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"Psout": The clinical intersection of psoriatic arthritis and gout

"Psout": Psoriatik artrit ve gut hastalığının klinik kesişimi

Sadettin Uslu, Özgül Soysal Gündüz, Timur Pırıldar

Celal Bayar University Faculty of Medicine, Department of Internal Medicine, Division of Rheumatology, Manisa, Türkiye

Abstract

The coexistence of gout and psoriatic arthritis (PsA) has been recognized for many years; however, increasing awareness of their shared comorbidities and overlapping clinical phenotypes has led to more frequent identification of this association in recent clinical practice. This overlap presents several diagnostic and therapeutic challenges, particularly in terms of differential diagnosis and the need for individualized treatment strategies. Epidemiological studies have demonstrated an increased risk of developing gout in patients with both cutaneous psoriasis and PsA. Gout and PsA share common risk factors, suggesting the presence of underlying and closely interconnected pathophysiological mechanisms. This review explores the intersection between gout and PsA in the context of shared clinical features, common inflammatory pathways, and associated comorbidities. Moreover, the emerging concept of a novel "overlap syndrome" termed "Psout" is discussed. This new framework may offer meaningful contributions to clinical practice by improving diagnostic accuracy and optimizing therapeutic approaches.

Keywords: Psoriasis, psoriatic arthritis, hyperuricemia, gout, psout

Özet

Gut ve psoriatik artrit (PsA) birlikteliği uzun süredir bilinmekle birlikte, ortak komorbiditeler ve benzer klinik fenotiplere yönelik artan farkındalık nedeniyle son yıllarda klinik pratikte daha sık karşılaşılmaktadır. Bu durum, tanı sürecinde ayrıştırma güçlükleri ve tedavi stratejilerinin bireyselleştirilmesi açısından çeşitli zorluklara yol açmaktadır. Epidemiyolojik çalışmalar hem kutanöz psoriasisli hem de PsA'lı hastalarda gut gelişme riskinin arttığını göstermektedir. PsA ve gut, ortak risk faktörlerine sahip olup, bu durum altta yatan ve birbiriyle sıkı bir şekilde bağlantılı patofizyolojik mekanizmaların varlığını düşündürmektedir. Bu derlemede, gut ve PsA arasındaki örtüşme; paylaşılan klinik özellikler ortak enflamatuvar süreçler ve eşlik eden komorbiditeler bağlamında ele alınmakta ve yeni bir "örtüşen sendrom" olarak "Psout" adıyla yeni bir tanımlama tartışılmaktadır. Bu yeni yaklaşım, klinik pratiğe tanı doğruluğu ve tedavi optimizasyonu açısından anlamlı katkılar sunabilir.

Anahtar Kelimeler: Psöriasis, psöriatik artrit, hiperürisemi, gut, psout

Introduction

Although the coexistence of gout and psoriasis/psoriatic arthritis (PsA) has long been recognized, increasing awareness in recent years regarding their shared clinical features and comorbidities has led to more frequent identification of this overlap in clinical practice.[1] This has introduced various challenges in the diagnostic and therapeutic processes.

Gout and PsA are common forms of inflammatory arthritis, both of which are chronic inflammatory diseases associated with significant morbidity. Gout is the most prevalent form of inflammatory arthritis, with its prevalence varying across different geographic regions and ethnic groups. For instance, prevalence rates have been reported as 2.3% in the United Kingdom, 5.1% in the United States, and 3.7% in Canada.[2,3] The highest prevalence is observed among certain ethnic populations, such as the Indigenous peoples of Taiwan (10.4-15.2%) and the Maori population (6.1%).[4,5] PsA, on the other hand, is a seronegative chronic inflammatory arthritis that may involve axial and/or peripheral joints. While its prevalence in the general population ranges from 0.1% to 1%, it increases to 20-30% among individuals with cutaneous psoriasis.[6,7] These findings highlight PsA as one of the most common systemic comorbidities associated with psoriasis and emphasize the significant public health burden posed by both gout and PsA.

Correspondence / İletişim: Saadettin Uslu Assoc. Prof.,
Celal Bayar University Faculty of Medicine, Department of Internal Medicine, Division of Rheumatology, Manisa, Türkiye
E-mail: sadouslu@gmail.com **ORCID ID:** orcid.org/0000-0001-6266-2454
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Epidemiological studies indicate that individuals affected by both psoriasis and gout share similar demographic characteristics, lifestyle factors, and metabolic comorbidities (e.g., obesity, hypertension, dyslipidemia, type 2 diabetes).[8-10] Cross-sectional studies have shown an increased risk of developing gout among patients with psoriasis and PsA. These shared risk factors suggest the presence of underlying and potentially interconnected pathophysiological mechanisms.[11]

