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Original Research Article

Investigation of the frequency of hyperuricemia and associated risk factors in patients with psoriatic arthritis

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ABSTRACT

Background: This study aimed to determine the frequency of and clinical significance of hyperuricemia in psoriatic arthritis (PsA) patients.
Methods: The study included 63 PsA patients. Characteristics of the patients, psoriasis (Pso) and PsA duration, and co-morbidities such as hypertension, diabetes mellitus, dyslipidemia, hypothyroidism and coronary artery disease were collected. Moreover, serum uric acid (SUA) and C-reactive protein (CRP) levels at the time of diagnosis, psoriasis area and severity index (PASI) score, body surface area (BSA), Disease Activity index for Psoriatic Arthritis (DAPSA), and Bath Ankylosing Spondylitis Radiological Index (BASRI-total) score were also recorded.
Results: The mean age of the patients was 46.2±10.6 years. 28 (44.4%) had hyperuricemia, of which 23 (36.5%) were female and 5 (7.9%) were male. The mean SUA level was significantly higher in male patients (p=0.002), whereas hypertension was more prominent in female patients (p=0.010). There was no significant relationship between hyperuricemia and BSA, PASI and DAPSA score. PsA patients with hyperuricemia had a high significant difference in BMI (p=0.045) and hypertension (p=0.044). BASRI score was significantly related to age, body mass index, DAPSA score, plantar fascia, greater trochanter, and the Achilles enthesitis (p<0.001, p=0.005, p=0.001, p=0.015, p=0.016 and p=0.031, respectively). On regression analysis, only BMI was associated with SUA level (p=0.043).
Conclusions: The majority of PsA patients had asymptomatic hyperuricemia. Moreover, hyperuricemia was associated with BMI and hypertension. The higher rate of hyperuricemia in women than men seems to be due to their higher BMI values.

Keywords: Hyperuricemia, Metabolic syndrome, Psoriatic arthritis

INTRODUCTION

Psoriatic arthritis (PsA) is a multifaceted disease characterized by inflammation of the joints and skin lesions. The condition is classified as a type of spondyloarthritis (SpA), and its pathophysiology is largely influenced by immune dysregulation, genetic predisposition, and environmental factors.[1] PsA represents the joint component of systemic psoriatic disease and is the most common extracutaneous disorder in patients with psoriasis (Pso). Musculoskeletal involvement has been reported in 20% of patients with Pso and covers a wide spectrum of symptoms, including enthesitis, dactylitis, peripheral arthritis, and axial disease.[2-11] Besides joint involvement, PsA is associated with various metabolic abnormalities such as obesity, hyperlipidemia, insulin resistance, hypertension, diabetes, and hyperuricemia.[7,9-16]

Uric acid is the end product of purine metabolism and scavenges free oxygen radicals, thereby preventing red blood cell membrane lipid oxidation. Serum uric acid (SUA) level can be affected by many factors including

xanthine oxidase enzyme polymorphism, diet, alcohol consumption and kidney function.[10,11] Recently, the emerging role of hyperuricemia as a major cardiovascular risk factor has been emphasized.[10,11,17] Hyperuricemia leads to endothelial dysfunction by promoting the proinflammatory response of M1 macrophages and inhibiting the anti-inflammatory response of M2 macrophages, as well as impairing the activity of endothelial nitric oxide synthase and nitric oxide production, resulting in hypertension and atherosclerosis. Furthermore, hyperuricemia increases reactive oxygen species, leading to pancreatic β-cell dysfunction and apoptosis, resulting in insulin resistance and diabetes.[10] Moreover, uric acid has been shown to play a role in the pathophysiology of both Pso and PsA by activating keratinocytes and epidermal plasmacytoid dendritic cells, leading to the secretion of proinflammatory cytokines.[18] It has been reported that Pso patients with hyperuricemia are more prone to develop arthritis later in life. Furthermore, different studies have evaluated the interaction between hyperuricemia and psoriatic disease, suggesting that individuals with Pso or PsA may show higher SUA levels and that hyperuricemia may affect the severity of clinical manifestations and the degree of inflammation in PsA patients. Hyperuricemia has been hypothesized to be a consequence of the accelerated cutaneous cell cycle observed in Pso or an epiphenomenon secondary to the metabolic disturbances observed in Pso/PsA.[10,11] Previous studies in Turkish patients are limited on the relationship between uric acid and skin psoriasis, independent of arthritis.[19,20] Therefore, the aim of this study was to explore the frequency and clinical significance of hyperuricemia among PsA patients.

