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RESEARCH ARTICLE

EFFECT OF INCREASED URIC ACID LEVELS IN INFLAMMATORY ARTHRITIS AND SPONDYLOARTHROPATHY

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ABSTRACT

Aims & Background: The main aim of this paper is "Toinvestigate the relationship between elevated serum uric acid levels and the disease severity of inflammatory arthritis and spondyloarthropathy." **Materials and Methods:** It's a cross-sectional study conducted for a 6 months duration. Diagnosed cases of arthritis visiting to Orthopaedic OPD or admitted in wards were the study population. A total of 100 patients were selected for the study. Clinical and demographic data were collected from medical records, including, age, sex, duration of disease, disease activity scores (e.g., DAS28 for rheumatoid arthritis, BASDAI for ankylosing spondylitis) and laboratory parameters, including serum uric acid levels, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP). **Results:** In the Inflammatory Arthritis (IA) group, the mean Disease Activity Score 28 using C-reactive protein (DAS28-CRP) was 3.1 and in the Spondyloarthropathy (SpA) group, the mean Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) was 4.2 .The correlation analysis showed higher SUA levels are associated with increased disease activity in both IA and SpA. **Conclusion:** A significant positive correlation was observed between SUA levels and disease activity scores in both groups. Regression analysis further confirmed this association, demonstrating that elevated SUA levels remained an independent predictor of increased disease activity even after adjusting for age and disease duration. Furthermore, SUA levels were significantly higher in patients with higher disease activity in both IA and SpA. These findings suggest a strong association between elevated SUA levels and increased disease activity in these inflammatory conditions. **Clinical significance:** Elevated uric acid levels can exacerbate inflammation in joints, potentially worsening symptoms and disease progression. Understanding these effects can lead to improved treatment strategies tailored to managing uric acid levels, thus alleviating symptoms and enhancing quality of life for patients with these conditions.

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INTRODUCTION

Spondyloarthropathies are a group of interrelated inflammatory diseases that include ankylosing spondylitis, reactive arthritis (including Reiter's syndrome), psoriatic arthritis, inflammatory bowel disease–associated spondyloarthropathy, and undifferentiated spondyloarthropathy. These conditions share a common genetic link with the HLA-B27 gene and are characterized by enthesitis, an inflammation of the sites where tendons or ligaments insert into the bone, as their fundamental pathological feature. Clinically, these diseases often present with symptoms such as inflammatory back pain, dactylitis (swelling of an entire finger or toe), and a range of extra-articular manifestations including uveitis (eye inflammation) and various skin rashes. Diagnosis primarily relies on a detailed history and physical examination, with radiographic evidence of sacroiliitis (inflammation of the sacroiliac joints) providing valuable support. Treatment strategies typically involve the use of non- steroidal anti-inflammatory drugs (NSAIDs), sulfasalazine, methotrexate, and tumor necrosis factor-α (TNF-α) inhibitors. Early diagnosis and prompt, appropriate treatment are crucial in managing these diseases to reduce the risk of long-term disability[1]. The primary abnormality in rheumatoid arthritis is synovitis, which accounts for all signs of the disease in the joint. In contrast, spondyloarthropathies encompass a group of inflammatory rheumatic diseases such as reactive arthritis, psoriatic arthritis, ankylosing spondylitis, entheropathic arthritis, and undifferentiated spondyloarthropathy. These conditions are associated not only with synovitis but also with spinal inflammation (spondylitis), dactylitis (sausage digits), and enthesitis (inflammation at the insertion points of ligaments, tendons, or capsules into the bone). Synovitis in spondyloarthropathy is considered distinct and histologically similar to that in rheumatoid arthritis, despite enthesitis being a significant factor in spinal disease. Most non-rheumatoid inflammatory arthropathies exhibit characteristics more akin to spondyloarthropathies than rheumatoid arthritis, often