From clinical, pathophysiological, and radiographic perspectives, PsA and gout may exhibit overlapping features. Particularly in advanced disease stages, it may become difficult to distinguish between a gout flare and a PsA exacerbation based on clinical presentation alone. Moreover, the topographic similarities in joint involvement patterns further complicate the diagnostic process.[1] Table 1 presents the comparative features of PsA and gout. The coexistence of both diseases in a single patient is not uncommon in clinical practice. In this context, the term "Psout" was first introduced by Felten et al.[12] in 2020 as a descriptive concept for patients presenting with both PsA and gout. The authors derived this definition from a concrete case experience (Figure 1). The term "Psout" not only reflects the coexistence of these two diseases but also implies potential shared pathophysiological pathways. Therefore, in patients with concurrent PsA and gout, achieving an accurate diagnosis and developing an effective treatment plan require an integrated evaluation of both inflammatory and metabolic components. Figure 2 illustrates the general clinical and pathophysiological characteristics of Psout.

This narrative review aims to provide a comprehensive overview of "Psout", a concept representing the clinical, pathophysiological, and epidemiological intersection of gout and PsA, and to raise awareness of its clinical relevance in light of the current scientific literature.

Shared Pathogenesis Between Psoriatic Disease and Gout

There are overlapping pathophysiological mechanisms at the molecular level between PsA and gout. In both diseases, hyperuricemia acts as both a cause and a consequence of the inflammatory process. Hyperuricemia results from either increased uric acid production or decreased excretion and is closely linked to purine metabolism, intestinal transport systems, and hepatic function.[13]

Damage-associated molecular patterns, such as microbial peptides found in psoriatic plaques, keratinocyte-derived alarmins, and monosodium urate (MSU) crystals characteristic of gout, stimulate plasmacytoid dendritic cells and trigger the release of proinflammatory cytokines including interleukin (IL)-1β, IL-6, tumour necrosis factor alpha (TNF-α), and interferon (IFN)-γ. This inflammatory cascade continues with the activation of Th1 and Th17 cells via dendritic cell signaling. IL-17 and IFN-γ secreted by Th17 cells re-activate keratinocytes, leading to the establishment of a self-perpetuating autoinflammatory loop.[12,14,15] This same cytokine profile may contribute to the development of non-alcoholic fatty liver disease and elevate serum uric acid (SUA) levels due to ATP depletion in hepatocytes. Furthermore, the subclinical intestinal inflammation reported in PsA may impair uric acid homeostasis by disrupting the function of enterocytic transporters such as BCRP/ABCG2.[11,13]

In gout, phagocytosis of MSU crystals activates the NLRP3 inflammasome, which in turn enhances IL-1β production through caspase-1 activation. In addition, MSU crystals induce NETosis in neutrophils, activating innate immunity and promoting Th17 polarization through antigen presentation. Consequently, IL-17, IL-1, and other proinflammatory cytokines stimulate keratinocyte proliferation and accelerate purine metabolism, reinforcing the hyperuricemia-gout cycle.[12,15]

Table 1. Comparative clinical characteristics of psoriatic arthritis and gout

Feature	Psoriatic arthritis	Gout
Age/sex at onset	30-50 years, equal in males and females	>40 years, predominantly males
Pattern of joint involvement	Generally asymmetric	Generally asymmetric
Number of affected joints	Most commonly oligoarticular	Typically monoarticular or oligoarticular
Associated symptoms	Periarticular erythema is common	Periarticular erythema may occur
Cutaneous and nail manifestations	Characterized by psoriatic plaques, nail pitting, and onycholysis	Presence of tophi in chronic disease stages
Predominant sites (hands and feet)	Predominantly distal joints	Predominantly distal joints
Dactylitis (diffuse digital swelling)	A common and characteristic clinical feature	Occasionally observed
First metatarsophalangeal joint involvement	May be affected	Frequently affected and often the initial site of involvement
Axial/spinal involvement	Common; includes sacroiliitis and syndesmophyte formation	Rarely observed
Hyperuricemia	Observed in approximately 30% of patients	Common, with fluctuations during acute attacks
Monosodium urate crystal deposition (joint fluid)	Reported in ~3.3% of cases	Characteristic but variable among patients
Radiographic features	Erosions with new bone formation	"Punched-out" erosions, tophi

