, 51), the combination of glucocorticoids and hydroxychloroquine could have protective effects against cardiovascular disease. This combined treatment seems to reduce systemic inflammation, a main cause of vascular damage and cardiovascular risk in SLE. Furthermore, hydroxychloroquine could improve vascular health by enhancing endothelial function and reducing thrombotic risk, thereby augmenting the therapeutic effects of glucocorticoids (51).

These results highlight the need to modify therapy strategies to balance the control of SLE symptoms with the lowering of long-term risks, including metabolic syndrome and cardiovascular diseases (52). Combining treatment with glucocorticoids and hydroxychloroquine offers a fantastic chance to improve patient outcomes and lower side effects. To improve dosing strategies and probe the basic mechanisms of various medications to increase their potency, further research is required (53). These updates highlight the ongoing research into how autoimmune diseases, like rheumatoid arthritis and systemic lupus erythematosus, interact with metabolic syndrome, heart disease risk factors, and the effects of different treatment strategies.

Psoriatic arthritis

With an incidence of 0.1 to 1.23 per 100,000 individuals depending on the studied population (55), psoriatic arthritis (PsA) is an inflammatory disease affecting both the skin and joints. Although later studies revealed significant differences between psoriasis and psoriatic arthritis with MetS (56), an initial association between psoriasis and metabolic syndrome (MetS) was observed. Studies indicate that those with PsA have a much higher incidence of MetS than those with psoriasis alone. Studies indicate that patients with psoriatic arthritis (PsA) have a much higher incidence of obesity and hypertension; the prevalence of metabolic syndrome (MetS) ranges between 23.5% and 58.1% among this population (57). Moreover, relative to psoriasis patients, PsA patients often have elevated intima-media thickness (IMT) in the carotid artery, a sign of atherosclerosis, implying an increased risk for cardiovascular disease (CVD). One interesting finding is the correlation between metabolic syndrome (MetS) and this higher cardiovascular risk (59), a main cause of death in persons with psoriatic arthritis (PsA), and the elevated risk of cardiovascular disease (CVD). PsA patients show higher carotid IMT than those with psoriasis; thus, the treatment of MetS is crucial for both the prevention of cardiovascular effects and the control of disease activity (60). All of which increase the risk of myocardial infarction and stroke. An increasing corpus of research has underlined the importance of inflammation in the beginning of insulin resistance, endothelial dysfunction, and atherosclerosis in PsA (61). Individuals with psoriatic arthritis (PsA) use glucocorticoids and hydroxychloroquine than those with rheumatoid arthritis (RA) and systemic lupus erythematosus (1, 62). Many PsA patients show hyperuricemia, which could affect the differences in MetS and cardiovascular complications connected to PsA, RA, and SLE. Furthermore, obesity is acknowledged as a major risk factor for metabolic syndrome in psoriatic arthritis patients. Metabolic Syndrome (MetS) affects more than one-third of patients with Psoriatic Arthritis (PsA), which is significantly higher than its prevalence in the general population (10). Many important studies have helped us to better understand how PsA and MetSt (63). Research shows that patients with PsA show a significantly higher prevalence of MetS than the general population; MetS is mostly driven by obesity, hypertension, and dyslipidemia, so increasing the risk of cardiovascular diseases (64). Furthermore, studies show that those with PsA and MetS are more likely to have type 2 diabetes and atherosclerosis, which emphasizes the need to control MetS in PsA patients to lower these risks risks (65). Since obesity aggravates disease activity and metabolic dysfunction, it is clearly a major factor in the development of MetS in PsA patients (66). Targeting inflammatory cytokines (e.g., TNF-α, IL-6, IL-17) associated with PsA has highlighted their role in insulin resistance and metabolic dysfunction, suggesting that targeting these cytokines may help reduce the negative effects experienced by patients with PsA. Offering a potential therapeutic approach for lowering MetS in PsA, anti-TNF drugs have shown improvements in disease activity and metabolic parameters, including declines in visceral fat and improved lipid profiles. Studies on individuals with PsA have also shown higher carotid intima-media thickness (IMT), so highlighting the link between inflammation and cardiovascular risk. For patients with PsA and MetS, statin drugs have been shown to lower inflammation and enhance lipid profiles, thereby reducing cardiovascular risk. Combining pharmacological interventions with lifestyle changes has produced improvements in PsA disease activity as well asS (69). Early cardiovascular screening and treatment of MetS (69) are especially important since individuals with PsA and MetS show a much higher incidence of cardiovascular events than the general population. While helping with disease symptoms and metabolic health, biologic treatments, like IL-17 inhibitors, have been effective in lowering insulin resistance and belly fat. Using methotrexate along with biologics—especially TNF inhibitors—has been found to better control both the disease and metabolic issues compared to using methotrexate alone. Furthermore, important in the management of PsA and Mets is the success of biologic treatments targeted against IL-12/23 in improving cardiovascular risk factors and disease activity (71). Studies have shown that PsA patients have more MetS, linked to a higher risk of cardiovascular events. The prompt commencement of anti-inflammatory therapies and cardiovascular risk management has