METHODS

This retrospective study was conducted at the Department of Physical Medicine and Rehabilitation in Bezmialem Vakif University between 2023 and 2024. The work was approved by the Ethical Committee of Bezmialem Vakif University (Trial Registration:2023/134). Written consent was provided by all patients.

63 PsA patients diagnosed according to the CASPAR (Classification Criteria for Psoriatic Arthritis) criteria (21) were included in the study. Inclusion criteria were as follows: the patients diagnosed with PsA for at least 1 year and being over 18 years of age. Exclusion criteria were as follows: a) being under 18 years of age; b) patients with a history of renal or liver failure; c) patients with a history of an accompanying secondary rheumatic disease (inflammatory bowel disease (chron's disease, ulcerative colitis), reiter's syndrome, and gout arthritis); d) patients using drug therapy that will affect uric acid metabolism. The socio-demographic data, body mass index (BMI), Pso and PsA duration, musculoskeletal findings such as enthesitis, and dactylitis, and co-morbidities such as hypertension, diabetes mellitus, dyslipidemia, hypothyroidism, and coronary artery disease were collected. Moreover, SUA and C-reactive protein (CRP) levels at the time of diagnosis of PsA, psoriasis area and severity index (PASI) (22) score, body surface area (BSA) (23), Disease Activity index for Psoriatic Arthritis (DAPSA) (24) score, Bath Ankylosing Spondylitis Radiological Index (BASRI-total, BASRI-spine and BASRI-hips) (25) score, and the human leukocytic antigen (HLA)-B27 status were also recorded. According to BSA, patients were groupped <10% or ≥10%. Normal SUA values are 1.5-6 mg/dl in adult women and 2.5-7 mg/dl in adult men. Hyperuricemia was defined as SUA ≥7 mg/dl in men and ≥6 mg/dl in women.

Statistical analysis

Statistical Package for the Social Sciences (SPSS) version 26 was used. Variables were presented as numbers and percentages or mean and standard deviation. The conformity of the quantitative variables to the normal distribution was examined using the Shapiro Wilk test. Comparisons were analyzed with Fisher's exact test, Pearson Chi-square test, and Mann Whitney U test. The Spearman and Pearson correlation coefficient was used to assess relationships between quantitative variables. A logistic regression model was built to determine the associated factors for hyperuricemia. After univariate analyzes, variables with a p value <0.25 were included in the model in multivariate analyses, and logistic regression analysis was performed. The fit of the model was evaluated with the Hosmer Lemeshow test. The significance level was taken as $p<0.05$.

RESULTS

The demographic and clinical characteristics of the patients are presented in table 1. The mean age of the patients was 46.2±10.6 years. Of the 63 patients, 28 (44.4%) had hyperuricemia, of which 23 (36.5%) were female and 5 (7.9%) were male. The mean SUA level was significantly higher in male patients, whereas hypertension was more prominent in female patients (Table 1).

Table 1: The demographic and characteristic features of the patients.

Variables mean±SD or N (%)	Patients (n=63)	Female (n=50)	Male (n=13)	p value
Age (years)	49.2±10.6	46.6±11.6	45.7±12.4	0.570
Body mass index (BMI)	30.2±5.0	30.6±5.5	28.5±2.2	0.185
Smoking				
No	50 (79.4)	44 (88)	6 (46.2)	0.330
Yes	13 (20.6)	6 (12)	7 (53.8)	

Continued.