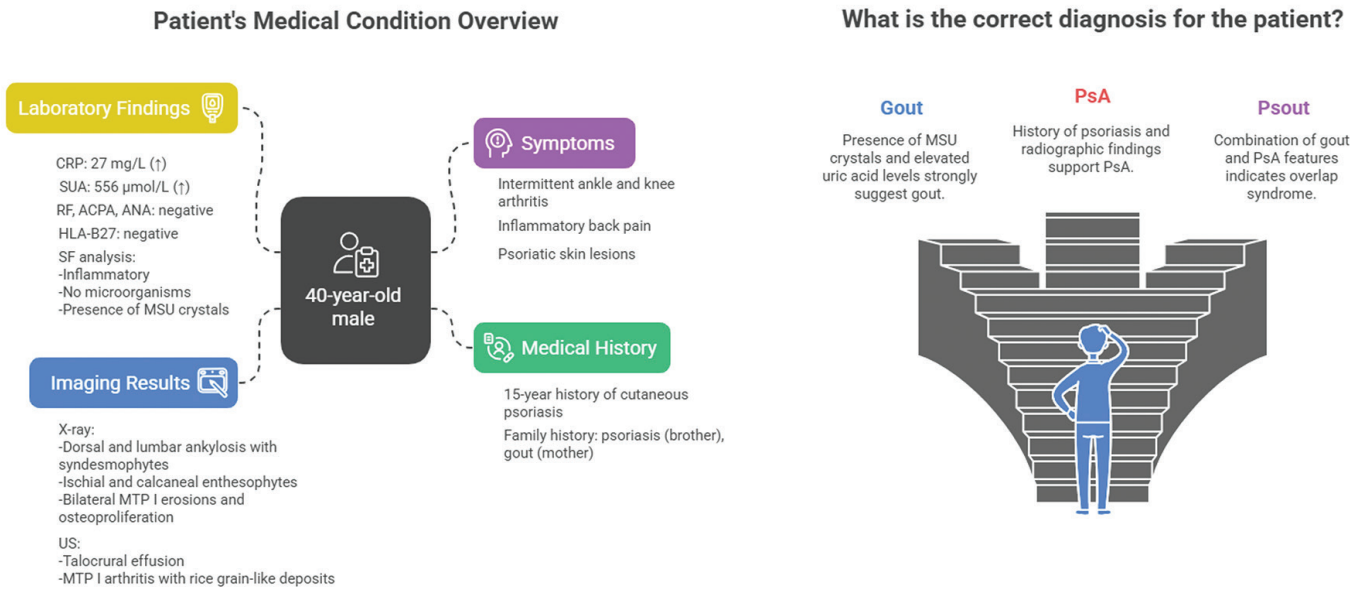

Figure 1. Diagnostic dilemma in a patient with psoriasis, arthritis, and hyperuricemia: gout, psoriatic arthritis, or the emerging overlap entity "Psout"
ACPA: Anti-citrullinated protein antibody, ANA: Antinuclear antibody, CRP: C-reactive protein, HLA: Human leukocyte antigen, MSU: Monosodium urate, MTP: Metatarsophalangeal, PsA: Psoriatic arthritis, RF: Rheumatoid factor, SF: Synovial fluid, SUA: Serum uric acid, US: Ultrasound