demonstrated efficacy in improving patients (72). These data indicate a conclusive association between PsA and MetS, with inflammation serving a pivotal role in metabolic impairment. Efficient management of these disorders, especially with biological medicines, is essential for enhancing patient outcomes and mitigating cardiovasculars (73). A multidisciplinary strategy, encompassing regular cardiovascular evaluations, anti-inflammatory therapies, and lifestyle alterations, is essential for enhancing therapy for PsA patients withS (74).

Gout

Monosodium urate (MSU) crystals deposited in soft tissues and joints cause gout, an inflammatory arthritis marked by periods of severe pain and inflammation. Hyperuricemia, resulting from serum uric acid concentrations exceeding the MSU crystal solubility limit, intimately relates to it (75). Between 0.1% and 0.10% of the population is the estimated global frequency of gout; men, particularly middle-aged and elderly people (76), show higher rates. Apart from its typical inflammatory symptoms, gout is sometimes linked with metabolic disorders including obesity, dyslipidemia, hyperglycemia, and hypertension, so generating metabolic syndrome (MetS). With rates between 30% and 82% and over 70% of these people displaying at least two components of metabolic syndrome (77), recent studies have confirmed the great frequency of metabolic syndrome (MetS) among gout patients. Gout is closely associated with metabolic syndrome, in which case endothelial dysfunction greatly increases the cardiovascular risk associated with the condition. Raised uric acid levels compromise arterial function and reduce nitric oxide availability, so increasing the risk of atherosclerosis and cardiovascular events (78). In patients with gout, cardiovascular disease remains a main factor causing morbidity and death. Relative to the general population, studies indicate that those with gout and metabolic syndrome have a significantly higher risk of cardiovascular events, including myocardial infarctions and cerebrovascular accidents (79). Recent studies have looked at how obesity might affect gout onset and related metabolic issues. While also encouraging insulin resistance and dyslipidemia, abdominal obesity significantly increases the likelihood of hyperuricemia and gout flare-ups (80). Research has shown a stronger connection between gout and insulin resistance by finding that people with gout have higher rates of insulin resistance and highlighting how hyperuricemia contributes to both gout and metabolic syndrome.

Recent research has focused most attention on the effects of urate-lowering treatments on metabolic syndrome in gout sufferers. By focusing on important parts of metabolic syndrome, allopurinol, which is a xanthine oxidase inhibitor, has been effective in improving blood fat levels, lowering blood pressure, and reducing insulin resistance. Prolonged treatment with urate-lowering agents has been associated with reduced central adiposity, highlighting these agents' ability to address metabolic issues in gout sufferers (83). Inflammatory cytokines are crucial in the link between gout and metabolic syndrome. In gout patients with metabolic syndrome, increased concentrations of pro-inflammatory cytokines, such as TNF-α and IL-6, aggravate systemic inflammation, insulin resistance, and endothelial dysfunction, thereby increasing cardiovascular and metabolic risks. Emphasizing these inflammatory pathways provides a potential course of treatment (84). All told, gout and metabolic syndrome have common risk factors, including hyperuricemia, obesity, insulin resistance, and dyslipidemia. People with these diseases run a greater chance of having heart problems (85). Particularly allopurinol, urate-lowering drugs offer two benefits in the treatment of gout and in the correction of metabolic disorders. Effective treatment depends on changes in lifestyle, including alcohol moderation and weight control. We should create more personalized treatment plans by conducting more research on how gout and metabolic syndrome are connected, looking at the long-term effects of urate-lowering treatments, and addressing differences in how often and severely these conditions occur in various groups. With an incidence of 0.1 to 1.23 per 100,000 individuals, depending on the studied population (55), psoriatic arthritis (PsA) is an inflammatory disease affecting both the skin and joints. Although later studies revealed significant differences between psoriasis and psoriatic arthritis with MetS (56), an initial association between psoriasis and metabolic syndrome (MetS) was observed. Studies indicate that those with PsA have a much higher incidence of MetS than those with psoriasis alone. Studies indicate that patients with psoriatic arthritis (PsA) have a much higher incidence of obesity and hypertension; the prevalence of metabolic syndrome (MetS) ranges between 23.5% and 58.1% among this population (57). Additionally, compared to patients with psoriasis, those with Psoriatic Arthritis (PsA) often have thicker walls in the carotid artery, which is a sign of atherosclerosis and suggests a higher chance of heart disease. One interesting finding is the correlation between metabolic syndrome (MetS) and this higher cardiovascular risk (59), a main cause of death in individuals with psoriatic arthritis (PsA), and the elevated risk of cardiovascular disease (CVD). PsA patients show higher carotid IMT than psoriasis patients; thus, the treatment of MetS is crucial for both the control of disease activity

