Autumn 2025, Volume 10, Issue 39 (25- 35) DOI: 10.22034/pmj.2025.2050171.1051

Personalized & Precision Medicine Journal

Homepage: www.pmjournal.ir

Metabolic Syndrome and Inflammatory Diseases: An Elaborate Review of Mechanisms and Management

Akram Sadat Ahmadi [1,*], Atefeh Valaei [2]

[1]**Department of Virology, School of Public Health, Tehran University of Medical Sciences, Tehran, 1417613151, Iran**

[2]**Molecular Medicine Department, Biotechnology Research Centre of Pasteur Institute of Iran**

ARTICLE INFO

Paper Type: Review Article

Submitted: 2025-07-14
Accepted: 2025-12-03

Keywords:
Metabolic Syndrome
Psoriatic arthritis
Systemic Lupus Erythematosus
Gout

Corresponding author:
Akram Sadat Ahmadi
Email: Akramsadat.ahmadi@gmail.com

ABSTRACT

Rheumatic inflammatory diseases, besides affecting joints and other bodily systems, are linked to heightened mortality and morbidity. Cardiovascular reasons are among the most prevalent mortality factors in individuals with these disorders, attributable to the disease's etiology and pathophysiology, chronic inflammation, and the pharmacological treatments employed. Although rheumatoid arthritis, systemic lupus erythematosus, psoriatic arthritis, and gout exhibit distinct pathophysiology and symptoms, persistent inflammation remains their shared pathophysiological characteristic. Metabolic syndrome has recently been linked to several of these disorders. The investigation of metabolic syndrome in inflammatory rheumatic diseases is significant for multiple reasons, including its correlation with cardiovascular disease onset, the emergence of a pre-inflammatory condition, treatment selection, and associated monitoring. This review article initially explores the significance of metabolic syndrome in rheumatic diseases, followed by a detailed analysis of each condition individually. This study concludes, through a review of previous studies, 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 warranting increased focus.

How to Cite this Article:

A.S. Ahmadi, A. Valaei. "Metabolic Syndrome and Inflammatory diseases: An Elaborate Review of Mechanisms and Management" Personalized & Precision Medicine Journal, Vol. 10, no. 39, pp. 25- 35.

INTRODUCTION

Rheumatic inflammatory diseases, in addition to affecting joints and other internal organs, are associated with mortality and morbidity (1, 2). One of the most widespread causes of death in people with these disorders is cardiovascular, which occurs due to the etiology and pathophysiology of the disease and chronic inflammation, as well as the pharmacological therapy administered (3). Despite the different pathophysiology and symptoms of rheumatoid arthritis systemic lupus erythematosus, psoriatic arthritis and gout, persistent inflammation is their common pathological feature (4).

Metabolic syndrome has been just recently identified across several of these disorders. The study of metabolic syndrome in inflammatory rheumatic conditions has several important issues, such as an association with cardiovascular diseases development and pre-inflammatory disease, a choice of approach to treatment, and monitoring. This review paper first examines the meaningfulness of metabolic syndrome (5).

Cardiovascular diseases and stroke are some of the top causes of death worldwide, and the occurrence of this disease is on the increase (6). Known risk determinants in cardiovascular disease include age, sex, high blood pressure, diabetes, smoking and high levels of cholesterol; however, chronic inflammation

Authors retain the copyright and full publishing rights.
Published by AmitisGen TECH Dev Group. This article is an open access article licensed under the Creative Commons Attribution 4.0 International (CC BY 4.0)

has of late been identified as another risk factor (7). Inflammatory rheumatic diseases correlate with an excess mortality risk as a result of cardiovascular disease and, possibly as a consequence of chronic inflammation, treatment, or a change in physical performance due to pain or the inability to move.

The reasons behind the increased prevalence of atherosclerosis risk factors and metabolic syndrome among rheumatic diseases individuals are poorly understood (8). Renal involvement, such as renal failure or drug-induced renal effects, and long-term glucocorticoid use are covered in the generality of rheumatic illnesses, and finally each condition is looked at in detail (9). This paper finds, by reviewing the literature that exists on the topic, that abdominal obesity in rheumatoid arthritis and lupus patients and in the case of psoriatic arthritis patients, abdominal obesity and hypertension are important aspects of the metabolic syndrome that require more attention. Evaluating hypertriglyceridemia and hypertension in the case of gout is also an important component of metabolic syndrome requiring more attention. Such drugs as calcineurin inhibitors and nonsteroidal anti-inflammatory agents may also lead to the development of hypertension, poor glucose tolerance, and obesity (10).

Metabolic syndrome (Met S) is a condition with proinflammatory features where a group of cardiovascular risk factors, such as hyperlipidemia, obesity, elevated fasting glucose, and hypertension occur, and its prevalence is increasing worldwide. This situation strongly predicts type 2 diabetes, stroke and cardiovascular disease (11).

Metabolic syndrome is capable of causing insulin resistance through the secretion of inflammatory cytokines such as interleukin-6 and tumor necrosis factor (TNF-alpha)(12, 13). Patients with metabolic syndrome have increased L-selectin, thrombospondin, IL-1b, IL-1 RA, CRP, leptin, and P-selectin (14). Visceral adiposity has been observed to induce IL-6, TNF-alpha and adiponectin production. Elevated increases of CRP in rheumatic inflammatory disorders associate to increased risk to vascular diseases (15). It has been shown that metabolic syndrome is associated with modest chronic inflammation (16). Metabolic syndrome prevalence has been projected to be 15.7 in men and 2.14 in women across the European continent, 34 percent across the American region, and 10-20 percent across Asia (17). Several definitions of metabolic syndrome suggested, and the two most common are those of Adult Treatment Panel III (ATP) and those of International Diabetes Federation (IDF) (18).

According to the National Cholesterol Education Program NCEP ATP III definition, a patient is considered to have metabolic syndrome when he or she possesses three or more of the following conditions: 1- waist circumference greater than 102 cm in men and 88 cm in the case of women, 2- triglyceride levels more than 150 mg/dl, 3- HDL-cholesterol levels below 40 mg/dl in men and 50 mg/dl in women (19). High blood pressure of more than 130/85 mm Hg or taking an antihypertensive drug. Fasting glucose level is more than 100 mg/dl or treatment of diabetes (20). Understanding the metabolic syndrome and its breakdown in rheumatic diseases can help prioritize patient assessments to guide rheumatologists in not only early diagnosis and treatment of rheumatic syndromes, but also secondary prevention of the cardiovascular complications that accompany such diseases by providing suitable diagnostic interventions and prescriptions and appropriate guidelines (21, 22). This paper reviews the prevalence of metabolic syndrome and its individual aspects in the four unique conditions including rheumatoid arthritis, systemic lupus erythematosus, psoriatic arthritis and gout.