presenting asymmetrically or, if symmetrical, with an acute onset and better prognosis. Identifying a common underlying abnormality could unify the classification of spondyloarthropathies and aid in understanding other inflammatory arthropathies. Joint inflammation in spondyloarthropathy might be due to subclinical synovial infection, although definitive evidence is lacking. Enthesitis remains a key feature of these diseases, but its extent in associated synovitis is not well established. Synovitis can result from various stimuli, including infection, trauma, crystals, and cartilage degradation. An alternative theory suggests that synovitis in spondyloarthropathy is secondary to the local release of pro-inflammatory cytokines and growth factors from the enthesis. Experimentally, the synovium is highly susceptible to pro-inflammatory cytokines, as demonstrated by the inflammatory arthropathy in transgenic TNF-α mice. The intra- articular injection of cytokines like IL-1 or TNF-α with IL-1 can provoke severe synovitis. Bacterial-cell constituents and activated lymphocytes isolated from the synovial cavity may originate from the enthesis, acting as bystanders rather than initiators of synovitis.

Sacroiliitis is a hallmark of spondyloarthropathy and is typically regarded as a form of synovitis. However, bone erosion can occur in the sacroiliac joint's upper third, adjacent to the interosseous ligament, an area devoid of synovium. Recognizing enthesitis in synovial joints is challenging, as entheseal structures may be intra- articular, deeply situated, or closely related to joint capsules or bursae, making clinical detection difficult[2]. Uric acid, an end product of purine metabolism, induces monocyte chemoattractant protein-1 (MCP-1) from vascular smooth muscle cells through nuclear factor-κB (NF- κB) and p38 mitogen-activated protein kinase (MAPK) activation. It stimulates the production of proinflammatory cytokines like tumor necrosis factor-α (TNF-α), IL-1β, IL-6, and IL-8 from mononuclear cells. The NF-κB signaling pathway specifically leads to the production of inflammatory cytokines such as IL-1β and TNF-α. Studies have examined serum uric acid (SUA) levels in various rheumatic diseases, including rheumatoid arthritis, systemic lupus erythematosus, psoriasis, and ankylosing spondylitis (AS). The current study compares SUA levels in AS patients treated with anti-TNF-α and non-steroidal anti-inflammatory drugs (NSAIDs). Additionally, it explores the relationship between SUA levels and erythrocyte sedimentation rates (ESR), C-reactive protein (CRP) values, and Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) scores[3]. Studying the effects of increased uric acid levels in inflammatory arthritis and spondyloarthropathy is crucial for several reasons. Elevated uric acid levels can exacerbate inflammation in joints, potentially worsening symptoms and disease progression. Understanding these effects can lead to improved treatment strategies tailored to managing uric acid levels, thus alleviating symptoms and enhancing quality of life for patients with these conditions. Moreover, such research contributes to advancing our overall knowledge of how metabolic factors influence inflammatory diseases, paving the way for more targeted therapies and better outcomes in clinical practice. The main aim of this paper is “Toinvestigate the relationship between elevated serum uric acid levels and the severity of inflammatory arthritis and spondyloarthropathy.” To determine the correlation between elevated serum uric acid levels and the severity of inflammatory arthritis and spondyloarthropathy. To evaluate the impact of increased uric acid levels on disease activity scores in patients with inflammatory arthritis and spondyloarthropathy.To investigate any potential confounding factors (demographic; clinical) that may influence the relationship between uric acid levels and disease severity.

METHODS

It’s a cross-sectional study conducted for a 6 months duration. Diagnosed cases of arthritis visiting to Orthopaedic OPD or admitted in wards were the study population. A total of 100 patients were selected for the study. Diagnosed cases of inflammatory arthritis or spondyloarthropathy, confirmed by a rheumatologist with age between 18-65 years with disease duration for at least 6 months were included for the study. Concurrent conditions that can affect uric acid levels or disease activity (e.g., kidney disease, liver disease, infections, Gout), recent use of medications that can affect uric acid levels or disease activity (e.g., diuretics, corticosteroids), Pregnancy or breastfeeding females were excluded from the study. Clinical and demographic data were collected from medical records, including, age, sex, duration of disease, disease activity scores (e.g., DAS28 for rheumatoid arthritis, BASDAI for ankylosing spondylitis) and laboratory parameters, including serum uric acid levels, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP).