and the avoidance of cardiovascular effects (60). All of which increase the risk of myocardial infarction and stroke. An increasing corpus of research has underlined the importance of inflammation in the beginning of insulin resistance, endothelial dysfunction, and atherosclerosis in PsA (61). Individuals with psoriatic arthritis (PsA) use glucocorticoids and hydroxychloroquine than those with rheumatoid arthritis (RA) and systemic lupus erythematosus (1, 62). Many PsA patients show hyperuricemia, which could affect the differences in MetS and cardiovascular complications connected to PsA, RA, and SLE. Furthermore, obesity is acknowledged as a major risk factor for metabolic syndrome in psoriatic arthritis patients. Metabolic Syndrome (MetS) affects more than one-third of patients with Psoriatic Arthritis (PsA), which is significantly higher than its prevalence in the general population (10). Many important studies have helped us to better understand how PsA and MetSt (63). Research shows that patients with PsA show a significantly higher prevalence of MetS than the general population; MetS is mostly driven by obesity, hypertension, and dyslipidemia, so increasing the risk of cardiovascular diseases (64). Furthermore, studies show that individuals with PsA and MetS are more likely to develop type 2 diabetes and atherosclerosis, highlighting the necessity of managing MetS in PsA patients to reduce these risks. Since obesity aggravates disease activity and metabolic dysfunction, it is clearly a major factor in the development of MetS in PsA patients (66). Targeting inflammatory cytokines (e.g., TNF-α, IL-6, IL-17) associated with PsA has highlighted their role in insulin resistance and metabolic dysfunction, suggesting that targeting these cytokines may help reduce the negative effects experienced by patients with PsA. Offering a potential therapeutic approach for lowering MetS in PsA, anti-TNF drugs have shown improvements in disease activity and metabolic parameters, including decreases in visceral fat and improved lipid profiles. Studies on individuals with PsA have also shown higher carotid intima-media thickness (IMT), so highlighting the link between inflammation and cardiovascular risk. For patients with PsA and MetS, statin drugs have been shown to lower inflammation and enhance lipid profiles, thereby reducing cardiovascular risk. Combining pharmacological treatments with lifestyle changes has produced improvements in both PsA disease activity andS (69). Early cardiovascular screening and treatment of MetS (69) are especially important since individuals with PsA and MetS show a much higher incidence of cardiovascular events than the general population. Biologic treatments, like IL-17 inhibitors, not only help with disease activity and metabolic health but also effectively lower insulin resistance and belly fat. Using methotrexate along with biologics especially TNF inhibitors has been found to better control both the disease and metabolic issues compared to using methotrexate alone. Additionally, biologic treatments aimed at IL-12/23 play a key role in managing PsA and MetS by improving heart health and reducing disease symptoms. Studies have shown that PsA patients have more MetS, linked to a higher risk of cardiovascular events. Early starting of anti-inflammatory treatments and cardiovascular risk control has shown effectiveness in enhancing patient outcomes (72). These results show a clear correlation between PsA and MetS; inflammation is clearly important for metabolic damage. Improving patient outcomes and lowering cardiovascular risks depend on effective management of these diseases, particularly with regard to biological treatments (73). A multidisciplinary approach that includes frequent cardiovascular assessments, anti-inflammatory treatments, and lifestyle changes is indispensable for improving therapy for patients with PsA (74).

Discussion

MetS and inflammatory rheumatic diseases, like rheumatoid arthritis (RA), systemic lupus erythematosus, psoriatic arthritis (PsA), and gout, are connected in a complex way influenced by several key factors. These cover patient demography, treatment approaches, disease activity, and chronic inflammation. Numerous studies over the past ten years have looked at the frequency of MetS in different diseases, pointing up a higher risk of cardiovascular disease (CVD) and related effects relative to the general population (88). Reported to be 54.5%, the incidence of metabolic syndrome (MetS) in rheumatoid arthritis (RA) varies depending on demographic factors, diagnostic criteria, and disease length (89). Recent studies indicate that over 40% of rheumatoid arthritis sufferers have metabolic syndrome, more commonly diagnosed in women than in men, so highlighting gender-specific differences in the metabolic effects of the condition (89). Common MetS elements seen in RA patients are abdominal obesity, hypertriglyceridemia, and hypertension. TNF-α and IL-6 are among the pro-inflammatory cytokines that significantly contribute to the development of insulin resistance and increase the risk of atherosclerosis, which can lead to cardiovascular events (11). Some rheumatoid arthritis medications, such as methotrexate and hydroxychloroquine, have shown the capacity to improve lipid profiles and reduce reliance on corticosteroids, which are acknowledged to aggravate metabolic syndrome (90). This