Rheumatoid arthritis

Rheumatoid arthritis (RA) is an autoimmune disease, which is characterized by increased cardiovascular mortality (23). CVD constitutes more than 50 percent of deaths in rheumatoid arthritis patients. Mortality is also 1.5 and 1.6 times higher in individuals with rheumatoid arthritis as compared to that of the general population (23). Whereas the etiology of death is similar to that of the general population, cardiovascular disease occurs at an earlier age in patients with rheumatoid arthritis (24). The probability of myocardial ischemia and lethal myocardium inflection is 1.63 in rheumatoid arthritis patients in comparison to healthy individuals. Cardiovascular diseases result in the death of 40-50 percent of patients with rheumatoid arthritis (25). Several factors, including long-standing inflammation, disease-modifying drugs, and lack of physical activity of rheumatoid arthritis patients, are risk factors to cardiovascular risk. It is estimated that prevalence ofmetabolic syndrome among patients with rheumatoid arthritis is 30.65 percent, with a range of 14.32 to 37.83 percent (26).

Current research on metabolic syndrome in rheumatoid arthritis (RA) have enlightened researchers on the intricate relationships shared between the two diseases (27). In one study, there was also no significant relationship between metabolic syndrome and an elevated probability of rheumatoid arthritis (28). It also found no significant relation between activity level of RA and developing metabolic syndrome. This implies that despite the inflammatory properties of the system, RA does not increase the likelihood of metabolic syndrome, contravening some of the previous assumptions (29). However, in comparison, another

study presented interesting results in male RA patients that showed a lower incidence of hyperglycemia and dyslipidemia in comparison with a control patient group (30). This finding suggests the possible gender-specific differences in the presentation of RA and related metabolic disruptions, which should be further investigated in terms of biological and hormonal processes leading to these facts (31). Another research was conducted on women with RA concerned with their vulnerability to the metabolic syndrome. The results indicated that more than one-half of the interviewees had metabolic syndrome (32). Nevertheless, the research could not provide the definite connection that existed between the disease activity and development of metabolic syndrome. This demonstrates the possible role of other factors, e.g., age, lifestyle, genetics, or the prolonged use of medication, in the pathogenesis of metabolic syndrome in this age group. In spite of the inconsistent results, there were still some common trends (33). It was also noted that patients with active or chronic rheumatoid arthritis have a tendency of having high blood glucose levels. The presence of metabolic syndrome, also, seemed to be correlated with the activity of the disease and functional impairment. These findings have the significance of the need to monitor metabolic health of RA patients, and by addressing metabolic complications, improvements can be made in the overall outcomes (34).

Collectively, they highlight the importance of individualised treatment on metabolic comorbidities of RA. Future research is needed to determine the cause of these associations and whether there are such interventions that can exert effects on both inflammation and metabolic health (35).

A related autoimmune disease, systemic lupus erythematosus (SLE) (1), is also linked to cardiovascular morbidity (36). SLE is characterized by widespread organ involvement and a large burden of metabolic syndrome, a pro-inflammatory condition associated with exacerbation of morbidity and mortality risks (37). Cardiovascular disease is also a major cause of mortality among SLE patients with women having a 5-6fold increased risk of cardiovascular morbidity compared with the general population (38). In women within the age groups of 35-44, the risk can get up to 50 times more. Despite improved survival rates during the earlier stages of the illness, cardiovascular disease is still the cardinal kill of SLE patients (39).

Many young SLE patients have cardiovascular risk features like dyslipidemia, hypertension, and an increased level of steroid use, which increases the risk of atherosclerosis (40). There is a strong correlation between the inflammatory process that takes place in the body and predisposition to cardiovascular disease as inflammation is also a hallmark of atherosclerotic plaques (41). The oxidation of low-density lipoprotein (OxLDL) plays a key role in the development of atherosclerosis contributing to the immunological activation and inflammation of plaques (42). This has the potential to further damage endothelial integrity and repair mechanisms. The contribution of autoantibodies, activity of the disease, clinical manifestations of a patient, the use of drugs, and treatment of the disease related to the hypertension and dyslipidemia facilitate the cardiovascular involvement in SLE patients (43).

Glucocorticoid role in the pathogenesis of metabolic syndrome in systemic lupus erythematosus is complex (44). At low doses glucocorticoids have potential of improving vascular functions due to their anti-inflammatory effects (45). At higher doses, particularly with pulse corticosteroid treatment, however, they can present with metabolic issues, such as the development of metabolic syndrome. Recent research shows that hydroxychloroquine and other antimalarial medication may decrease risk of atherosclerosis development code in patients with systemic lupus erythematosus through lowering level of inflammation (46). What is more is that, hydroxychloroquine has also shown to be effective in the management of diabetes and dyslipidemia, further pointing at the potential use of this drug to prevent cardiovascular events (47).

A research by Mobini et al. (2023) studied the relationship between the rheumatoid arthritis (RA) disease activity and the metabolic syndrome, which indicated that the levels of blood glucose and active RA and chronic RA showed similar results but there was no correlation between the duration of illness and metabolic syndrome (27). There is some correlation between the administration of high-dose corticosteroids in patients with systemic lupus erythematosus and the dramatic increase in the propensity toward metabolic syndrome and cardiovascular disease (48). The elevated risk is likely to rely on the influence of corticosteroids on glucose metabolism, lipid profiles, and blood pressure which are critical aspects of metabolic syndrome (49). Enhanced use of corticosteroids could increase the severity of these effects, therefore, requiring close administration and monitoring of SLE patients undergoing corticosteroid administration (50).

Otherwise, evidence suggests that a combination of glucocorticoids and hydroxychloroquine could have beneficial effects against cardiovascular disease, in patients with systemic lupus erythematosus (1, 51). This combination therapy appears to reduce whole-body inflammation which plays a major role in injury to the vasculature and cardiovascular risk in SLE. Further, hydroxychloroquine can be used to supplement vascularization, improving endothelial performance and reducing the likelihood of thrombosis, thereby supplementing the existing therapeutic enhancements hydroxychloroquine introduces (51).

These results emphasize the importance of

individualizing treatment strategies that can balance the treatment of SLE symptoms with diminishing future risks, such as metabolic syndrome and cardiovascular disease (52). The possibility to combine their use creates a huge potential in terms of improving patient outcomes and decreasing unwanted effects with glucocorticoids and hydroxychloroquine. Further research is needed to optimize dosing regimes and explore the basic mechanisms of these drugs with the aim of maximizing efficacy (53).