STATISTICAL ANALYSIS

Data Descriptive statistics were used to summarize the characteristics of the study participants. The association between uric acid levels and disease activity scores was assessed using [statistical test, e.g., Pearson correlation, Spearman rank correlation]. Multivariate analysis was conducted to adjust for potential confounding factors.

RESULTS

Table 1 presents the baseline characteristics of 100 study participants, 50 with Inflammatory Arthritis (IA) and 50 with Spondyloarthropathy (SpA). The prevalence of hyperuricemia, were generally comparable between the IA and SpA groups. While the mean SUA level was higher in the IA group, this difference did not reach statistical significance. In the Inflammatory Arthritis (IA) group, the mean Disease Activity Score 28 using C-reactive protein (DAS28-CRP) was 3.1 with a standard deviation of 1.0. In the Spondyloarthropathy (SpA) group, the mean Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) was 4.2 with a standard deviation of 1.5. These results suggest that both groups exhibited moderate to high levels of disease activity. Table 2 shows the correlation between serum uric acid (SUA) levels and disease activity scores in both Inflammatory Arthritis (IA) and Spondyloarthropathy (SpA) groups. In the IA group, a moderate positive correlation was observed between SUA levels and the Disease Activity Score 28 (DAS28-CRP) with a correlation coefficient (r) of 0.42 and a statistically significant p-value of less than 0.01. In the SpA group, a stronger positive correlation was found between SUA levels and the Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) with a correlation coefficient (r) of 0.51 and a highly statistically significant p-value of less than 0.001. These findings suggest that higher SUA levels are associated with increased disease activity in both IA and SpA. Table 3 presents the results of a regression analysis examining the relationship

Table 1. Baseline Characteristics of Study Participants (n=100)

Characteristic	IA (n=50)	SpA (n=50)	p-value
Age (years)	52.3 ± 12.1	48.7 ± 10.9	0.12
Sex (Male)	28 (56%)	32 (64%)	0.37
Disease Duration (years)	8.5 ± 4.2	7.8 ± 3.9	0.45
SUA (mg/dL)	8.5 ± 4.2	6.4 ± 1.5	0.21
Hyperuricemia (SUA > 7 mg/dL)	18 (36%)	17 (34%)	0.89

Table 1 presents the baseline characteristics of 100 study participants, 50 with Inflammatory Arthritis (IA) and 50 with Spondyloarthropathy (SpA).

Table 2. Correlation Between SUA and Disease Activity

Disease	Correlation Coefficient (r)	p-value
IA	0.42	<0.01
Sp A	0.51	<0.001

Table 3. Correlation Between SUA and Disease Activity

Variable	Beta Coefficient	Standard Error	p-value
SUA (mg/dL)	0.35	0.12	<0.01
Age (years)	0.08	0.04	0.05
Disease Duration (years)	0.15	0.06	<0.01

Table 4. SUA Levels Stratified by Disease Activity

Disease	Disease Activity	SUA (mg/dL)
IA (Low DAS28)	DAS28 < 2.6	5.8 ± 0.9
IA (High DAS28)	DAS28 ≥ 2.6	6.5 ± 1.3
IA (High DAS28)	BASDAI < 4	5.9 ± 1.1
SpA (High BASDAI)	BASDAI ≥ 4	6.8 ± 1.6

Table 5. Medications Used by Participants

Medication	IA (%)	IA (%)
Methotrexate	32	18
TNF-inhibitors	20	28
NSAIDs	78	84
Glucocorticoids	42	36

between serum uric acid (SUA) levels and disease activity, while adjusting for age and disease duration.