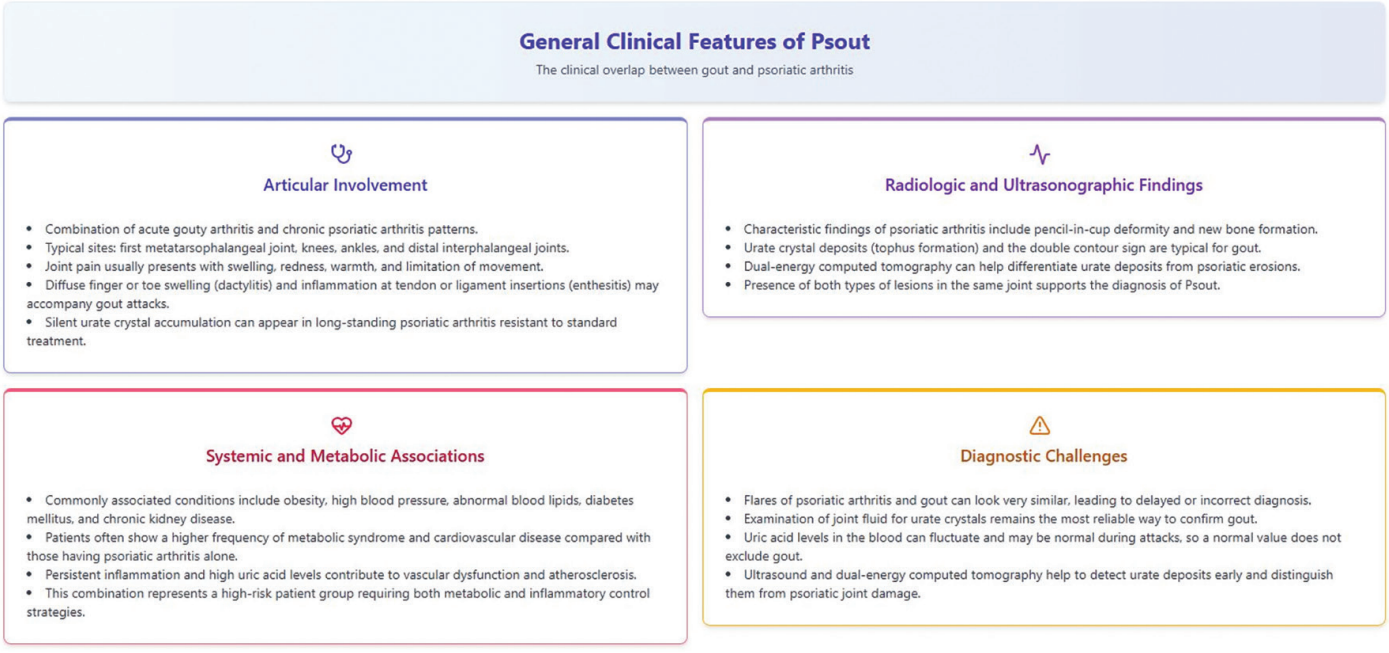

Figure 2. General clinical characteristics of Psout

In conclusion, the potential of MSU crystals to activate the IL-23/IL-17 axis and keratinocyte-T cell interactions may contribute to the emergence of "Psout." This condition is characterized by a more severe, widespread, and destructive clinical course.[1,12]

Hyperuricemia in PsA

Hyperuricemia is approximately three times more prevalent in patients with PsA compared to the general population.[16] In PsA, accelerated cutaneous cell turnover and chronic systemic inflammation may contribute to elevated SUA levels. The global prevalence of hyperuricemia in patients with psoriasis and/or PsA has been reported to range between 13% and 40.7%.[9,11] Although the potential association between psoriasis and hyperuricemia was first described by Walker in 1958, subsequent studies have yielded inconsistent results.[17] A meta-analysis including 14 studies and a total of 29.416 participants revealed

that the association between hyperuricemia and psoriasis varies according to ethnicity and geographic region. While a significant positive correlation was found in Western Europe, similar associations were not observed in Asian or Middle Eastern populations. These discrepancies are thought to be attributable to differences in study design, characteristics of the target populations, the presence of concomitant PsA, and varying levels of disease severity.[18]

Several studies have demonstrated a significant association between hyperuricemia and psoriasis, and some have shown that SUA levels increase in parallel with the extent of skin involvement. Hyperuricemia is proposed not only as a metabolic abnormality but also as a potential independent risk factor for the development of PsA.[13,19] Supporting this hypothesis, a retrospective case-control study conducted in Japan analyzed data from 331 patients and found that the prevalence of hyperuricemia was significantly higher in patients with PsA compared to those with psoriasis alone (22% vs. 9%). Regression analysis further identified hyperuricemia as an independent predictor for the development of PsA [odds ratio (OR) 4.18, 95% confidence interval (CI) 1.60-10.96]. These findings underscore the potential role of hyperuricemia within the psoriatic disease spectrum.[20]

PsA and Gout

The reported prevalence of gout among patients with PsA varies significantly across studies. In a Canadian cohort of 265 patients followed for six years, the incidence was 0.8%, whereas in another cohort predominantly composed of white males, this rate increased to 8.6% over a median follow-up of 19.5 years.[21,22] Retrospective case-control data from France also confirmed a notably higher prevalence of gout in male PsA patients (6.2%) compared to the general population (0.9%).[23] Compared to normouricemic cases, hyperuricemic PsA patients had significantly higher proportions of male sex (72.6% vs. 39.1%), higher body mass index (BMI) (30.9 vs. 28.7 kg/m²), and greater comorbidity burden (Charlson index 2.6 vs. 1.8). Multivariate analyses revealed that hyperuricemia was independently associated with male sex, hypertension, moderate-to-severe chronic kidney disease, prior PUVA therapy, peripheral joint involvement, and poor treatment response. Hyperuricemia was found to triple the risk of peripheral PsA (OR 2.98, 95% CI: 1.15-7.75) and significantly reduce the likelihood of good therapeutic response (OR 0.35, 95% CI: 0.15-0.87). A ten-year retrospective French dataset demonstrated that hyperuricemia was associated with a more polyarticular and destructive PsA phenotype, with erosions observed more frequently in hyperuricemic patients (43.7% vs. 28%), particularly when SUA levels exceeded 300 µmol/L. However, it was suggested that this association might be partially exaggerated due to more frequent SUA testing in treatment-resistant cases. Furthermore, hyperuricemic PsA patients tended to be older, predominantly male, and had later disease onset.