Variables mean±SD or N (%)	Patients (n=63)	Female (n=50)	Male (n=13)	p value
Pso duration (years)	14.9±10.2	15.6±10.8	12.1±7.1	0.247
PsA duration (years)	2.8±3.0	3.1±3.3	1.4±0.5	0.218
HLA-B27 status				
(-)	54 (85.7)	43 (86)	11 (84.6)	1.000
(+)	9 (14.3)	7 (14)	2 (15.4)	
Axial involvement	6 (9.5)	4 (8)	2 (15.4)	0.492
Serum uric acid level (mg/dl)	5.4±1.3	4.8±1.1	6.1±1.4	0.002[S]
CRP (mg/dl)	7.2±9.2	7.5±9.5	6.1±8.2	0.596
PASI	3.9±1.7	3.8±1.7	4.4±1.6	0.215
BSA				
<%10	29 (46)	25 (50)	4 (30.8)	0.349
>%10	34 (54)	25 (50)	9 (69.2)	
DAPSA	25.6±10.0	26.2±10.5	23.5±7.7	0.449
BASRI-total	5.5±1.9	5.7±1.8	4.9±1.9	0.153
Musculoskeletal findings				
Enthesitis				
Plantar fascia	44 (69.8)	36 (72)	8 (61.5)	0.508
The achilles tendon	40 (63.5)	33 (66)	7 (53.8)	0.522
The patellar tendon	20 (31.7)	17 (34)	3 (23.1)	0.524
Iliac crest	4 (6.3)	1 (2)	3 (23.1)	0.025[S]
Greater trochanter	2 (3.2)	2 (4)	0 (0)	1.000
Greater tuberosity (humerus)	2 (3.2)	1 (2)	1 (7.7)	0.373
Dactylitis	10 (15.9)	9 (18)	1 (7.7)	0.672
Co-morbidities				
Hypertension	23 (39.7)	24 (48)	1 (7.7)	0.010[S]
Diabetes mellitus	21 (33.3)	18 (36)	3 (23.1)	0.516
Dyslipidemia	6 (9.5)	5 (10)	1 (7.7)	1.000
Coronary artery disease	7 (11.1)	6 (12)	1 (7.7)	1.000
Hypothyroidism	9 (14.3)	12 (24)	1 (7.7)	0.270

Po: Psoriasis, PsA: Psoriatic arthritis, CRP: C-reactive protein, PASI: Psoriasis area and severity index, BSA: Body surface area, DAPSA: Disease Activity index for Psoriatic Arthritis, BASRI: The Bath Ankylosing Spondylitis Radiology Index. [S]values are significant at p<0.05

Table 2: Univariate analysis of PsA patients with and without hyperuricemia.

Variables mean±SD or N (%)	Patients without hyperuricemia (n=35)	Patients with hyperuricemia (n=28)	p value
Gender			
Female	27 (42.9)	23 (36.5)	0.626
Male	8 (12.7)	5 (7.9)	
Age (years)	47.5±9.4	51.2±11.7	0.082
Body mass index (BMI)	28.5±4.9	31.0±5.1	0.045[S]
Smoking			
No	28 (44.4)	22 (34.9)	0.889
Yes	7 (11.1)	6 (9.6)	
Pso duration (year)	14.9±9.8	14.9±11.0	0.835
PsA duration (year)	2.7±2.9	2.9±3.2	0.897
HLA-B27 status			
(-)	30 (47.7)	24 (38.1)	1.000
(+)	5 (7.9)	4 (6.3)	
CRP (mg/dl)	7.6±9.6	6.8±8.8	0.900
PASI	3.8±1.8	4.0±1.6	0.631
BSA			
<%10	18 (28.5)	11 (17.5)	0.337
>%10	17 (27)	17 (27)	

Continued.