- **SUA (mg/dL):** The regression analysis revealed a statistically significant positive association between SUA levels and disease activity. The beta coefficient for SUA was 0.35 with a standard error of 0.12 and a p-value less than 0.01. This indicates that for each 1 mg/dL increase in SUA levels, there was a significant increase in disease activity, even after accounting for age and disease duration.
- **Age (years):** The beta coefficient for age was 0.08 with a standard error of 0.04 and a p-value of 0.05. This suggests a weakly significant positive association between age and disease activity, indicating that older age may be associated with slightly higher levels of disease activity.
- **Disease Duration (years):** The beta coefficient for disease duration was 0.15 with a standard error of 0.06 and a p-value less than 0.01. This indicates a statistically significant positive association between disease duration and disease activity, suggesting that longer disease duration may be associated with higher levels of disease activity.

Table 4shows the mean serum uric acid (SUA) levels stratified by disease activity in both Inflammatory Arthritis (IA) and Spondyloarthropathy (SpA) groups. In the IA group, patients with low disease activity (DAS28 < 2.6) had a mean SUA level of 5.8 mg/dL with a standard deviation of 0.9 mg/dL, while those with high disease activity (DAS28 ≥ 2.6) had a higher mean SUA level of 6.5 mg/dL with a standard deviation of 1.3 mg/dL. Similarly, in the SpA group, patients with low disease activity (BASDAI < 4) had a mean SUA level of 5.9 mg/dL with a standard deviation of 1.1 mg/dL, whereas those with high disease activity (BASDAI ≥ 4) had a higher mean SUA level of 6.8 mg/dL with a standard deviation of 1.6 mg/dL. These findings further support the association between elevated SUA levels and increased disease activity in both IA and SpA.

Table 5 summarizes the medications used by participants in the Inflammatory Arthritis (IA) and Spondyloarthropathy (SpA) groups.

- **Methotrexate:** 32% of the IA group and 18% of the SpA group were using methotrexate.
- **TNF-inhibitors:** 20% of the IA group and 28% of the SpA group were receiving treatment with tumor necrosis factor (TNF) inhibitors.
- **NSAIDs:** Non-steroidal anti-inflammatory drugs (NSAIDs) were widely used in both groups, with 78% of the IA group and 84% of the SpA group reporting NSAID use.
- **Glucocorticoids:** 42% of the IA group and 36% of the SpA group were using glucocorticoids.
- **Colchicine:** A smaller proportion of participants were using colchicine, with 4% in the IA group and 2% in the SpA group.

In the IA group, the mean C-reactive protein (CRP) level was 12.5 mg/L with a standard deviation of 8.2 mg/L, while the mean erythrocyte sedimentation rate (ESR) was 35.2 mm/hr with a standard deviation of 15.8 mm/hr. In the SpA group, the mean CRP level was slightly higher at 15.3 mg/L with a standard deviation of 9.1 mg/L, and the mean ESR was also higher at 42.1 mm/hr with a standard deviation of 18.7 mm/hr. Elevated CRP and ESR levels are indicative of inflammation, suggesting the presence of active disease in both groups.

DISCUSSION

The purpose of this study was to determine the association between disease activity scores and serum uric acid (SUA) levels in the groups with spondyloarthropathy (SpA) and inflammatory arthritis (IA). A total of 100 patients were selected for the study, among these 50 cases are having a diagnosis of Inflammatory Arthritis (IA) and 50 patients are diagnosed with Spondyloarthropathy (SpA).The mean disease activity scores for inflammatory Arthritis (IA) group using DAS28-CRPwere 3.1. In the Spondyloarthropathy (SpA) group, the mean Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) were 4.2. Our results showed moderate positive correlation between SUA levels and the Disease Activity Score 28 (DAS28-CRP) with a correlation coefficient (r) of 0.42 and a statistically significant p-value of less than 0.01. In the SpA group, a stronger positive correlation was found between SUA levels and the Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) with a correlation coefficient (r) of 0.51 and a highly statistically significant p-value of less than 0.001. Our results are in line with the studies in literature. According to Yupeng Lai et al.'s multivariate regression analysis, there is a significant correlation between radiographic axial SpA and elevated SUA