In a Canadian prospective cohort study, 1019 patients with psoriasis and/or PsA were followed for 6 to 12 months to assess the impact of hyperuricemia on disease characteristics and comorbidities. Hyperuricemia was detected in 325 patients (35.9%), yet only 11 (3.4%) developed gout. Hyperuricemic patients had significantly longer PsA disease duration, and higher Psoriasis Area and Severity Index (PASI) scores compared to normouricemic individuals, although no significant differences were observed in tender or swollen joint counts. In a parallel case-control analysis of 318 hyperuricemic PsA patients matched for age, sex, and disease duration with 318 normouricemic PsA patients, hyperuricemia was associated with increased prevalence of cardiovascular and metabolic diseases, nephrolithiasis, and higher serum creatinine levels.[24] Persistent hyperuricemia during follow-up was found to significantly increase the incidence of myocardial infarction, heart failure, and renal dysfunction, underscoring its potential role not only in joint disease but also in systemic cardio-renal morbidity in PsA.

A large-scale Taiwanese cohort study including 114.623 individuals with gout and 114.623 controls demonstrated a strong association between gout and PsA (adjusted OR=2.50; 95% CI: 1.95-3.22), while the association between gout and psoriasis alone was weaker (adjusted OR=1.30; 95% CI: 1.20-1.42).[19] Prospective data from the US-based Health Professionals Follow-Up Study and Nurses' Health Study cohorts support these findings, showing that physician-diagnosed psoriasis and PsA independently increased the risk of incident gout [multivariable hazard ratio (HR) for psoriasis=1.71; 95% CI: 1.36-2.15; for PsA=4.95; 95% CI: 2.72-9.01].[25] Gout diagnoses were confirmed using the 1977 American College of Rheumatology classification criteria. In sex-stratified analyses, psoriasis alone conferred a higher risk of gout in men than in women (HR=2.72; 95% CI: 1.75-4.25); however, this difference was no longer significant when PsA was present, likely due to the comparable prevalence of PsA between sexes and the shared inflammatory/metabolic burden equalizing the risk.

Another population-based cohort study from Taiwan revealed that individuals with both psoriasis and gout had a significantly higher risk of cardiovascular disease compared to those with psoriasis alone (relative risk=2.39), highlighting the substantial cardiovascular burden associated with gout as a comorbidity within the psoriatic disease spectrum.[26]

MSU Crystals in PsA

Even in patients without a clinical diagnosis of gout, MSU crystals can be frequently detected in synovial fluid (SF), supporting the possibility of a biological overlap between PsA

and gout. In a large-scale analysis of 5,020 SF samples, MSU crystals were identified in 3.34% of PsA cases, compared to 0.30% in rheumatoid arthritis, 0.70% in other spondylarthritis, and 0.80% in calcium pyrophosphate deposition disease.[27] Galozzi et al.[28] reported that MSU crystals, rather than hyperuricemia, may be a stronger trigger for the PsA-gout overlap (Psout). In their initial study analyzing seven categories of arthritis, the prevalence of MSU crystals was found to be 83.3% in gout and 10% in PsA. A subsequent, broader study involving ten arthritis categories confirmed these findings, with MSU crystal prevalence rates of 97.96% in gout and 3.34% in PsA.[29]

In another case-control series, MSU crystals were found in 68.6% of PsA patients, with crystal-positive cases being more frequently associated with metabolic comorbidities such as obesity, diabetes, ischemic heart disease, and dyslipidemia. Moreover, the presence of crystals was strongly correlated with disease activity (OR: 15.96; 95% CI: 5.76-44.23).[30] Age and sex distributions were similar between crystal-positive and crystal-negative PsA subgroups, emphasizing the potential role of MSU crystals in PsA pathophysiology and the importance of considering metabolic risk profiles in clinical management.

It can be challenging to distinguish acute monoarthritis flares in PsA from crystal-induced gout attacks. Although the presence of pathogenic crystals and data on SF inflammation can help reduce misclassification, they may still be insufficient to determine the exact etiology of acute joint swelling.[31] In a retrospective Italian series of 213 PsA patients, the overall prevalence of MSU crystals was 2.4%, increasing to 10.5% in the hyperuricemic subgroup. Hyperuricemic patients were typically older and predominantly male.[29] However, hyperuricemia was not significantly associated with SF inflammatory features (white blood cell counts <2,000, 2,000-5,000, or 5,000-50,000/mm^3), and crystals were mostly observed in specimens with low-grade inflammation. The presence of MSU crystals in normouricemic patients may reflect transient SUA reductions during acute gout attacks. As the study only included patients presenting with joint swelling and lacked SUA measurements, PsA flares or hyperuricemia may have been underdiagnosed. Indeed, three crystal-positive patients were normouricemic but had high percentages of polymorphonuclear leukocytes in the SF (50-90%).