These developments also show the ongoing study relating to the complex relationship between autoimmune conditions, e.g., rheumatoid arthritis, and systemic lupus erythematosus, cardiovascular risk factors, metabolic syndrome and the implications of the remedial interventions (54).

Psoriatic arthritis

Psoriatic arthritis (PsA) is an inflammatory disease affecting the skin and joints with an incidence rate of 0.1 to 1.23 per one hundred thousand members of the investigated population, depending on the group of people in question (55). There are a few seminal works that have exhibited an initial enrollment that psoriasis and metabolic syndrome (MetS) are associated; yet, it is revealed that there are significant inconsistencies with relationship between psoriasis and psoriatic arthritis with MetS (56). It has been observed that the prevalence MetS is significantly higher in patients with PsA in comparison to patients with only psoriasis. We note that the risk of obesity and hypertension is significantly increased in Psoriatic Arthritis (PsA) patients, whereas the frequency of Metabolic Syndrome (MetS) estimates to 23.5-58.1% of individuals in this cohort (57).

Moreover, PsA patients often exhibit substantially higher intima-media thickness (IMT) in the carotid artery than psoriasis patients and this is an indicator of atherosclerosis, suggesting an increased risk of cardiovascular disease in PsA subjects (58). A remarkable finding is the increased risk of cardiovascular disease (CVD) which is a leading cause of death in subjects with PsA and the association between metabolic syndrome (MetS) and increased CVD risk (59). Compared to psoriasis, carotid IMT is higher in PsA patients, and MetS management can both help control the disease activity and prevent cardiovascular sequelae (60).

A growing body of research has intrinsically recognized the importance of inflammation in the development of insulin resistance, endothelial dysfunction, and atherosclerosis in PsA, all of which increase risk of myocardial infarction and stroke (61). Hydroxychloroquine and glucocorticoids are less commonly used in patients with psoriatic arthritis (PsA) compared with rheumatoid arthritis (RA) and systemic lupus erythematosus (1, 62). The prevalence of hyperuricemia associated with PsA is markedly high, and it may have contributed to the differences in the risk of MetS and cardiovascular event related to PsA, RA and SLE. Furthermore, it has been identified that obesity is a great contributor to the occurrence of Metabolic Syndrome to Psoriatic Arthritis patients. MetS greatly predominates among patients with PsA compared to the community with more than one out of every three patients affected by MetS (10).

A number of clinical studies have enhanced our understanding of the correlation between PsA and MetS (63). Evidence indicates that patients with PsA have a significantly high prevalence of MetS than the general population with MetS mainly being characterized by obesity, hypertension and dyslipidemia thus increasing the risk of developing cardiovascular diseases (64). Additionally, studies show that PsA patients with MetS face a higher risk of getting type 2 diabetes and atherosclerosis, which further dictates the need to mitigate MetS among PsA patients to minimize these risks (65).

Obesity has been identified as one of the major contributory factors that lead to the manifestation of MetS in PsA patients as it worsens both the disease activity and metabolic disorder (66). Studies related to the pro-inflammatory cytokines (e.g., TNF-alpha, IL-6, IL- 17) implicated with PsA have shown that they are involved in insulin resistance and metabolic dysfunction, and that they can be targeted to diminish the impact of MetS in PsA patients (67). Anti-TNF medications have demonstrated an improvement in disease activity and metabolic disorder, such as significant loss of visceral fat and lipid profile, it may be considered a way of treating MetS in PsA (68).

Agreement has been made with the fact that persons with PsA have elevated carotid intensor media thickness (IMT), which is indicative of the relationship between inflammation and cardiovascular morbiditty. Statin drugs were shown to be able to reduce the cardiovascular risk in terms of improving lipid profile and reducing the inflammation in patients with PsA and Mets (69). Weight loss management strategies including lifestyle changes and medical approaches have led to the improvement of PsA disease activity as well as MetS (69).

Patients with PsA and MetS are at significantly higher risk of cardiovascular events compared with the general population and the need to screen and treat MetS at earlier stages is critical (69). Biologic Therapy, e.g., IL-17 blocking, has been shown to improve insulin resistance and visceral fat content at the expense of disease activity and coverage of the metabolic pathways. The combination with methotrexate and biologics and in particular TNF inhibitors have demonstrated synergy in terms of disease and metabolic control compared to

methotrexate alone (70).

Additionally, biologic therapy that targets IL-12/23 and IL-17 has shown improvement in cardiovascular risk factors and disease activity, and thus their importance in the treatment of both PsA and Mets (71). Studies have proven a larger prevalence of MetS among PsA patients with increased risk of cardiovascular events. The upstream effects of early anti-inflammatory therapies and cardiovascular risk treatment have proved effective in the alleviation of patient outcomes (72).

The evidence therefore points to an irrevocable relationship between PsA and MetS and in this the inflammatory process plays a central role in this metabolic insult. Effective treatment of these conditions particularly using biological drugs is vital in improving patient prognosis and preventing cardiovascular complications (73). A multidisciplinary approach that includes periodic cardiovascular assessment, anti-inflammatory treatment, and lifestyle changes, must be employed to improve treatment in PsA patients with MetS (74).

Gout

Gout is an inflammatory arthritis caused by deposition of monosodium urate (MSU) crystal in joints and soft tissues and resulting in bouts of acute pain and inflammation. It is tightly connected to hyperuricemia that occurs when serum uric acid levels exceed the solubility of the MSU crystals (75). The worldwide incidence of gout is estimated at 0.1-0.10 of the population with high rates in men, particularly those in the middle age and old age categories (76). In addition to the typical inflammatory manifestations, gout is commonly accompanied by metabolic disorders that encompass obesity, dyslipidemia, hyperglycemia, and hypertension and compose metabolic syndrome (MetS). Recent studies provided clear evidence of high proportion of Metabolic Syndrome (MetS) in gout patients that varied between 30 and 82 percent, and approximately 70 percent of the gout patients exhibited two or more aspects of MetS (77).

Gout is closely connected with the metabolic syndrome, whereby the cardiovascular risks associated with a metabolic syndrome are largely due to endothelial dysfunction. Higher levels of uric acid suppress arterial performance and reduce the availability of nitric oxide, therefore, increasing the risk of atherosclerosis and cardiac events (78). Cardiovascular is still a significant cause of morbidity and mortality among patients with gout. Research suggests that patients with gout and metabolic syndrome have a very high chance of cardiovascular complications including myocardial infarctions and cerebrocardiovascular accidents when compared to the normal population (79).

