Original Paper

Metabolic and Inflammatory Disorders-Main Influencers of Psoriatic Arthritis Activity

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ABSTRACT: Background: Psoriatic arthritis is a systemic, potentially severe condition associated with multiple comorbidities. It is characterized by peripheral and axial joint involvement, enthesitis, and association with various comorbidities. Objective: This study aims to identify some of the determinants of the severity of psoriatic arthritis. We studied the prevalence and common pathogenic mechanisms of various comorbidities associated with PsA. Methods: We studied 103 patients diagnosed with psoriatic arthritis. 69 met the inclusion criteria. We evaluated the disease activity index DAPSA, ESH, CRP, BMI, uric acid, cholesterol, triglycerides. Disease duration and type of joint involvement were also included. Results: The patients in the studied group had an average age of 54.75 (sd±10.02) years and there were 36 (55.38%) women. Duration of the diseases 8.254 (sd±5.307) years and an average of DAPSA values of 20.58 (sd±8.522). Clinical manifestations: 37 (56.92%) had peripheral damage; 6 (9.23%) had axial damage and 22 (33.85%) had mixed axial and peripheral damage. Inflammatory markers: ESR with an average serum value of 39.55 (sd±26.43) mm/h and average CRP values of 17.69mg/L (sd±28.71). The body mass index-an average value of 29.69Kg/m² (sd±5.979). Biological markers: uric acid with an average serum value of 9.832 mg/dl (sd±2.089), triglycerides 262.6 mg/dl (sd±41.58) and cholesterol 319.8mg/dl (sd±56.14). Conclusions: Serum levels of uric acid, cholesterol, triglycerides, ESR, and CRP influence psoriatic arthritis activity and response to treatment.

KEYWORDS: *Psoriatic arthritis, uric acid, dyslipidemia, metabolic syndrome.*

Introduction

Psoriatic arthritis (PsA) is a systemic rheumatic inflammatory disease that can precede, accompany or succeed psoriasis and which include wide spectrum of clinical manifestations, currently being able to talk about a real ‘psoriatic disease’.

Acute episodes of joint swelling in PsA patients can have different causes and require specific treatments.

It is known that the risk of gout is higher among patients with PsA so there are authors who describe the term known as "psout" [1].

That refers to patients with psoriatic arthritis who simultaneously develop gout at a certain time during the evolution of the disease.

Also, we can be able to talk about an overlap syndrome.

The mechanism responsible for the occurrence of hyperuricemia, frequently encountered in patients with psoriatic arthritis, remains insufficiently elucidated and is a subject of debate in the scientific literature.

Uric acid is the final product of purine metabolism and represents the etiological substrate of gout.

Monosodium urate crystals phagocytosed by monocytes or neutrophils cause the generation of pro-inflammatory cytokines such as IL-1.

In addition, internalization of crystals by leukocytes leads to free radical production, cathepsin B release, and inflammasome activation [2].

Hyperuricemia, described as elevated serum uric acid levels, is three times more prevalent in patients with psoriasis than in the general population.

Although it is essential to have elevated serum uric acid levels for monosodium urate crystal formation, not all hyperuricemic individuals will develop gout [1].

Gout is significantly associated with psoriasis and psoriatic arthritis. This varies according to the patient's age. It is more common between 40 and 50 years of age [4].

The mechanism underlying the interaction between hyperuricemia and PsA is not fully understood. Hight levels of serum uric acid in patients with PsA may result from either increased skin cell turnover or from the systemic inflammatory state. This is well known to be associated with PsA [1].

There is a higher prevalence of monosodium urate crystals identified in synovial fluid in

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10.12865/CHSJ.52.01.06

patients with PsA, in contrast to other rheumatic conditions such as rheumatoid arthritis or osteoarthritis. It is considered that hyperuricemia could affect the severity of clinical manifestations and the degree of inflammation in patients with PsA [2].

Patients with psoriatic arthritis have a significantly increased risk of simultaneously developing hyperuricemia and also gout. These patients, who have concomitant PsA and hyperuricemia, have demonstrated a poorer response to treatment for psoriatic arthritis.

They also develop more destructive and peripheral forms of arthritis [6].

The management of PsA is often a challenge for the clinician. First of all due to the lack of specific biomarkers but also due to the presence of several associated comorbidities. Another important cause could be the multisystemic involvement, such as cardiovascular diseases, obesity, hypertension, metabolic syndrome, hyperuricemia, malignancy, liver and kidney diseases or infections [10].