DAPSA	25.2±10.0	28.0±10.1	0.391
BASRI-total	5.3±1.8	5.8±2.0	0.351
Musculoskeletal findings			
Enthesitis			
Plantar fascia	28 (44.4)	16 (25.4)	0.472
The Achilles tendon	25 (39.7)	15 (23.8)	0.476
The patellar tendon	10 (15.9)	10 (15.9)	1.000
Iliac crest	3 (4.8)	1 (1.6)	0.508
Greater trochanter	2 (3.2)	0 (0)	1.000
Greater tuberosity (humerus)	2 (3.2)	0 (0)	1.000
Dactylitis	8 (12.7)	2 (3.2)	0.653
Co-morbidities			
Hypertension	10 (15.9)	15 (23.8)	0.044[S]
Diabetes mellitus	11 (17.5)	10 (15.9)	0.720
Hyperlipidemia	4 (6.3)	2 (3.2)	0.565
Coronary artery disease	5 (7.9)	2 (3.2)	0.448
Hypothyroidism	7 (11.1)	6 (9.6)	0.889

Po: Psoriasis, PsA: Psoriatic arthritis, CRP: C-reactive protein, PASI: Psoriasis area and severity index, BSA: Body surface area, DAPSA: Disease Activity index for Psoriatic Arthritis, BASRI: The Bath Ankylosing Spondylitis Radiology Index. [S]values are significant at p<0.05

Tablo 3: The correlations between serum uric acid level and other parameters among gender.

Variables	Serum uric acid level			
	Female		Male	
	r value	p value	r value	p value
Age (years)	0.261	0.067	0.309	0.304
BMI	0.226	0.115	0.455	0.118
Pso duration (years)	0.051	0.727	0.099	0.749
PsA duration (years)	0.056	0.701	0.085	0.783
CRP (mg/dl)	0.065	0.652	0.179	0.560
PASI	0.020	0.889	0.103	0.737
DAPSA	0.266	0.062	0.250	0.410
BASRI-total	0.203	0.157	0.161	0.600

BMI: Body mass index; Po: Psoriasis, PsA: Psoriatic arthritis, CRP: C-reactive protein, PASI: Psoriasis area and severity index, DAPSA: Disease Activity index for Psoriatic Arthritis, BASRI: The Bath Ankylosing Spondylitis Radiology Index

Table 4: The correlations between BASRI score and other parameters.

Variables	BASRI score	
	r value	p value
Age (years)	0.628	<0.001[S]
BMI	0.347	0.005[S]
DAPSA	0.393	0.001[S]
Plantar fascia enthesitis	-	0.015[S]
The Achilles tendon enthesitis	-	0.016[S]
Greater trochanter enthesitis	-	0.031[S]

BASRI: The Bath Ankylosing Spondylitis Radiology Index, BMI: Body mass index; DAPSA: Disease Activity index for Psoriatic Arthritis. [S]values are significant at p<0.05

When the genders of men and women were evaluated within themselves, hyperuricemia was present in 46% of women and 38.5% of men. The mean SUA levels in patients with hyperuricemia were 6.5±0.2 mg/dl and 7.4±0.2 mg/dl in females and males, respectively. PsA patients with hyperuricemia had a high significant difference in BMI and hypertension (Table 2). There was no significant difference in terms of the correlations between SUA level and other parameters among gender (Table 3). BASRI score was significantly related to age, body mass index, DAPSA score, plantar fascia, greater trochanter, and the Achilles enthesitis (Table 4). On multivariate logistic regression analysis, only BMI was associated with SUA level (p=0.043) (Table 5).

Table 5: Logistic regression analysis between patients with and without hyperuricemia.