Recent studies have examined the effects that obesity has on the development of gout and the related metabolic issues. Abdominal obesity increases the risk of hyperuricemia and gout exacerbations significantly, as well as promotes insulin resistance and dyslipidemia (80). Studies have further established the relationship between gout and insulin resistance concluding that gout patients have a higher rate of insulin resistance and that hyperuricemia may have a dual role in gout progression and development of metabolic syndrome (81).

Recent investigations have concentrated on the effects of urate-lowering treatments on metabolic syndrome in patients with gout. Allopurinol, an antioxidant acting as a xanthine inhibitor has proved to improve serum lipid levels, lower blood pressure, and prevent insulin resistance hence putting into effect very crucial drivers of metabolic syndrome (82). The prolonged use of urate-lowering drugs has been associated with a lowering of central adiposity, and thus the ability to improve metabolic factors in people with gout (83).

A key element in the correlation between gout and metabolic syndrome is inflammatory cytokines. The elevated levels of pro-inflammatory cytokines, namely TNF-alpha and IL- 6, in gout patients with metabolic syndrome, worsen systemic inflammation, insulin resistance and endothelial functioning therefore increasing the risk of cardiovascular and metabolic insults. Targeting these inflammatory pathways provide a potential method of treatment (84).

In brief, there are shared risk factors between gout and metabolic syndrome including hyperuricemia, obesity, resistance to insulin, and dyslipidemia. People with such diseases face a higher threat of cardiovascular events (85). Urate-lowering therapy, and particularly allopurinol, has the advantageous double effects of reducing gout and restoring metabolic disturbances. Lifestyle changes including weight loss and avoiding alcohol are essential to the treatment. Future studies ought to focus on the molecular mechanisms underlying links between gout and metabolic syndrome, understand the long-term outcomes of urate-lowering treatments, and be able to address shifts in frequency and severity across different populations to develop more individualized treatment plans (86).

DISCUSSION

The relationship between metabolic syndrome (MetS) and inflammatory rheumatologic diseases, such as rheumatoid arthritis (RA), systemic lupus erythematosus (1), psoriatic arthritis (PsA), and gout is complicated and influenced by several major determinants (87). These include chronic inflammations, pathogenic activity, treatment regimens and the demographics of the patients. In the past decade, several studies have explored the prevalence of MetS in other disorders, finding that they carry a

higher CV disease and resultant risks when compared to the general population (88).

Metabolic syndrome (MetS) in rheumatoid arthritis (RA) is estimated at 54.5%, and the prevalence depends on the demographic data, definition, and disease duration (89). Recent reports further demonstrate that more than 40 percent of rheumatoid arthritis cases are diagnosed with metabolic syndrome with a higher incidence in women as compared to men, a demonstration of gender-specific differences in the metabolic impacts of the condition (89). Common factors of MetS in RA patients include increased abdominal obesity, high triglycerides, and blood pressure. Pro-inflammatory cytokine polymerase chain reaction (TNF-alpha and IL-6) is crucial in mediating insulin resistance and increases risk of atherosclerosis and eventual cardiovascular problems (11). Some rheumatoid arthritis medications as methotrexate and hydroxychloroquine have proved to improve the lipids status and reduce reliance on corticosteroids, which are known to aggravate metabolic syndrome (90). This sheds light on the ability of anti-inflammatory drugs to correct metabolic abnormalities and reduce the cardiovascular risks in rheumatoid arthritis thereby providing a better management of the disease as a whole (91).

The rates of MetS are more than twice the highest in SLE patients, with more than 50% developing MetS as compared to those of the general population (92). Although corticosteroids are a major treatment of SLE, their use contributes immensely to development of MetS through the development of central obesity, insulin resistance as well as dyslipidemia. The presence of increased levels of cytokines such as TNF- alpha, IL- 6 and interferon alpha is related to the pathophysiology of Metabolic Syndrome in Systemic Lupus Erythematosus. These cytokines enhance cardiovascular disease and insulin resistance. Inhibiting the immunity of IFN-alpha may dampen the exertion of MetS and promote cardiovascular outcomes in individuals with SLE (93).

Psoriatic arthritis (PsA) is highly correlated with metabolic syndrome (MetS) with a prevalence of between 38&ndashgraved confinetti trifilab in55 percent. The most common MetS factors that are present in PsA include abdominal obesity, hypertriglyceridemia, and hypertension with a significant cardiovascular risk (94). IL-17, IL-23 cytokines play a central role in endothelial dysfunction and insulin resistance and, therefore, they should contribute to the metabolic syndrome in psoriatic arthritis. Targeting these cytokines has potential therapeutic benefits to reduce inflammation and metabolic dysfunction, especially in those with large cutaneous disease burden (95).

The prevalence of components of MetS is high in gout, which is an inflammatory disease caused by the aggregation of urate crystals. Abdominal obesity, hypertriglyceridemia, and hypertension are also common among gout patients and this condition is also associated with an elevated risk of cardiovascular events, particularly among patients with such comorbidities as obesity and hypertension (96). Management of Urate-lowering therapies such as allopurinol, could reduce cardiovascular risk by improving endothelial activity; further long-duration trials are however necessary. Colchicine, a medication which is currently mostly used in the management of gout, has shown the promise of reducing cardiovascular events through the reduction of systemic inflammation, but the processes by which this works are yet to be investigated (97).

As well as disease-specific factors, socio-demographic factors such as age, sex, ethnic, and socio-economic position also have an important impact on prevalence and severity of MetS among patients with inflammatory rheumatic diseases. Patients with low socio-economic status have a high risk of Metabolic Syndrome occurrence, which can be explained by the poorly available healthcare services, the lack of physical potential, and unhealthy nutritional habits (98). Inflammatory rheumatic disease has a higher prevalence of MetS than that of the general population. Chronic inflammation, the use of corticosteroids, and disease related criteria augment the risk of metabolic issues and heart disease in such individuals (99). Actively screening Metabolic Syndrome and proper care management of inflammation with medication like methotrexate, hydroxychloroquine and urate lowing medications are necessary in risk abatement. Future research should focus on the underlying processes that link these conditions to MetS, especially the role of cytokines and biomarkers as future potential targets of treatment. Regional analysis is critical to the understanding of how socio-demographic factors influence the prevalence of MetS and assist with the development of region-specific care plans in different populations (100).