In a retrospective case-control study, 156 PsA patients and 50 gonarthrosis (GoA) controls were compared using pain (visual analog scale), disease activity (disease activity in psoriatic arthritis, PASI, modified composite psoriatic disease activity index), and functional limitation (health assessment questionnaire - disability index) measures. SF crystal analysis revealed that 23.7% of PsA patients had detectable crystals, while none were identified in the GoA group ($p<0.001$). Among PsA patients, 67.6% of the crystals were MSU and 21.6% were calcium pyrophosphate. Crystal-positive PsA patients had higher prevalence of ischemic heart disease and hyperuricemia, and crystal presence was significantly associated with increased disease activity, severe pain, and marked functional impairment. Notably, the OR for severe pain associated with crystal presence was 157.25 (95% CI: 39.50-625.94). These findings suggest that synovial crystals may be linked to heightened inflammatory burden and reduced quality of life in PsA and support the consideration of SF analysis and urate-lowering therapy in patients with active disease.[30]

Differentiating Gout and PsA Using Imaging Modalities

The 2016 The European Alliance of Associations for Rheumatology guidelines emphasized the identification of MSU crystals in SF as the gold standard for the diagnosis of gout. The 2018 update highlighted the diagnostic value of joint ultrasonography (US) in cases where joint aspiration is not feasible, and the clinical presentation is atypical. Indeed, subclinical MSU crystal deposits detectable by US have been reported in 15-25% of individuals with asymptomatic hyperuricemia.[11] In a single-center, cross-sectional observational study conducted in patients followed with a diagnosis of PsA, coexisting gout was identified in approximately 10% of cases, and it was demonstrated that SUA measurement combined with US screening may help predict gout risk in PsA patients.[32] Moreover, the association of concomitant gout with increased cardiovascular morbidity in PsA underscores the critical importance of early gout screening and personalized treatment strategies in disease management.

Classical radiographic features of PsA—such as periarticular and diaphyseal periostitis, “pencil-in-cup” deformities due to osteolysis, acro-osteolysis, ankylosis, and spondylitis—offer important clues for differentiating chronic PsA from chronic gouty arthritis. However, in rare cases of spinal gout, sacroiliitis may develop, mimicking axial involvement seen in PsA.[33] In advanced gout arthritis, bone erosions and complete joint ankylosis may be observed, and intraosseous peripheral tophi may resemble the marginal erosions typical of PsA (Figure 3). Additionally, both conditions may present with enthesopathy or tophus formation in the Achilles tendon and plantar fascia, further complicating the clinical distinction. The characteristic bone proliferation and distal interphalangeal joint involvement in PsA facilitate differentiation from rheumatoid arthritis, while the marginal erosions in PsA—often associated with periostitis—contrast with the central erosions seen in erosive osteoarthritis (Figure 4). These marginal erosions can form sharp protrusions resembling “mouse ears”, producing the so-called “Mickey Mouse” sign in imaging.[12]

US can provide information on both synovitis and crystal deposition by identifying features such as the double contour sign, MSU aggregates, and tophi. In contrast, dual-energy computed tomography (DECT) directly visualizes crystal deposits.

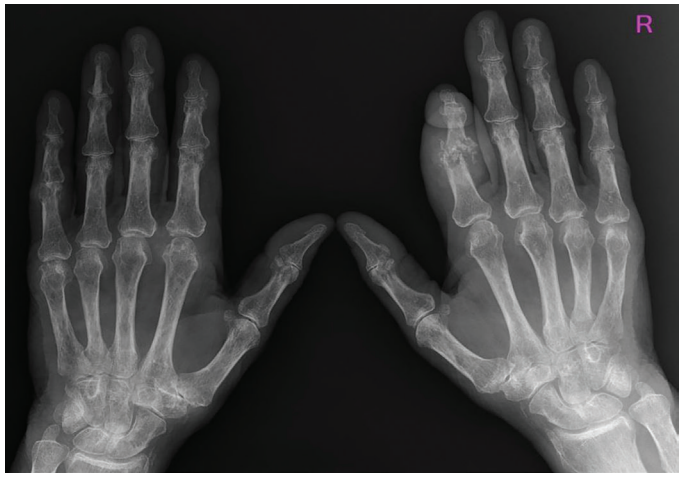

Figure 3. Radiographic features of gout showing bone erosions, ankylosis, and intraosseous tophi mimicking psoriatic marginal erosions (from the authors' collection)

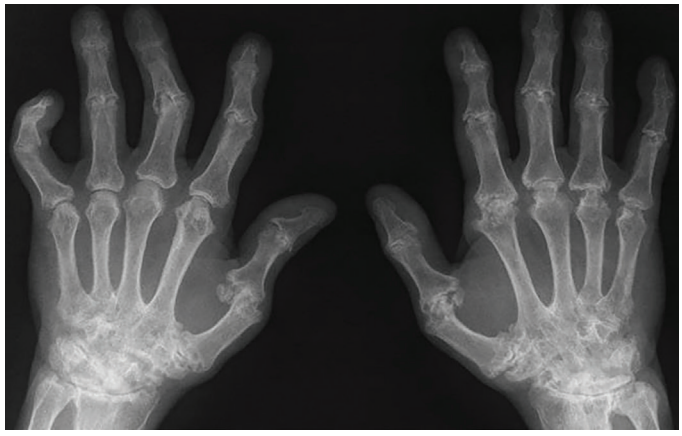

Figure 4. Radiographic features of psoriatic arthritis showing periostitis, bone proliferation, and distal interphalangeal joint involvement (from the authors' collection)

[34,35] Although US findings are considered more characteristic for gout arthritis, their specificity is limited; for instance, the double contour sign can also be observed in calcium pyrophosphate deposition disease and asymptomatic hyperuricemia.