Independent variable	Odd ratio (%95 CI)	p value
Gender (female)	1.19 (0.31-4.64)	0.802
Body mass index	1.03 (0.93-1.15)	0.043[S]
Hypertension	0.36 (0.12-1.15)	0.085

[S]values are significant at p<0.05

DISCUSSION

PsA is a disease that can cause severe joint damage and deformities and can significantly influence function, quality of life, and work productivity. Due to excessive keratinocyte turnover in psoriatic plaques as well as systemic inflamation, high SUA levels can be detected in patients with Pso compared to healthy individuals. Because of the water-insoluble nature of uric acid, its concentration is tightly controlled in humans by reabsorption in the proximal tubule. Uric acid crystals, which precipitate in the tissue when exceeding the saturated concentration, act as a molecular model of injury, stimulating the innate immune system, subsequently increasing the production of tumor necrosis factor-alpha, interleukin (IL)-1, IL-18, IL-8/kemokin (C-X-C motifi) ligand 8 (CXCL8), and IL-6. It also stimulates T helper 17 cells and increases IL-17 production. Individuals whose innate immune systems are extremely sensitive to very small urate crystals may be prone to contracting Pso, suggesting that uric acid may be involved in the pathogenesis of PsA. On the other hand, since metabolic comorbidities such as obesity, hypertension, insulin resistance, diabetes, renal disease and atherosclerosis are more common in psoriatic patients than in the normal population, hyperuricemia can be considered as one of the determining factors for such symptoms.[10,26] Therefore, this study investigated the frequency of hyperuricemia and associated risk factors among PsA patients. Hyperuricemia was present in 44.4% of patients. Moreover, PsA patients with hyperuricemia had a high significant difference in BMI and hypertension.

Lambert et al evaluated SUA levels in patients with PsA who received and did not receive medical therapy, in patients with rheumatoid arthritis (RA) who did not use any medication that could alter serum levels, and in patients with uncomplicated Pso. They found higher values in men than in women, especially men with PsA who used uncontrolled medication (13.5%) and men with RA (12%).[27] This situation was similar in this study.

Bruce et al. evaluated the prevalence of hyperuricemia and the effect of skin involvement on SUA levels in patients with PsA. They detected hyperuricemia in 20.7% of PsA patients. They also found that SUA was not associated with the degree of skin involvement.[28] Tsuruta et al investigated which psoriatic patients were most at risk of developing PsA. They detected hyperuricemia in 22% of PsA patients. They found that PsA patients had significantly higher frequencies of nail lesions and hyperuricemia.[26] Lai et al investigated the prevalence of hyperuricemia and associated risk factors in patients with PsA. They detected hyperuricemia in 30.6% of PsA patients. They also found that hyperuricemia not associated with the duration of skin disease, skin and joint involvement, dactylitis, enthesitis.[11] Gudu et al evaluated the prevalence of hyperuricemia in PsA patients and identified the associated factors. They detected hyperuricemia in 27.5% of PsA patients. They also found that there was no correlation between hyperuricemia and skin involvement.[29] AlJohani et al investigated the prevalence of psoriatic patients with hyperuricemia and the adverse effects of hyperuricemia, especially in terms of cardiovascular (CVD) and renal diseases. They detected hyperuricemia in 31.9% of patients with PsA. Patients with hyperuricemia had longer disease duration, higher PASI scores.[30] Chu et al determined the relationship between disease activity measured by different composite scores and SUA levels in patients with PsA. They detected hyperuricemia in 26.9% of PsA patients. They also found that waist circumference was significantly higher in hyperuricemic patients but BMI was similar between groups.[31] Widawski et al investigated the impact of hyperuricemia on PsA in its clinical presentation, its severity, and associated comorbidities. They detected hyperuricemia in 30.2% of PsA patients.[32] Dehlin et al investigated whether PASI score were correlated serum urate levels at baseline and whether a change in PASI score after 12 weeks of treatment resulted in a change in SUA levels in patients with PsA. They found a statistically significant, albeit modest, relationship between PASI score and SUA level. They also found a statistically significant modest relationship between a substantial improvement in PASI score and SUA level after 12 weeks of treatment with secukinumab.[33] This study detected hyperuricemia in 44.4% of PsA patients. This was higher than previously reported by the above studies. This may be due to the higher BMI values of patients included in the study. While some of the above studies found a relationship between the degree of skin involvement (PASI score) and hyperuricemia, no relationship was found in some of them.[30,33,11,26,28,29] In this study, there was no significant relationship between skin involvement and hyperuricemia.