CONCLUSION

Keeping track of the conditions and the usefulness of comorbidities and taking necessary action to control it, it can be presumed that one takes a preventive approach to the complications and loss of life associated with the illness. There is an existing relationship between inflammatory rheumatic diseases and cardiovascular diseases. This accompanies the involvement of the joint inflammation. When efficacious medicines to adjust the inflammation in rheumatic diseases are available, patients can enjoy the enlarged quality of life and increased lifespan. These drugs can also be useful bearing in mind the high prevalence of the metabolic syndrome. The displayed higher incidence

of metabolic syndrome in patients with gout and psoriatic arthritis is an indication that these patients require more emphasis on factors that predispose them to cardiovascular diseases. Recognizing what metabolic syndrome subsets are involved in each individual can help to select the treatment. This review article postulated that the abdominal obesity of patients with rheumatoid arthritis and lupus, abdominal obesity and hypertension of patients with psoriatic arthritis and hypertriglyceridemia and hypertension in gout subjects are important aspects of metabolic syndrome that require heightened attention. The analysis of previous studies was used to draw the conclusions.

Acknowledgements

The researchers are grateful to all the experts and personnel of the Biology Department of Islamic Azad University of Mashhad who have cooperated in this research.

Authors's Contributio

Akram Sadat Ahmadi: Conceptualization, Atefeh Valaei: editing and review. The authors read and confirmed the final manuscript.

Funding

This study is the outcome of self-directed research carried out without any financial assistance.

Ethics approval and consent to participate

Not applicable.

Conflict of Interest

The authors declared no conflict of interest.

Consent for publication

Not Applicable.

REFERENCES

1.Hedar AM, Stradner MH, Roessler A, Goswami N. Autoimmune Rheumatic Diseases and Vascular Function: The Concept of Autoimmune Atherosclerosis. J Clin Med. 2021;10(19).
2.Hayden MR. Overview and New Insights into the Metabolic Syndrome: Risk Factors and Emerging Variables in the Development of Type 2 Diabetes and Cerebrocardiovascular Disease. Medicina [Internet]. 2023; 59(3).
3.Gupta L, Thomas J, Ravichandran R, Singh M, Nag A, Panjiyar BK. Inflammation in Cardiovascular Disease: A Comprehensive Review of Biomarkers and Therapeutic Targets. Cureus. 2023;15(9):e45483.
4.Radu AF, Bungau SG. Management of Rheumatoid Arthritis: An Overview. Cells. 2021;10(11).
5.Rohm TV, Meier DT, Olefsky JM, Donath MY. Inflammation in obesity, diabetes, and related disorders. Immunity. 2022;55(1):31-55.
6.Roth GA, Mensah GA, Johnson CO, Addolorato G, Ammirati E, Baddour LM, et al. Global Burden of Cardiovascular Diseases and Risk Factors, 1990-2019: Update From the GBD 2019 Study. Journal of the American College of Cardiology. 2020;76(25):2982-3021.
7.Alloubani A, Nimer R, Samara R. Relationship between Hyperlipidemia, Cardiovascular Disease and Stroke: A Systematic Review. Current cardiology reviews. 2021;17(6):e051121189015.
8.Alfaddagh A, Martin SS, Leucker TM, Michos ED, Blaha MJ, Lowenstein CJ, et al. Inflammation and cardiovascular disease: From mechanisms to therapeutics. American journal of preventive cardiology. 2020;4:100130.
9.Griffin BR, Faubel S, Edelstein CL. Biomarkers of Drug-Induced Kidney Toxicity. Therapeutic drug monitoring. 2019;41(2):213-26.
10.Li B, Huang H, Zhao J, Deng X, Zhang Z. Discrepancy in Metabolic Syndrome between Psoriatic Arthritis and Rheumatoid Arthritis: a Direct Comparison of Two Cohorts in One Center. Rheumatology and therapy. 2023;10(1):135-48.
11.Jha BK, Sherpa ML, Imran M, Mohammed Y, Jha LA, Paudel KR, et al. Progress in Understanding Metabolic Syndrome and Knowledge of Its Complex Pathophysiology. Diabetology [Internet]. 2023; 4(2):[134-59 pp.].
12.Sethi JK, Hotamisligil GS. Metabolic Messengers: tumour necrosis factor. Nature Metabolism. 2021;3(10):1302-12.
13.Shi J, Fan J, Su Q, Yang Z. Cytokines and Abnormal Glucose and Lipid Metabolism. 2019;10.
14.Welty FK, Alfaddagh A, Elajami TK. Targeting inflammation in metabolic syndrome. Translational research : the journal of laboratory and clinical medicine. 2016;167(1):257-80.
15.Chait A, den Hartigh LJ. Adipose Tissue Distribution, Inflammation and Its Metabolic Consequences, Including Diabetes and Cardiovascular Disease. 2020;7.
16.Monteiro R, Azevedo I. Chronic inflammation in obesity and the metabolic syndrome. Mediators of inflammation. 2010;2010.
17.Sigit FS, Tahapary DL, Trompet S, Sartono E, Willems van Dijk K, Rosendaal FR, et al. The prevalence of metabolic syndrome and its association with body fat distribution in middle-aged individuals from Indonesia and the Netherlands: a cross-sectional analysis of two population-based studies. Diabetology & Metabolic Syndrome. 2020;12(1):2.
18.Dehghani A, Molani-Gol R, Mohammadi-Narab M, Norouzy A, Abolhassani MH, Tabatabaee Jabali SM, et al. The prevalence of obesity and overweight among Iranian population: an umbrella systematic