emphasizes the effectiveness of anti-inflammatory drugs in correcting metabolic abnormalities and reducing cardiovascular risks in rheumatoid arthritis, thereby improving overall disease management (91). With over 50% of SLE patients diagnosed, the prevalence of metabolic syndrome (MetS) in systemic lupus erythematosus is much raised, a rate much higher than that of the general population (92). Corticosteroids, which are commonly used to treat SLE, significantly worsen central obesity, insulin resistance, and dyslipidemia, leading to the development of metabolic syndrome (MetS). The development of Metabolic Syndrome in Systemic Lupus Erythematosus is connected to higher levels of cytokines, such as TNF-α, IL-6, and IFN-α. These cytokines cause insulin resistance as well as cardiovascular disease. Targeting IFN-α signaling may help those with SLE (93) improve cardiovascular outcomes and reduce the incidence of MetS. With prevalence rates between 38% and 55%, psoriatic arthritis (PsA) is an inflammatory disorder intimately associated with metabolic syndrome (MetS). Common MetS in PsA are abdominal obesity, hypertriglyceridemia, and hypertension, all of which greatly raise cardiovascular risk (94). Important in both insulin resistance and endothelial dysfunction, inflammatory cytokines, including IL-17 and IL-23, help explain metabolic syndrome in psoriatic arthritis. Particularly in those with great skin involvement, therapeutic approaches targeted at these cytokines may have benefits by reducing inflammation and metabolic malfunction (95). In gout, an inflammatory condition brought on by urate crystal accumulation, elements of MetS are rather common. Often occurring in gout sufferers are abdominal obesity, hypertriglyceridemia, and hypertension; this condition is linked to a higher risk of cardiovascular events, particularly in those with comorbidities like obesity and hypertension (96). Allopurinol and other urate-lowering drugs enhance endothelial function, thereby lowering cardiovascular risk; however, further long-term research is necessary. Although its modes of action are yet under investigation, colchicine, usually used in the treatment of gout, has shown the potential to lower cardiovascular events by reducing systemic inflammation (97). Sociodemographic factors, including age, sex, ethnicity, and socioeconomic level, significantly influence the frequency and severity of MetS in individuals with inflammatory rheumatic diseases, in addition to disease-specific elements. People from lower socioeconomic levels are more likely to have metabolic syndrome, most likely due to poor eating habits, limited access to healthcare, and less physical activity (98). In inflammatory rheumatic disease, the frequency of MetS far exceeds that in the general population. In these individuals, chronic inflammation, corticosteroid use, and disease-associated factors raise the risk of metabolic disorders and cardiovascular disease (99). Essential for risk reduction is proactive screening for metabolic syndrome and effective management of inflammation with medications including methotrexate, hydroxychloroquine, and urate-lowering drugs. Later research should focus on the basic processes linking these diseases with MetS, especially the roles of cytokines and biomarkers as future therapy targets. Understanding the influence of sociodemographic factors on MetS prevalence and enabling the development of tailored care plans for different populations depends on knowledge of regional studies (100).

Conclusion

By monitoring the state and relevance of comorbidities and applying appropriate treatments to control them, one can assume a preventive role in the complications and mortality associated with the disease. There is a connection between inflammatory rheumatic diseases and a higher risk of cardiovascular disease. This balances out the joint inflammation participation. If efficient treatments for rheumatic diseases that reduce inflammation are available, patients could have longer lifespans and better qualities of life. Given the prevalence of metabolic syndrome, these drugs could also prove helpful. This review study highlights that important parts of metabolic syndrome needing more focus are belly fat in patients with rheumatoid arthritis and lupus, belly fat and high blood pressure in patients with psoriatic arthritis, and high triglycerides and high blood pressure in gout. The analysis of earlier studies helped one reach conclusions. The higher prevalence of metabolic syndrome in gout and psoriatic arthritis patients suggests that these people need more focus on the elements raising their risk of cardiovascular disease. Knowing the elements of metabolic syndrome connected to every disorder helps you choose the appropriate treatment.

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