Objective

This study aims to identify some of the determinants of the severity of psoriatic arthritis. Finding them is extremely useful in achieving the therapeutic target. Thus, we aim to study the prevalence and common pathogenic mechanisms of various comorbidities associated with PsA.

Methods

We conducted a retrospective cohort study, over a five-year period (2020-2025), within the Rheumatology Department of the Craiova County Emergency Clinical Hospital. The study included patients previously diagnosed with psoriatic arthritis by the attending rheumatologist, according to the CASPAR classification criteria.

Data Collection

We included in this study a group of patients who had already been diagnosed with psoriatic arthritis by the attending physician rheumatologist according to the CASPAR classification criteria. Those who had at least one measurement of the serum uric acid level were selected. A serum uric acid level greater than 7mg/dl for men and greater than 5mg/dl for women was considered hyperuricemia. Uric acid levels lower than this value were considered normal. These patients were classified as normouricemic.

Patients with incomplete medical documentation (lack of data required for DAPSA calculation or relevant biological determinations), those with acute infections or other intercurrent inflammatory states at the time of collection, patients with active neoplasia or oncological treatment, as well as those with concomitant autoimmune/inflammatory diseases that could have influenced inflammatory markers were excluded. Patients under high-dose systemic corticosteroid therapy in the period close to the evaluation were also excluded. Patients who presented with hyperuricemia of renal origin were also excluded. Therefore, acute or chronic renal disease represented an exclusion criterion for our study.

From the hospital admission files of the selected patients, we collected demographic, biological and clinical data. Demographic data included age and gender. The data about PsA included the duration of the disease, its activity evaluated by the Disease Activity Index for Psoriatic Arthritis (DAPSA) calculated by the attending physician during hospitalization, the presence or absence of skin manifestations recorded in the hospitalization record, as well as the joint clinical manifestations of the disease.

The intensity of the activity of the disease was evaluated according to the value of the DAPSA. The scores we evaluated are: <4 disease remission; 4-14 low disease activity; 14-28 moderate disease activity and >28 high disease activity. The clinical aspects of psoriatic arthritis were defined according to the five known subtypes: axial, polyarticular, oligoarticular, mutilating and distal interphalangeal. For our study, we included a category with axial damage, one with peripheral joint damage and one with mixed damage.

Other patient-related data were represented by inflammatory markers: erythrocyte sedimentation rate (ESR), C-reactive protein (CRP). We also collected data on lipid profile: cholesterol, triglycerides; carbohydrate profile; liver transaminases: alanine aminotransferase (ALT), aspartate aminotransferase (AST); renal function: urea, creatinine; body mass index (BMI).

By integrating these clinical and biological data, we aimed to analyze the relationship between disease activity and the inflammatory-metabolic status of patients with psoriatic arthritis.

Statistical analysis

Statistical analysis was performed using GraphPad Prism software, version 8 (GraphPad Software Inc., San Diego, CA, USA).

Continuous variables were expressed as mean±standard deviation (SD), while categorical variables were presented as absolute frequencies and percentages. Descriptive statistical methods were used to summarize the demographic, clinical, inflammatory, and metabolic characteristics of the study population.

To investigate the relationships between disease activity and clinical-biological parameters, a principal component analysis (PCA) was performed. The variables included in the analysis were disease activity assessed by the Disease Activity Index for Psoriatic Arthritis (DAPSA), inflammatory markers (erythrocyte sedimentation rate [ESR] and C-reactive protein [CRP]), metabolic parameters (serum uric acid, triglycerides, total cholesterol, and body mass index [BMI]), and disease duration.

PCA was applied as an exploratory multivariate statistical method to reduce data dimensionality and to identify patterns of association among variables influencing psoriatic arthritis activity. Principal component loadings were examined to evaluate the contribution of each parameter to the identified components. The relationships between variables were visually assessed using graphical representation of the PCA model.

All statistical tests were two-tailed, and a p-value <0.05 was considered statistically significant.

Results

We evaluated the medical records of 103 patients hospitalized in the Rheumatology department of the Craiova County Emergency Clinical Hospital, Romania. Only 69 met the inclusion criteria in our study.