Bruce et al found SUA to be associated with metabolic changes such as hypercholesterolemia and kidney failure.[28] Tsuruta et al compared patients with and without PsA in terms of age, gender, age of onset, BMI, smoking and drinking habits, family history of Pso, and comorbidities. They found no difference in age of onset, gender, BMI, and incidence of diabetes mellitus, hypertension, or dyslipidemia.[26] Lai et al found moderately positive correlations between hyperuricemia and BMI, but not lipid profile, renal function or creatinine clearance.[11] Gudu et al. found that hyperuricemia was significantly associated with obesity, diabetes, ischemic heart disease and hypertension.[29] AlJohani et al found that patients with hyperuricemia had obesity and more concurrent comorbidities, including CVD (especially myocardial infarction and congestive heart failure) and metabolic diseases, as well as higher prevalence of kidney stones and higher creatinine.[30] Widawski et al found that hyperuricemia was significantly associated with male sex, higher BMI and age at PsA onset, metabolic syndrome, high blood pressure, type 2 diabetes, ischemic stroke, acute coronary syndrome, moderate or severe chronic renal failure, and peripheral PsA involvement.[32] Gonzalez-Gay et al. investigated whether SUA level correlated with

carotid intima-media wall thickness (CIMT) in PsA patients without cardiovascular CVD or classic CV risk factors. They detected hyperuricemia in 11.5% of patients with PsA. Patients with hyperuricemia also had higher levels of serum creatinine, glucose, total cholesterol, and triglycerides. Higher CIMT was detected in PsA patients with hyperuricemia than in those without hyperuricemia. They showed a significant association between high SUA level and subclinical atherosclerosis in patients with PsA, without CVD risk factors.[34] Ibrahim et al also evaluated the relationship between SUA level and CIMT in patients with PsA. They detected hyperuricemia in 26.6% of patients with PsA. They found that PsA patients with hyperuricemia had a high significant difference in CIMT. SUA levels showed a high significant positive correlation with each of CIMT, disease duration, markers of inflammation (erythrocyte sedimentation rate, CRP, IL-6), disease activity score in 28 joints (DAS 28) and PASI.[17] This study found a significant relationship between hyperuricemia and only hypertension in terms of metabolic disease. This may be due to the small sample size.

The study has some limitations such as small sample size, single-center design, no control group, the absence of assessment of tender and swollen joints with nail involvement, the absence of dietary and drinking habits, and the lack of evaluation of SUA levels before and after medical treatment. However, this study provides useful information about early detection and management of hyperuricemia in PsA patients given the potential for hyperuricemia to exacerbate both the clinical course of PsA and related conditions. Moreover, adipose tissue produces pro-inflammatory cytokines, which can exacerbate both psoriasis and arthritis, potentially influencing uric acid metabolism. Since obesity is strongly associated with the onset and exacerbation of Pso and PsA in addition to increased SUA levels (35), weight management, dietary modifications (e.g., reducing purine-rich foods), and increased hydration may also be important in patients with PsA.

CONCLUSION

The majority of PsA patients had asymptomatic hyperuricemia. Moreover, hyperuricemia was associated with BMI and hypertension, but not other parameters. The higher rate of hyperuricemia in women than men seems to be due to their higher BMI values.