DECT evaluates tissue absorption and chemical composition using two different energy levels (80 and 140 kVp). It can be employed for diagnosing gout, monitoring therapeutic response, and assessing disease progression. However, its routine use is limited by cost and accessibility.[36] Nevertheless, DECT may be considered in PsA patients with persistent joint flares despite treatment and inconclusive SF/US findings. Additionally, DECT can be useful in PsA patients with axial involvement who respond poorly to standard non-steroidal anti-inflammatory drugs (NSAIDs) or biologic therapies and who have risk factors for gout. Still, crystal deposition detected by DECT or US in asymptomatic individuals is not a reliable predictor of future flares.[11]

Clinical Implications of Hyperuricemia in PsA

Gout and PsA are two distinct types of inflammatory arthritis that can share overlapping clinical features. Both conditions commonly affect similar peripheral joints—including the metatarsophalangeal joints, feet, ankles, and knees—and may present in an asymmetric monoarticular or oligoarticular pattern. Findings such as synovitis and dactylitis—characterized by localized erythema, swelling, and tenderness—can be observed in both diseases.[11,12] However, axial involvement and sacroiliitis are rarely seen in gout, whereas they are more frequently encountered in PsA. Genetic markers also play a key role in distinguishing the two; for instance, the HLA-B27 allele is found in only 8-10% of gout patients but in up to 50% of those with PsA.[12,37]

A prospective cohort study from Canada found that hyperuricemia was common among patients with psoriasis and PsA and was associated with an increased risk of developing gout. Hyperuricemic individuals had longer PsA disease duration and more severe skin involvement, as reflected by higher PASI scores. However, hyperuricemia did not correlate with increased joint activity, as the number of tender and swollen joints was similar between hyperuricemic and normouricemic patients.[24]

Conversely, a 10-year retrospective case-control study from France revealed that hyperuricemia was associated with a more destructive pattern of joint involvement in PsA. Multivariable analyses demonstrated a significant relationship between hyperuricemia and inadequate therapeutic response. In this study, hyperuricemic PsA patients exhibited less frequent axial involvement but had a more widespread (polyarticular) and destructive disease course. Joint erosions were also more commonly observed in hyperuricemic individuals compared to normouricemic patients (43.7% vs. 28%). This increased frequency of erosions was particularly noted in cases where SUA levels exceeded 300 µmol/L. Demographically, hyperuricemic PsA patients were found to be older, more frequently male, and to have a later age of PsA onset than normouricemic counterparts.[23]

Hyperuricemia in PsA has also been associated with renal and cardiovascular comorbidities.[38] The literature reports increased prevalence of hypertension, angina, diabetes mellitus, elevated liver enzymes, elevated inflammatory markers, increased serum creatinine, and nephrolithiasis in hyperuricemic individuals.[11,39] Particularly, the presence of persistent hyperuricemia—defined as elevated SUA levels over two consecutive clinical visits—has been significantly associated with higher prevalence of myocardial infarction and congestive heart failure. Multivariate analyses have shown that in PsA patients, persistent hyperuricemia correlates significantly with disease duration (OR: 4.430), BMI (OR: 1.176), and a history of kidney stones (OR: 4.430). In contrast, no significant correlation was found between PASI score and hyperuricemia.[40] However, some studies have reported conflicting findings. For example, a study from Korea demonstrated a significant association between SUA levels and both PASI score and BMI. These discrepancies may stem from differences in population characteristics and methodological approaches.[41]

Therapeutic Approaches at the Intersection of Psoriatic Disease and Hyperuricemia

Studies investigating the impact of PsA or psoriasis treatment on SUA levels remain limited. Nevertheless, current literature suggests that PsA patients are at a significantly increased risk for developing hyperuricemia and gout. Furthermore, hyperuricemic PsA patients have been reported to exhibit lower treatment responses and more pronounced peripheral and destructive joint damage.[23] A comprehensive post-hoc analysis using data from 2504 patients enrolled in phase 3 trials (FUTURE 2-5 and MAXIMISE) compared hyperuricemic patients (SUA ≥360 µmol/L) to normouricemic patients (SUA <360 µmol/L). Evaluations based on joint, skin, and nail involvement, radiographic progression, and quality of life indicated that hyperuricemia may adversely influence the clinical course of PsA.[42] These findings suggest that hyperuricemia could represent an important biomarker to consider in PsA management.