The patients in the studied group had an average age of 54.75 (sd±10.02) years and there were 36 (55.38%) women. The characteristics of the disease are represented by a duration of 8.254 (sd±5.307) years and an average of DAPSA values of 20.58 (sd±8.522). Regarding the clinical manifestations, 37 (±56.92%) had peripheral damage; 6 (9.23%) had axial damage and 22 (33.85%) had mixed axial and peripheral damage. The evaluated inflammatory markers were represented by ESR with an average serum value of 39.55 (sd±26.43) mm/h and average CRP values of 17.69mg/L (sd±28.71). The body mass index was also evaluated, with an average value of 29.69 Kg/m² (sd±5.979). The monitored biological markers were represented by uric acid with an average serum value of 9.832mg/dl (sd ±2.089), triglycerides 262.6mg/dl (sd±41.58) and cholesterol 319.8 mg/dl (sd±56.14).

Table 1. shows the main characteristics of the group of patients.

Table 1. The characteristics of the patients in the studied group.

Parameter	Value	
Age [years, mean, ds]	54,75	±10,02
Women [no., %]	36	55.38
Duration of the disease [years, mean, ds]	8,254	±5,307
DAPSA [mean, ds]	20.58	±8.522
Type of joint damage:		
peripheral (no., %)	37	56.92
axial (no., %)	6	9.23
mixed (no., %)	22	33.85
ESR [mm/h, mean, ds]	39.55	±26.43
CRP [mg/L, mean, ds]	17.69	±28.71
Uric acid [mg/dL, mean, ds]	9,832	±2,089
Triglycerides [mg/dL, mean, ds]	262.6	±41.58
Cholesterol [mg/dL, mean, ds]	319.8	±56.14
BMI [kg/m2, mean, ds]	29.69	±5.979

Sd=standard deviation

Figure 1 shows the main components of patients with psoriatic arthritis (MCA), highlighting the relationships between the activity of the disease (DAPSA), inflammatory markers (ESR and CRP), metabolic factors (uric acid, triglycerides, cholesterol, BMI) and the duration of the disease.

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The position of DAPSA on the PCA

Descriptive analysis of the cohort reveals a clinical profile characterized by moderate inflammatory disease activity (mean DAPSA 20.58±8.52), associated with elevated values of systemic inflammatory markers (ESR 39.55mm/h; CRP 17.69mg/L) and an altered metabolic status. The mean values of uric acid (9.83mg/dL), triglycerides (262.6mg/dL) and total cholesterol (319.8mg/dL) significantly exceed the ranges considered physiological, suggesting an increased prevalence of metabolic dysfunction in this population.

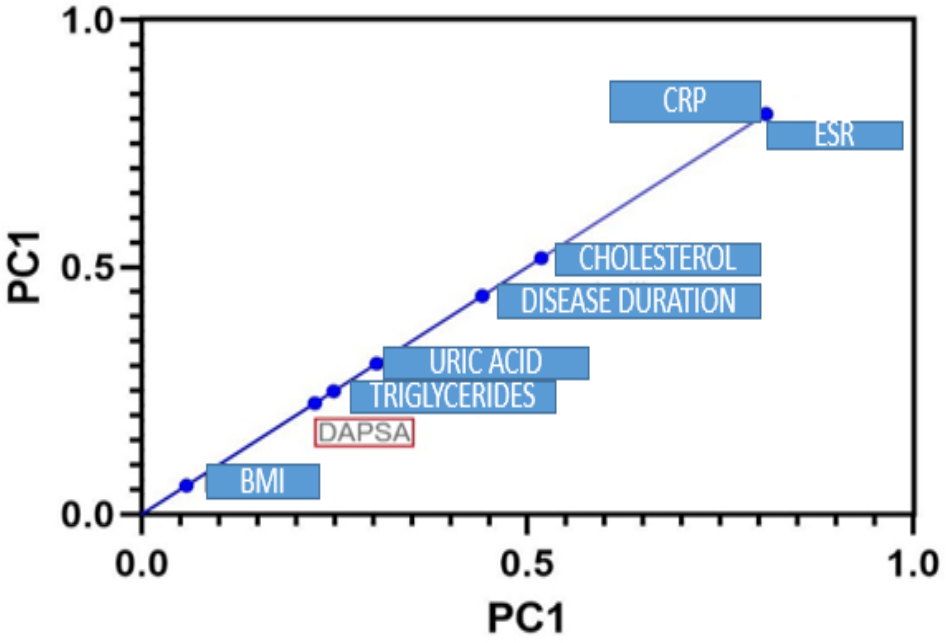

Figure 1. Principal component analysis (PCA) graph of patients with psoriatic arthritis, highlighting the relationship between disease activity (DAPSA), inflammatory markers (ESR and CRP), metabolic factors (uric acid, triglycerides, cholesterol, BMI) and duration of the disease.