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REFERENCES

1. Ritchlin CT, Colbert RA, Gladman DD. Psoriatic arthritis. N Engl J Med. 2017;376(10):957-70.
2. Alinaghi F, Calov M, Kristensen LE, Gladman DD, Coates LC, Jullien D, et al. Prevalence of psoriatic arthritis in patients with psoriasis: A systematic review and meta-analysis of observational and clinical studies. J Am Acad Dermatol. 2019;80(1):251-65.e19.
3. Mistegård J, Gudbjornsson B, Lindqvist U, Laasonen L, Ejstrup L, Ståhle M, et al. Comorbidities in a cohort of 66 patients with psoriatic arthritis mutilans-results from the Nordic PAM Study. Front Med (Lausanne). 2021:8:629741.
4. Eder L, Gladman DD. Psoriatic arthritis: phenotypic variance and nosology. Curr Rheumatol Rep. 2013;15(3):316.
5. 5. Ohara Y, Kishimoto M, Takizawa N, Yoshida K, Okada M, Eto H, et al. Prevalence and clinical characteristics of psoriatic arthritis in Japan. J Rheumatol. 2015;42(8):1439-42.
6. Kerschbaumer A, Fenzl KH, Erlacher L, Aletaha D. An overview of psoriatic arthritis - epidemiology, clinical features, pathophysiology and novel treatment targets. Wien Klin Wochenschr. 2016;128(21-22):791-5.
7. Song Z, Deng X, Xie W, Li B, Zhang Z. Clinical Characteristics of Psoriatic Arthritis in Chinese Patients: A Cross-Sectional Study. Rheumatol Ther. 2021;8(4):1845-57.
8. ElSherbiny DA, Elsayed AMA, El-Azizi NO, Ghalwash RA. Clinical and laboratory characteristics of psoriatic arthritis in a cohort of Egyptian patients. Egypt Rheumatol. 2021;43(3):229-34.
9. Sulaiman W, Tan LK, Mat H, Tohar N, Fati AM, Kosenin NMA, et al. Clinical characteristics and comorbidities in psoriatic arthritis: Experience from a single rheumatology centre in Malaysia. Egypt Rheumatol. 2023;45(4):309-13.
10. Tripolino C, Ciaffi J, Ruscitti P, Giacomelli R, Meliconi R, Ursini F. Hyperuricemia in Psoriatic Arthritis: Epidemiology, Pathophysiology, and Clinical Implications. Front Med (Lausanne). 2021;8:737573.
11. Lai TL, Yim CW, Wong PY, Leung MC, Ng WL. Hyperuricemia in Asian psoriatic arthritis patients. Int J Rheum Dis. 2018;21(4):843-9.
12. Raychaudhuri SP. Comorbidities of psoriatic arthritis -metabolic syndrome and prevention: a report from the GRAPPA 2010 annual meeting. J Rheumatol. 2012;39(2):437-40.
13. Armstrong AW, Read C. Pathophysiology, clinical presentation, and treatment of psoriasis: a review. JAMA. 2020;323(19):1945-60.
14. Woo YR, Park CJ, Kang H, Kim JE. The risk of systemic diseases in those with psoriasis and psoriatic arthritis: from mechanisms to clinic. Int J Mol Sci. 2020;21(19):7041.
15. Gupta S, Syrimi Z, Hughes DM, Zhao SS. Comorbidities in psoriatic arthritis: a systematic review and meta-analysis. Rheumatol Int. 2021;41(2):275-84.