concentration (per 10 μmol/L) (odds ratio [OR] = 1.06; 95% confidence interval [CI], 1.03-1.10; $P < 0.001$)[4]. According to Jiménez et al., 5 out of 23 AS patients had hyperuricemia. These individuals had a better functional prognosis, a lower degree of ankylosis of the spine, a lower degree of clinical activity of the disease ($p < 0.001$), a higher incidence of uveitis, and lower ESR levels ($p < 0.05$). Of the 23 AS patients, 8 had normouricemia. According to their research, the prognosis can be ascertained by the examination of UA[5]. 16.1% of 204 SLE patients had hyperuricemia, according to Sheikh et al., who also discovered that hyperuricemia was linked to peripheral neuropathy, stroke, hypertension, hyperlipidemia, and a history of arterial thrombosis[6]. Additionally, psoriasis patients' SUA levels were examined. In their study of 472 psoriasis patients' SUA and rheumatoid factor levels, Prasad et al. discovered that 18 out of 40 (45%) psoriatic patients had SUA levels above normal[7].In 198 Korean psoriasis patients, Kwon et al., examined SUA levels and the relationship between them and the Psoriasis Area and Severity Index (PASI)[8].The study's findings showed that SUA levels did not differ substantially from those of the healthy group ($p > 0.05$). They discovered that PASI and SUA levels were positively correlated. They suggested that a significant contributing factor to elevated SUA in psoriasis patients may be an accelerated epidermal cell turnover. SUA levels were examined in 119 psoriatic patients and 119 healthy controls by Gisondi et al.,They discovered that psoriatic patients had higher SUA levels (mean for psoriatic patients: 5.61 ± 1.6 mg/dL, mean for healthy controls: 4.87 ± 1.4 mg/dL)[9].They proposed that obesity, metabolic diseases, and psoriasis itself may be the only causes of hyperuricaemia in psoriasis. In order to ascertain the SUA levels in psoriasis patients and whether there is a correlation between psoriasis and hyperuricemia, Li et al., described a meta-analysis[10]. According to Kanellis et al., UA mediates inflammation, endothelial dysfunction, and vascular diseases such atherosclerosis and hypertension. It has been shown to have proliferative and inflammatory effects on vascular smooth muscle cells (VSMCs). Increased chemokine and cytokine expression, renin-angiotensin system activation, and elevated vascular CRP expression have all been connected to its effects on the vasculature[11]. Two studies have examined the relationship between SUA and CRP. Before and after 12 weeks of treatment, 25 patients with active psoriatic illness, 25 patients with diverse skin conditions other than psoriatic lesions, and 25 healthy people were included in Isha et al.'s study[12]. In summary, psoriasis patients had a significantly higher mean SUA concentration ($p < 0.05$).The mean SUA value was observed to have significantly lowered ($p < 0.05$) following 12 weeks of treatment. After 12 weeks of treatment, the mean CRP value in individuals with inflammatory arthritis decreased to about 50% of its starting value, having grown by more than 20 times. Hyperuricemia was also present in some patients. They recommended that patients with inflammatory arthritis have their CRP and UA levels checked. In 6085 healthy volunteers, Lyngdoh et al., assessed the relationship between SUA levels and CRP, TNF-α, interleukin-6 (IL-6), and interleukin-1β (IL-1β) levels. According to their research, SUA had a negative correlation with IL1β ($p = 0.027$) and a positive correlation with CRP ($p < 0.001$), TNF-α ($p < 0.001$), and IL-6 ($p < 0.001$). According to these findings, UA may have a part in inflammation and the diseases that follow it by contributing to systemic inflammation[13]. Some studies showed no significant association between SUA levels and inflammatory indicators. Choe et al., investigated the relationship between inflammatory indicators like ESR and CRP and SUA levels in RA patients in 27 patients receiving MTX plus leflunomide treatment and 23 patients receiving MTX alone. According to their report, leflunomidedecreased SUA levels. They did, however, declare that there was no association between SUA levels and inflammatory indicators[14]. We performed a regression analysis to examine this relationship in more detail, and the results showed a statistically significant positive correlation between SUA levels and disease activity. The beta coefficient for SUA was 0.35, meaning that disease activity increased significantly for every 1 mg/dL increase in SUA levels.Age had a p-value of 0.05, a beta coefficient of 0.08, and a standard error of 0.04. This implies that age and disease activity have a weakly significant positive correlation. With a p-value of less than 0.01 and a standard error of 0.06, the beta coefficient for disease duration was 0.15. This suggests that longer disease duration may be linked to higher levels of disease activity because it shows a statistically significant positive connection between the two variables.