Relationships with specific parameters

Principal component analysis (PCA) provides a multivariate perspective on the interaction between inflammation and metabolism in determining disease activity.

Disease duration: positioned relatively close to DAPSA on the PCA plot, indicates that longer disease duration may be moderately correlated with higher DAPSA scores. This suggests that disease activity may increase with disease duration. Disease duration, positioned relatively close to DAPSA on the plot, suggests a possible progressive accumulation of inflammatory and metabolic burden over time, with a trend toward higher activity scores in patients with longer disease duration.

The positioning of DAPSA in proximity to uric acid and triglycerides, as well as their moderate loadings on PC1 (uric acid 0.304; triglycerides 0.248; cholesterol 0.517) suggests the existence of a metabolic component that contributes to the variability of disease activity.

This spatial distribution in the factorial plane indicates a moderate correlation between joint activity and dysmetabolic parameters, supporting the hypothesis of an interdependence between immune-mediated inflammation and metabolic dysfunction.

Also, cholesterol, with a relatively higher loading compared to triglycerides and uric acid, seems to contribute significantly to strengthening the link between dyslipidemia and the inflammatory profile of the disease.

ESR (Loading: 0.808) and CRP (Loading: 0.809): these markers have the highest loadings on PC1, indicating that they are the strongest influencers of this component. Although DAPSA has a moderate loading, it is not as tightly clustered with these inflammatory markers on the graph, suggesting that although inflammation is a significant contributor to disease activity, other factors also play a role.

In contrast, BMI has a minimal loading on PC1 (0.058), suggesting that overweight, although prevalent, is not directly and linearly correlated with disease activity measured by DAPSA in this cohort. This result may indicate that the impact of obesity on disease activity is mediated by indirect mechanisms (adipokines, insulin resistance, low-grade inflammation), rather than a directly proportional relationship.

Clinical Implications

Thus, the analysis suggests that the activity of psoriatic arthritis is the result of a multifactorial interaction between: systemic inflammation, as a major determinant, metabolic dysfunction and disease duration as adjuvant factors, and weight status as an indirect factor.

The results support the hypothesis that hyperuricemia and dyslipidemia are not simple comorbidities, but may actively participate in amplifying the inflammatory response and maintaining disease activity. The moderate association between DAPSA and uric acid suggests that hyperuricemia could have prognostic or predictive value in assessing disease severity, especially in patients with an altered metabolic profile.

From a pathogenetic perspective, the data obtained are compatible with the integrative inflammation-metabolism model, in which proinflammatory cytokines and lipid dysfunction contribute synergistically to the perpetuation of the disease and increased cardiovascular risk.

Discussion

Analyzing the clinical and biological characteristics of patients diagnosed with psoriatic arthritis who presented concomitant hyperuricemia, it can be concluded that the activity of the disease can be influenced by serum uric acid values together with the presence of other metabolic factors such as cholesterol and triglycerides. We have analyzed how uric acid may be involved in the pathogenesis of PsA and how the clinical manifestations of PsA and inflammatory mediators are affected by uric acid concentrations. The patients in our study had a predominantly peripheral type of disease and a smaller percentage of them had mixed or axial types.

The epidemiological characteristics of our cohort are comparable to those in the literature [2,7].

There are studies that have shown a close interconnection between the conditions that we have analyzed. AlJohani R, et al. [11] identified correlations of PsA with hyperuricemia related to the associated metabolic syndrome. These conclusions are suggesting the possibility of the existence of common pathophysiological mechanisms which are not yet well known.

Therefore, we can say that the concomitant presence of psoriatic arthritis with hyperuricemia and gouty atrophy in the same patient is not just a coincidence. This could represent a combination of interconnected pathogenic mechanisms [12].

There are even discussion about the existence of an overlap syndrome between the two diseases at the border between inflammation and metabolic disorders [7,12].

There are studies that have attempted to elucidate the link between PsA and hyperuricemia, trying to define the term Psout in the context of metabolic disorders associated with PsA or as a condition itself [13].

However, there is still no standard definition of this term or a consensus on therapeutic management.