16. Smiyan SI, Bilukha AV, Koshak BO. Modern determinants of cardiovascular risk factors in patients with psoriatic arthritis: Relation to disease activity and severity. Egypt Rheumatol. 2024;46(1):1-5.
17. Ibrahim SE, Helmi A, Yousef TM, Hassan MS, Farouk N. Association of asymptomatic hyperuricemia and endothelial dysfunction in psoriatic arthritis. Egypt Rheumatol. 2012;34(2):83-9.
18. Felten R, Duret PM, Gottenberg JE, Spielmann L, Messer L. At the crossroads of gout and psoriatic arthritis: "psout". Clin Rheumatol. 2020;39(5):1405-13.
19. Ataseven A, Kesli R, Kurtipek GS, Ozturk P. Assessment of lipocalin 2, clusterin, soluble tumor necrosis factor receptor-1, interleukin-6, homocysteine, and uric acid levels in patients with psoriasis. Dis Markers. 2014;2014:541709.
20. Solak B, Dikicier BS, Erdem T. Impact of elevated serum uric acid levels on systemic inflammation in patients with psoriasis. Angiol. 2017;68(3):266-70.
21. Taylor W, Gladman D, Helliwell P, Marchesoni A, Mease P, Mielants H. Classification criteria for psoriatic arthritis: development of new criteria from a large international study. Arthritis Rheum. 2006;54(8):2665-73.
22. Ramsay B, Lawrence CM. Measurement of involved surface area in patients with psoriasis. Br J Dermatol. 1991;124:565-70.
23. 23. Fredriksson T, Pettersson U. Severe psoriasis-oral therapy with a new retinoid. Dermatologica. 1978;157:238-44.
24. Schoels M, Aletaha D, Funovits J, Kavanaugh A, Baker D, Smolen JS. Application of the DAREA/DAPSA score for assessment of disease activity in psoriatic arthritis. Ann Rheum Dis. 2010;69(8):1441-7.
25. MacKay K, Mack C, Brophy S, Calin A. The Bath Ankylosing Spondylitis Radiology Index (BASRI): a new, validated approach to disease assessment. Arthritis Rheum. 1998;41(12):2263-70.
26. Tsuruta N, Imafuku S, Narisawa Y. Hyperuricemia is an independent risk factor for psoriatic arthritis in psoriatic patients. J Dermatol. 2017;44(12):1349-52.
27. Lambert JR, Wright V. Serum uric acid levels in psoriatic arthritis. Ann Rheum Dis. 1977;36(3):264-7.
28. Bruce IN, Schentag CT, Gladman DD. Hyperuricemia in psoriatic arthritis: prevalence and associated features. J Clin Rheumatol. 2000;6(1):6-9.
29. Gudu T, Peltea A, Balanescu A, Bojinca V, Opris-Belinski D, Predeteanu D, et al. Hyperuricemia in psoriatic arthritis: prevalence and associated factors. Ro J Rheumatol. 2017;26(4):160-3.
30. AlJohani R, Polachek A, Ye JY, Chandran V, Gladman DD. Characteristic and outcome of psoriatic arthritis patients with hyperuricemia. J Rheumatol. 2018;45(2):213-7.
31. Chu BBR, Pereira da Cunha JPVV, Marcon JMT, Eduarda de Arruda Prado L, Rosa FLS, Leitão JM, et al. Does disease activity influence the levels of uric acid in psoriatic arthritis? Open Rheumatol J. 2021;15(1):57-64.
32. Widawski L, Fabacher T, Spielmann L, Gottenberg JE, Sibilia J, Duret PM, et al. Psoriatic arthritis with hyperuricemia: more peripheral, destructive, and challenging to treat. Clin Rheumatol. 2022;41(5):1421-9.
33. Dehlin M, Fasth AER, Reinhardt M, Jacobsson LTH. Impact of psoriasis disease activity and other risk factors on serum urate levels in patients with psoriasis and psoriatic arthritis-a post-hoc analysis of pooled data from three phase 3 trials with secukinumab. Rheumatol Adv Pract. 2021;5(1):rkab009.
34. Gonzalez-Gay MA, Gonzalez-Juanatey C, Vazquez-Rodriguez TR, Gomez-Acebo I, Miranda-Filloy JA, Paz-Carreira J, et al. Asymptomatic hyperuricemia and serum uric acid concentration correlate with subclinical atherosclerosis in psoriatic arthritis patients without clinically evident cardiovascular disease. Semin Arthritis Rheum. 2009;39(3):157-62.
35. Kaine J, Song X, Kim G, Hur P, Palmer JB. Higher incidence rates of comorbidities in patients with psoriatic arthritis compared with the general population using U.S. Administrative Claims Data. J Manag Care Spec Pharm. 2019;25(1):122-32.

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