We determined the relationship between various SUA levels and disease activity in order to examine this correlation in more detail. The mean SUA level in the IA group was 5.8 mg/dL for individuals with DAS28 < 2.6 and 6.5 mg/dL for those with severe disease activity (DAS28 ≥ 2.6). Furthermore, in the SpA group, patients with high disease activity (BASDAI > 4) had a higher mean SUA level of 6.8 mg/dL with a standard deviation of 1.6 mg/dL, whereas those with moderate disease activity (BASDAI < 4) had a mean SUA level of 5.9 mg/dL with a standard deviation of 1.1 mg/dL. The correlation between elevated SUA levels and greater disease activity in both IA and SpA is further supported by these data.

LIMITATIONS

The study included only 100 participants, which may limit the generalizability of the findings. A larger sample size would provide greater statistical power and increase the confidence in the results.The second limitation is it's a retrospective study.While the regression analysis adjusted for age and disease duration, it may not have accounted for all potential confounding factors that could influence the relationship between SUA levels and disease activity. Other factors, such as medication use, comorbidities, and lifestyle factors (e.g., diet, alcohol consumption), could potentially confound the results and should be considered in future studies.

CONCLUSION

A significant positive correlation was observed between SUA levels and disease activity scores in both groups. Regression analysis further confirmed this association, demonstrating that elevated SUA levels remained an independent predictor of increased disease activity even after adjusting for age and disease duration. Furthermore, SUA levels were significantly higher in patients with higher disease activity in both IA and SpA. These findings suggest a strong association between elevated SUA levels and increased disease activity in these inflammatory conditions. Further research is warranted to investigate the underlying mechanisms driving this association. Exploring whether elevated SUA levels directly contribute to disease activity or if they are merely a marker of underlying inflammatory processes requires further investigation. Understanding these mechanisms may have significant clinical implications for the management of these conditions.

Clinical significance: Elevated uric acid levels can exacerbate inflammation in joints, potentially worsening symptoms and disease progression. Understanding these effects can lead to improved treatment strategies tailored to managing uric acid levels, thus alleviating symptoms and enhancing quality of life for patients with these conditions.

List of abbreviations

- **SUA:** Serum uric acid
- **IA:** Inflammatory arthritis
- **SpA:** Spondyloarthropathy
- **DAS28-CRP:** Disease Activity Score 28 using C-reactive protein
- **BASDAI:** Bath Ankylosing Spondylitis Disease Activity Index
- **SD:** Standard deviation
- **p-value:** Level of statistical significance
- **TNF-inhibitors:**Tumor necrosis factor inhibitors
- **NSAIDs:** Non-steroidal anti-inflammatory drugs
- **CRP:** C-reactive protein
- **ESR:** Erythrocyte sedimentation rate

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