Patrycja Ozdowska et al. [8] conducted a case-control study that showed that among the seronegative spondyloarthritis, psoriatic arthritis is the one most frequently associated with dyslipidemia. Also, unlike patients with axial spondyloarthritis, those with psoriatic arthritis have a more atherogenic lipid profile represented by more frequent increases in triglycerides, decreases in HDL cholesterol, and a higher atherogenic index. Association between inflammation and metabolic disorders in patients with psoriatic arthritis leads to progressive endothelial dysfunction with a moderate increase in the risk of death from cardiovascular disease after 10 years. The cardiovascular risk profile highlighted by our results coincides with the conclusions from a review by Mike J Peters et al. [14].

By generating secondary inflammation, metabolic syndrome represents not only an important comorbidity of psoriatic arthritis, but also an important factor in determining disease activity [9].

Careful and frequent monitoring of inflammatory marker values, lipids, and serum uric acid should be mandatory in all patients diagnosed with psoriatic arthritis [3,7].

All the more so since chronic inflammation is associated with endothelial dysfunction as a

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risk factor for cardiovascular events, as demonstrated by several studies [15].

It has also been demonstrated that the association of hyperuricemia with metabolic syndrome represents a risk factor for endothelial dysfunction [16], even suggesting the idea of including hyperuricemia as part of the metabolic syndrome [17-19].

The role of hyperuricemia as a risk factor for endothelial dysfunction in the general population has been demonstrated [20].

Our results show that in patients with PsA, other risk factors such as inflammation and metabolic dysfunction are also added.

All these data obtained by the mentioned authors, are consistent with the results of our analysis, identifying the correlation between psoriatic disease activity with inflammatory markers, elevated uric acid levels and components of the metabolic syndrome, suggesting cardiovascular risk in this patient profile.

Moreover, the association of these markers with the disease activity suggests the importance of early diagnosis and integrated management of these comorbidities in patients with psoriatic arthritis.

Study limitations

The present study has a retrospective design, which allowed the analysis of a relevant number of patients from real clinical practice, providing a complex picture of the inflammatory and metabolic profile of patients with psoriatic arthritis. However, the observational nature of the research limits the possibility of establishing direct causal relationships between the variables analyzed, the results mainly reflecting clinical associations.

The evaluation focused predominantly on the articular and biological components of the disease, without including a standardized quantification of skin extension (e.g., PASI score), an aspect that could bring additional information in future studies. So we can consider as limitation the lack of assessment of the cutaneous extension of psoriasis. The lack of adjustment for confounding factors as well as the the lack of comparison with the control group also represents a limitation of our study.

The varied treatment that patients received may modify the results of our study.

Conclusions

The activity of psoriatic arthritis is influenced by both inflammatory and metabolic markers.

DAPSA is a multidimensional index in psoriatic arthritis, moderately correlated with metabolic markers (uric acid, triglycerides, cholesterol) and disease duration.

It is influenced, but not dominated, by inflammatory markers (ESR and CRP).

This highlights the complexity of psoriatic arthritis and the need for comprehensive management strategies.

Metabolic disorders are not just comorbidities.

They play a central role in the disease progression, treatment resistance and cardiovascular risk.

A multidisciplinary approach that targets both inflammation and metabolic disorders is essential for the management of psoriatic arthritis.

A better understanding of the interaction between the pathogenic processes of psoriatic arthritis and metabolic syndrome may open the way to new therapeutic options.

Through optimal control of disease activity, will be achieved a reduction in cardiovascular risk and an increase in quality of life.

Acknowledgement

None to declare.

Author Contributions

LIS: literature search and selection of relevant studies, data interpretation, drafting and revision af the manuscript; PLC: methodological support, data interpretation, manuscript revision. CEB: analysis of metabolic aspects related to psoriatic arthritis, manuscript drafting; FP: conceptualization and design of the study, comprehensive literature search; FAV: conceptualization and design of the study, comprehensive literature search, critical analysis of data, drafting of the manuscript.

All authors read and approved the final manuscript.

Funding

This research received no specific funding.

Conflicts of interest

The authors declare no competing interests.

Institutional Review Board

The study was conducted according to the guidelines of the Declaration of Helsinki; the

study and the protocols utilized therein were approved by the University and Scientific Ethics and Deontology Commission of University of Medicine and Pharmacy, Craiova, no. 152/24.09.2021.

Consent Statement

All human subjects involved in this study provided a written informed consent upon admission to the hospital, including the consent of publishing their anonymized data.

Data availability

All data presented in the manuscript are available from the authors upon request.

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 10.12865/CHSJ.52.01.06