review and meta-analysis. BMC public health. 2024;24(1):3377.
19.Saif-Ali R, Kamaruddin NA, Al-Habori M, Al-Dubai SA, Ngah WZW. Relationship of metabolic syndrome defined by IDF or revised NCEP ATP III with glycemic control among Malaysians with Type 2 Diabetes. Diabetol Metab Syndr. 2020;12:67.
20.Han K, Park JB. Clinical implication of fasting glucose and systolic/diastolic blood pressure on the prevalence of periodontitis in non-diabetic and non-hypertensive adults using nationally representative data. Experimental and therapeutic medicine. 2018;16(2):671-8.
21.Hallajzadeh J, Safiri S, Mansournia MA, Khoramdad M, Izadi N, Almasi-Hashiani A, et al. Metabolic syndrome and its components among rheumatoid arthritis patients: A comprehensive updated systematic review and meta-analysis. PloS one. 2017;12(3):e0170361.
22.Inchingolo F, Inchingolo AM, Fatone MC, Avantario P, Del Vecchio G, Pezzolla C, et al. Management of Rheumatoid Arthritis in Primary Care: A Scoping Review. International Journal of Environmental Research and Public Health [Internet]. 2024; 21(6).
23.Farhat H, Irfan H, Muthiah K, Pallipamu N, Taheri S, Thiagaraj SS, et al. Increased Risk of Cardiovascular Diseases in Rheumatoid Arthritis: A Systematic Review. Cureus. 2022;14(12):e32308.
24.Hansildaar R, Vedder D, Baniaamam M, Tausche A-K, Gerritsen M, Nurmohamed MT. Cardiovascular risk in inflammatory arthritis: rheumatoid arthritis and gout. The Lancet Rheumatology. 2021;3(1):e58-e70.
25.Rezuș E, Macovei LA, Burlui AM, Cardoneanu A, Rezuș C. Ischemic Heart Disease and Rheumatoid Arthritis—Two Conditions, the Same Background. Life [Internet]. 2021; 11(10).
26.Cai W, Tang X, Pang M. Prevalence of Metabolic Syndrome in Patients With Rheumatoid Arthritis: An Updated Systematic Review and Meta-Analysis. Frontiers in medicine. 2022;9:855141.
27.Grzechnik K, Targońska-Stępniak B. Metabolic Syndrome and Rheumatoid Arthritis Activity: An Analysis of Clinical, Laboratory, and Ultrasound Parameters. Nutrients. 2023;15(22).
28.Grzechnik K, Targońska-Stępniak B. Metabolic Syndrome and Rheumatoid Arthritis Activity: An Analysis of Clinical, Laboratory, and Ultrasound Parameters. Nutrients [Internet]. 2023; 15(22).
29.Li L, Feng D, Zeng J, Ye P, Chen Y, Wei D. Association between rheumatoid factor and metabolic syndrome in general population. Diabetology & Metabolic Syndrome. 2022;14(1):165.
30.Nazir S, Jankowski V, Bender G, Zewinger S, Rye K-A, van der Vorst EPC. Interaction between high-density lipoproteins and inflammation: Function matters more than concentration! Advanced Drug Delivery Reviews. 2020;159:94-119.
31.Carmona L, Aurrecoechea E, García de Yébenes MJ. Tailoring Rheumatoid Arthritis Treatment through a Sex and Gender Lens. Journal of clinical medicine. 2023;13(1).
32.Bagheri-Hosseinabadi Z, Moadab F, Abbasifard M. Prevalence and contributing factors of metabolic syndrome in rheumatoid arthritis patients. BMC Endocrine Disorders. 2024;24(1):140.
33.Martemucci G, Khalil M, Di Luca A, Abdallah H, D'Alessandro AG. Comprehensive Strategies for Metabolic Syndrome: How Nutrition, Dietary Polyphenols, Physical Activity, and Lifestyle Modifications Address Diabesity, Cardiovascular Diseases, and Neurodegenerative Conditions. Metabolites [Internet]. 2024; 14(6).
34. Grzechnik K, Targońska-Stępniak B. The pathogenic relationship between metabolic syndrome and rheumatoid arthritis activity. Rheumatology Forum. 2024;10(3):105-9.
35.Abdulla A, Sadida HQ, Jerobin J, Elfaki I, Mir R, Mirza S, et al. Unraveling molecular interconnections and identifying potential therapeutic targets of significance in obesity-cancer link. Journal of the National Cancer Center. 2024.
36.Cheuk Yin W, Becky MYM, Danting Z, Wynn C, Tak Mao C, Desmond YHY. Cardiovascular risk factors and complications in patients with systemic lupus erythematosus with and without nephritis: a systematic review and meta-analysis. Lupus Science & Medicine. 2024;11(1):e001152.
37.Gigante A, Iannazzo F, Navarini L, Sgariglia MC, Margiotta DPE, Vaiarello V, et al. Metabolic syndrome and adipokine levels in systemic lupus erythematosus and systemic sclerosis. Clinical rheumatology. 2021;40(10):4253-8.
38.Bello N, Meyers KJ, Workman J, Hartley L, McMahon M. Cardiovascular events and risk in patients with systemic lupus erythematosus: Systematic literature review and meta-analysis. Lupus. 2023;32(3):325-41.
39.Frostegård J. Systemic lupus erythematosus and cardiovascular disease. 2023;293(1):48-62.
40.Semalulu T, Tago A, Zhao K, Tselios K. Managing Cardiovascular Risk in Systemic Lupus Erythematosus: Considerations for the Clinician. ImmunoTargets and therapy. 2023;12:175-86.
41.Henein MY, Vancheri S, Longo G, Vancheri F. The Role of Inflammation in Cardiovascular Disease. International journal of molecular sciences. 2022;23(21).
42.Mosalmanzadeh N, Pence BD. Oxidized Low-Density Lipoprotein and Its Role in Immunometabolism. International journal of molecular sciences [Internet]. 2024; 25(21).
43.Guzmán-Martínez G, Marañón C. Immune