There is currently no direct clinical evidence regarding the effects of gout or hyperuricemia treatment on disease activity in PsA or psoriasis. However, a limited number of studies have shown that urate-lowering therapies such as allopurinol and febuxostat significantly reduce proinflammatory cytokines, including TNF-α, IL-6, and IL-17.[43-45] These reductions have been correlated with decreases in SUA levels. Such findings imply that uric acid may play a role in the pathogenesis of psoriasis and that treatment of hyperuricemia could potentially contribute to clinical improvement in PsA by attenuating inflammatory activity.

To date, only one diagnostic and treatment algorithm has been proposed for Psout—the coexistence of PsA and gout. Widawski et al.[23] outlined a series of criteria aimed at guiding a structured diagnostic and therapeutic approach to this clinical phenotype: (1) thorough documentation of any history of gout; (2) assessment and control of SUA levels ≥360 µmol/L; (3) monitoring the response to urate-lowering therapy; (4) evaluating the response to concomitant PsA treatment; (5) confirmation of MSU crystal presence via SF analysis or DECT; and (6) assessment of family history of gout. An illustrative figure regarding the management of Psout has been presented (Figure 5). These recommendations underscore the need for a comprehensive evaluation of both inflammatory and metabolic components in diagnosing Psout and advocate for a personalized treatment approach.

Discussion

While the association between gout and psoriasis/PsA has long been acknowledged, growing recognition of their common comorbidities and similar clinical presentations has contributed to the increasing identification of this overlap in routine clinical

Figure 5. Proposed management approach for Psout
DECT: Dual-energy computed tomography, MSU: Monosodium urate, PsA: Psoriatic arthritis, SF: Synovial fluid, SUA: Serum uric acid, ULT: Urate-lowering therapy

settings. The simultaneous presence of gout and PsA in the same patient is not uncommon and was first termed "Psout" by Felten et al.[12] in 2020. This review summarizes the current literature on the Psout concept; however, it should be noted that Psout is not yet a clinically validated entity.

The association between gout and PsA was first described in 1982, and elevated SUA levels have been reported in patients with psoriasis ± PsA even in the absence of clinical manifestations of gout. Epidemiological data suggest that shared pathophysiological mechanisms may underlie this relationship, supporting the concept of a distinct "Psout" syndrome.[1,11] However, most existing studies are retrospective or cross-sectional in nature, limiting the ability to establish temporal associations or causal outcomes. This overlap may be at least partially explained by the high prevalence of metabolic syndrome in both disease populations.

Given the pathogenic role of MSU crystals in triggering inflammation, urate-lowering therapies such as allopurinol and febuxostat may represent potential treatment options for controlling psoriatic disease activity, especially in refractory cases.[12,23] This therapeutic approach aims to reduce SUA levels, thereby limiting crystal formation and suppressing associated inflammatory responses. The Psout concept—defined by the coexistence of PsA and gout—highlights the importance of personalized treatment strategies that address both metabolic and inflammatory components. In this context, therapeutic interventions targeting hyperuricemia in PsA may reduce disease activity and improve clinical outcomes.

While biologic agents such as TNF-α and IL-17 inhibitors remain central in PsA management, some conventional disease-modifying antirheumatic drugs like leflunomide have demonstrated mild hypouricemic effects, potentially offering additional benefits in hyperuricemic patients.[11,46] Considering the strong association between hyperuricemia/gout and cardiovascular-metabolic comorbidities, treatment planning should incorporate lifestyle modifications and cardiovascular risk management. Furthermore, the widespread use of NSAIDs should be carefully evaluated in this patient population due to their potential to exacerbate cardiovascular risk. Despite these insights, randomized controlled trials assessing the direct efficacy of urate-lowering therapies in PsA patients are still lacking. Therefore, advanced clinical studies are needed to define optimal treatment strategies for this subgroup.

Conclusion

In conclusion, growing evidence supports the existence of a clinical and pathophysiological intersection between PsA and gout, conceptualized as "Psout". Although it is not yet formally recognized as a distinct disease entity, Psout highlights the need for heightened clinical awareness and comprehensive evaluation strategies that integrate both inflammatory and metabolic components. Future prospective studies and randomized controlled trials are warranted to elucidate its pathogenesis, refine diagnostic criteria, and develop targeted treatment protocols. Recognizing and addressing this overlap may ultimately improve clinical outcomes and quality of life in a specific subset of patients within the psoriatic disease spectrum.

Footnotes

Author Contributions

Concept: S.U., Ö.S.G., T.P., Design: S.U., Ö.S.G., T.P., Data Collection or Processing: S.U., Ö.S.G., T.P., Analysis or Interpretation: S.U., Ö.S.G., T.P., Literature Search: S.U., Ö.S.G., T.P., Writing: S.U., Ö.S.G., T.P.

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