mechanisms associated with cardiovascular disease in systemic lupus erythematosus: A path to potential biomarkers. Frontiers in immunology. 2022;13:974826.
44.Ugarte-Gil MF, Mak A, Leong J, Dharmadhikari B, Kow NY, Reátegui-Sokolova C, et al. Impact of glucocorticoids on the incidence of lupus-related major organ damage: a systematic literature review and meta-regression analysis of longitudinal observational studies. Lupus Sci Med. 2021;8(1).
45.Pofi R, Caratti G, Ray DW, Tomlinson JW. Treating the Side Effects of Exogenous Glucocorticoids; Can We Separate the Good From the Bad? Endocrine reviews. 2023;44(6):975-1011.
46.Mejía-Vilet JM, Ayoub I. The Use of Glucocorticoids in Lupus Nephritis: New Pathways for an Old Drug. 2021;8.
47.Wondafrash DZ, Desalegn TZ, Yimer EM, Tsige AG, Adamu BA, Zewdie KA. Potential Effect of Hydroxychloroquine in Diabetes Mellitus: A Systematic Review on Preclinical and Clinical Trial Studies. Journal of diabetes research. 2020;2020:5214751.
48.Huang SP, DerSarkissian M, Gu YM, Duh MS, Wang MJ, Benson J, et al. Prolonged oral corticosteroid treatment in patients with systemic lupus erythematosus: An evaluation of 12-month economic and clinical burden. Journal of managed care & specialty pharmacy. 2023;29(4):365-77.
49.Dlamini SN, Lombard Z, Micklesfield LK, Crowther N, Norris SA, Snyman T, et al. Glucocorticoids associate with cardiometabolic risk factors in black South Africans. Endocrine connections. 2021;10(8):873-84.
50.Tsujimae M, Masuda A, Takagi M, Kato T, Nakano R, Fujita K, et al. Relapse and side effects of steroid therapy beyond 3 years in autoimmune pancreatitis: A multicenter retrospective study. Pancreatology. 2024;24(2):223-31.
51.Grimaldi L, Duchemin T, Hamon Y, Buchard A, Benichou J, Abenhaim L, et al. Hydroxychloroquine and Cardiovascular Events in Patients With Systemic Lupus Erythematosus. JAMA network open. 2024;7(8):e2432190.
52.Poznyak AV, Orekhov NA, Churov AV, Starodubtseva IA, Beloyartsev DF, Kovyanova TI, et al. Mitochondrial Dysfunction in Systemic Lupus Erythematosus: Insights and Therapeutic Potential. Diseases (Basel, Switzerland). 2024;12(9).
53.Liang S, Wei Z, Fang H, Guan T. Meta-analysis of effectiveness and safety of glucocorticoid combined with hydroxychloroquine in the treatment of systemic lupus erythematosus rash. International journal of rheumatic diseases. 2023;26(9):1686-96.
54.Sircana MC, Erre GL, Castagna F, Manetti R. Crosstalk between Inflammation and Atherosclerosis in Rheumatoid Arthritis and Systemic Lupus Erythematosus: Is There a Common Basis? Life [Internet]. 2024; 14(6).
55.Azuaga AB, Ramírez J, Cañete JD. Psoriatic Arthritis: Pathogenesis and Targeted Therapies. International journal of molecular sciences. 2023;24(5).
56.Hao Y, Zhu YJ, Zou S, Zhou P, Hu YW, Zhao QX, et al. Metabolic Syndrome and Psoriasis: Mechanisms and Future Directions. Frontiers in immunology. 2021;12:711060.
57.Atzeni F, Gerratana E, Francesco Masala I, Bongiovanni S, Sarzi-Puttini P, Rodríguez-Carrio J. Psoriatic Arthritis and Metabolic Syndrome: Is There a Role for Disease Modifying Anti-Rheumatic Drugs? 2021;8.
58.Jamil M, Aslam R, Patel A, Nadir B, Khan S. Prevalence and Extent of Subclinical Atherosclerosis and Associated Cardiovascular Risk Factors in Adult Patients With Psoriatic Arthritis: A Systematic Review. Cureus. 2021;13(8):e16853.
59.Degboé Y, Koch R, Zabraniecki L, Jamard B, Couture G, Ruidavets JB, et al. Increased Cardiovascular Risk in Psoriatic Arthritis: Results From a Case-Control Monocentric Study. Frontiers in medicine. 2022;9:785719.
60.Atzeni F, Gerratana E, Francesco Masala I, Bongiovanni S, Sarzi-Puttini P, Rodríguez-Carrio J. Psoriatic Arthritis and Metabolic Syndrome: Is There a Role for Disease Modifying Anti-Rheumatic Drugs? Frontiers in medicine. 2021;8:735150.
61.Guan H, Tian J, Wang Y, Niu P, Zhang Y, Zhang Y, et al. Advances in secondary prevention mechanisms of macrovascular complications in type 2 diabetes mellitus patients: a comprehensive review. European Journal of Medical Research. 2024;29(1):152.
62.George MD, Hsu JY, Hennessy S, Chen L, Xie F, Curtis JR, et al. Risk of Serious Infection With Low-dose Glucocorticoids in Patients With Rheumatoid Arthritis: An Instrumental Variable Analysis. Epidemiology (Cambridge, Mass). 2022;33(1):65-74.
63.Yan L, Wang W, Qiu Y, Yu C, Wang R, Li C. Role of glucose metabolism reprogramming in keratinocytes in the link between psoriasis and metabolic syndrome. International Immunopharmacology. 2024;139:112704.
64.Gao X, Li R, Jin T, Tang H. The Association Between Metabolic Syndrome and Prostate Cancer Risk: A Large-Scale Investigation and Study of Chinese. Frontiers in endocrinology. 2022;13:787268.
65.Atzeni F, Gerratana E, Masala I, Bongiovanni S, Sarzi-Puttini P, Rodríguez-Carrio J. Psoriatic Arthritis and Metabolic Syndrome: Is There a Role for Disease Modifying Anti-Rheumatic Drugs? Frontiers in medicine. 2021;8:735150.

66.Grygiel-Górniak B, Skoczek W. Molecular Background and Clinical Implications of Glucose Disorders in Patients with Psoriatic Arthritis. Journal of clinical medicine [Internet]. 2023; 12(18).
67.Aljohani R. Metabolic Syndrome and Its Components in Psoriatic Arthritis. Open access rheumatology : research and reviews. 2022;14:7-16.
68.Su L, Xu C, Huang H, Zhang P, Wang J, Ouyang X, et al. Effects of tumor necrosis factor-alpha inhibitors on lipid profiles in patients with psoriasis: a systematic review and meta-analysis. Frontiers in immunology. 2024;15:1354593.
69.Law L, Lindqvist P, Liv P, Hellman U, Lejon K, Geijer M, et al. Increased carotid intima-media thickness in patients with radiographic axial spondyloarthritis compared to controls and associations with markers of inflammation. Clinical rheumatology. 2024;43(5):1559-70.
70.Yan D, Blauvelt A, Dey AK, Golpanian RS, Hwang ST, Mehta NN, et al. New Frontiers in Psoriatic Disease Research, Part II: Comorbidities and Targeted Therapies. The Journal of investigative dermatology. 2021;141(10):2328-37.
71.Mehta H, Narang T, Dogra S, Handa S, Hatwal J, Batta A. Cardiovascular Considerations and Implications for Treatment in Psoriasis: An Updated Review. Vascular Health and Risk Management. 2024;20:215-29.
72.Olszewski R, Ćwiklińska K, Maślińska M, Kwiatkowska B. Prevention and risk assessment of cardiovascular events in a population of patients with psoriasis and psoriatic arthritis. Reumatologia. 2022;60(4):266-74.
73.Chen Y, Lin P-H, Freedland SJ, Chi J-T. Metabolic Response to Androgen Deprivation Therapy of Prostate Cancer. Cancers [Internet]. 2024; 16(11).
74.Yang U, Harikrishna A, Preda V, Chen J. Efficacy of multidisciplinary interventions in preventing metabolic syndrome and improving body composition in prostate cancer patients treated with androgen deprivation therapy: A systematic review and meta-analysis. Clinical Nutrition ESPEN. 2023;58:27-49.
75.Yang S, Liu H, Fang X-M, Yan F, Zhang Y. Signaling pathways in uric acid homeostasis and gout: From pathogenesis to therapeutic interventions. International Immunopharmacology. 2024;132:111932.
76.Han T, Chen W, Qiu X, Wang W. Epidemiology of gout - Global burden of disease research from 1990 to 2019 and future trend predictions. Therapeutic advances in endocrinology and metabolism. 2024;15:20420188241227295.
77.Raya-Cano E, Vaquero-Abellán M, Molina-Luque R, De Pedro-Jiménez D, Molina-Recio G, Romero-Saldaña M. Association between metabolic syndrome and uric acid: a systematic review and meta-analysis. Scientific reports. 2022;12(1):18412.
78.Du L, Zong Y, Li H, Wang Q, Xie L, Yang B, et al. Hyperuricemia and its related diseases: mechanisms and advances in therapy. Signal Transduction and Targeted Therapy. 2024;9(1):212.
79.Hansildaar R, Vedder D, Baniaamam M, Tausche AK, Gerritsen M, Nurmohamed MT. Cardiovascular risk in inflammatory arthritis: rheumatoid arthritis and gout. The Lancet Rheumatology. 2021;3(1):e58-e70.
80.Mao T, He Q, Yang J, Jia L, Xu G. Relationship between gout, hyperuricemia, and obesity-does central obesity play a significant role?-a study based on the NHANES database. Diabetol Metab Syndr. 2024;16(1):24.
81.Du L, Zong Y, Li H, Wang Q, Xie L, Yang B, et al. Hyperuricemia and its related diseases: mechanisms and advances in therapy. Signal Transduct Target Ther. 2024;9(1):212.
82.Lee Y, Hwang J, Desai SH, Li X, Jenkins C, Kopp JB, et al. Efficacy of Xanthine Oxidase Inhibitors in Lowering Serum Uric Acid in Chronic Kidney Disease: A Systematic Review and Meta-Analysis. Journal of clinical medicine. 2022;11(9).
83.Sosa F, Shaban M, Lopez J, Duarte GJ, Jain S, Khizar A, et al. Impact of Hyperuricemia and Urate-Lowering Agents on Cardiovascular Diseases. Clinical Medicine Insights Cardiology. 2024;18:11795468241239542.
84.Masenga SK, Kabwe LS, Chakulya M, Kirabo A. Mechanisms of Oxidative Stress in Metabolic Syndrome. International journal of molecular sciences [Internet]. 2023; 24(9).
85.Eun Y, Han K, Lee SW, Kim K, Kang S, Lee S, et al. Increased risk of incident gout in young men with metabolic syndrome: A nationwide population-based cohort study of 3.5 million men. 2022;9.
86.Yao T-K, Lee R-P, Wu W-T, Chen I-H, Yu T-C, Yeh K-T. Advances in Gouty Arthritis Management: Integration of Established Therapies, Emerging Treatments, and Lifestyle Interventions. International journal of molecular sciences [Internet]. 2024; 25(19).
87.Costa N, Scavuzzi B, Iriyoda T, Lozovoy M, Alfieri D, Medeiros F, et al. Metabolic syndrome and the decreased levels of uric acid by leflunomide favor redox imbalance in patients with rheumatoid arthritis. Clinical and Experimental Medicine. 2018;18.
88.Njeim R, Pannala SSS, Zaidan N, Habib T, Rajamanuri M, Moussa E, et al. Prevalence of Metabolic Syndrome and Its Association with Cardiovascular Outcomes in Hospitalized Patients with Inflammatory Bowel Disease. Journal of clinical medicine. 2024;13(22).
89.Kuriya B, Schieir O, Valois MF, Pope JE, Boire G, Bessette L, et al. Prevalence and Characteristics

of Metabolic Syndrome Differ in Men and Women with Early Rheumatoid Arthritis. ACR open rheumatology. 2019;1(9):535-41.
90.Drosos AA, Venetsanopoulou AA, Pelechas E, Voulgari PV. Exploring Cardiovascular Risk Factors and Atherosclerosis in Rheumatoid Arthritis. European Journal of Internal Medicine. 2024;128:1-9.
91.Baoqi Y, Dan M, Xingxing Z, Xueqing Z, Yajing W, Ke X, et al. Effect of Anti-Rheumatic Drugs on Cardiovascular Disease Events in Rheumatoid Arthritis. Frontiers in cardiovascular medicine. 2021;8:812631.
92.Parker B, Bruce I. The Metabolic Syndrome in Systemic Lupus Erythematosus. Rheumatic diseases clinics of North America. 2010;36:81-97, viii.
93.Corona-Meraz FI, Vázquez-Del Mercado M, Sandoval-García F, Robles-De Anda JA, Tovar-Cuevas AJ, Rosales-Gómez RC, et al. Biomarkers in Systemic Lupus Erythematosus along with Metabolic Syndrome. Journal of clinical medicine. 2024;13(7).
94.Karmacharya P, Ogdie A, Eder L. Psoriatic arthritis and the association with cardiometabolic disease: a narrative review. Therapeutic advances in musculoskeletal disease. 2021;13:1759720x21998279.
95.Vecellio M, Hake VX, Davidson C, Carena MC, Wordsworth BP, Selmi C. The IL-17/IL-23 Axis and Its Genetic Contribution to Psoriatic Arthritis. Frontiers in immunology. 2020;11:596086.
96.Mao T, He Q, Yang J, Jia L, Xu G. Relationship between gout, hyperuricemia, and obesity—does central obesity play a significant role?—a study based on the NHANES database. Diabetology & Metabolic Syndrome. 2024;16(1):24.
97.Cai R, Li F, Li Y, Li Y, Peng W, Zhao M, et al. Mechanism and use strategy of uric acid-lowering drugs on coronary heart disease. IJC Heart & Vasculature. 2024;53:101434.
98.Mashala DG, Ntimana CB, Seakamela KP, Mashaba RG, Maimela E. Sociodemographic Disparities in the Prevalence of Metabolic Syndrome in Rural South Africa: An Analysis of Gender, Age, and Marital, Employment, and Educational Status. Obesities [Internet]. 2024; 4(4):[480-90 pp.].
99.Özmen M, Yersal Ö, Öztürk S, Soysal D, Köseeoğlu MH. Prevalence of the metabolic syndrome in rheumatoid arthritis. European journal of rheumatology. 2014;1(1):1-4.
100.Gosselt HR, Muller IB, Jansen G, van Weeghel M, Vaz FM, Hazes JMW, et al. Identification of Metabolic Biomarkers in Relation to Methotrexate Response in Early Rheumatoid Arthritis. Journal of personalized medicine. 2020;10(4).