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Original Article

Orthopaedics Section

Association of Serum Uric Acid Levels with Severity of Knee Osteoarthritis: A Cross-sectional Study

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ABSTRACT

Introduction: Osteoarthritis (OA) of the knee is a common degenerative joint disorder and a leading cause of chronic pain, stiffness and disability among middle-aged and elderly individuals. Serum Uric Acid (SUA) has been suggested as a potential metabolic factor associated with OA severity.

Aim: To evaluate the association between SUA levels and the severity of knee OA and to assess their relationship with pain intensity and functional status.

Materials and Methods: This cross-sectional observational study was conducted in the Department of Orthopaedics, Maharishi Markandeshwar (MM) Medical College and Hospital, Kumarhatti, Solan, Himachal Pradesh, India, over a period of 18 months (January 2023 to June 2024). A total of 100 patients aged 40-70 years with clinically and radiologically confirmed OA of the knee were included. SUA levels were categorised as normal (<6 mg/dL), moderately elevated (6-8 mg/dL) and high (>8 mg/dL). Pain severity was assessed using the Visual Analogue Scale (VAS), functional status using the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) and radiological severity using the Kellgren-Lawrence (KL) grading system. Statistical analysis was performed using Stata version 17.0. Associations between variables were assessed using the Pearson's Chi-square test, Analysis of Variance (ANOVA), while correlation analysis was performed to evaluate the relationship between SUA levels and WOMAC scores. A p-value <0.05 was considered statistically significant.

Results: The mean age of the participants was 57.65±5.65 years, with an age range of 48-68 years. Among the 100 participants, 37% had normal SUA levels, 35% had moderately elevated levels and 28% had high SUA levels. A significant association was observed between SUA levels and radiological severity of OA based on KL grading (χ^2=26.21, p=0.006). Higher SUA levels were also significantly associated with greater pain severity on the VAS scale (χ^2=16.15, p=0.013). Mean WOMAC scores increased progressively with rising SUA levels and a strong positive correlation was observed between SUA levels and total WOMAC score (r=0.67, p<0.001).

Conclusion: Elevated SUA levels are significantly associated with greater radiological severity, increased pain intensity and poorer functional status in patients with knee OA.

Keywords: Degenerative disease, Hyperuricaemia, Joint disorders, Kellgren-Lawrence grading, Osteoarthritis index, Pain severity

INTRODUCTION

The OA of the knee is one of the most common chronic degenerative joint disorders and a leading cause of pain, stiffness and disability among middle-aged and elderly individuals worldwide. It is characterised by progressive degeneration of articular cartilage, subchondral bone changes, osteophyte formation and varying degrees of synovial inflammation, which ultimately result in functional limitation and impaired quality of life [1].

The knee joint is particularly susceptible to osteoarthritic changes because it is the largest synovial joint in the body and bears substantial mechanical load during daily activities such as walking, standing and climbing stairs [2]. The pathogenesis of knee OA is multifactorial and involves mechanical stress, metabolic disturbances, genetic susceptibility, inflammatory mediators and ageing-related cartilage degeneration. The disease process is characterised by an imbalance between cartilage degradation and repair mechanisms, leading to gradual deterioration of joint structure and function [3].

The global burden of knee OA has increased considerably in recent decades owing to increasing life expectancy, rising prevalence of obesity and sedentary lifestyles. Knee OA imposes a significant socioeconomic burden because of chronic pain, disability and the growing demand for medical management and joint replacement procedures [4]. Early identification of factors associated with disease progression is therefore important for timely intervention and prevention of severe joint damage.

Diagnosis of knee OA is typically based on clinical symptoms supported by radiological findings such as joint space narrowing, osteophyte formation and subchondral sclerosis. However, radiographic changes usually appear in later stages of the disease, by which time considerable cartilage damage may already have occurred [5]. Consequently, there has been increasing interest in identifying biochemical markers that may reflect early pathological changes in the joint and help predict disease severity.

The SUA, the final product of purine metabolism, has been investigated as a potential metabolic factor associated with OA. Elevated SUA levels may contribute to inflammatory responses, oxidative stress and urate crystal deposition within joint tissues, which could accelerate cartilage degeneration and synovial inflammation [6]. Several studies have suggested that hyperuricaemia may be associated with increased radiographic severity, pain intensity and functional impairment in patients with knee OA [7-9].

Nevertheless, the association between SUA levels and the severity of knee OA remains controversial. Some studies have reported a significant relationship between elevated SUA levels and radiographic progression or clinical severity of OA, whereas others have found no clear correlation after adjusting for confounding variables such as age, sex and Body Mass Index (BMI) [10-12].

Considering these inconsistent findings, further studies are required to clarify the role of SUA in the progression and severity of knee OA. Therefore, the present study was conducted to evaluate the association between SUA levels and the severity of knee OA. The

study also aimed to assess the relationship between SUA levels, pain intensity and functional status in patients with knee OA.

MATERIALS AND METHODS

The present cross-sectional observational study was conducted in the Department of Orthopaedics at MM Medical College and Hospital, Kumarhatti, Solan, Himachal Pradesh, India, over a period of 18 months (January 2023 to June 2024). Ethical approval for the study was obtained from the Institutional Ethics Committee (MMMCH/IEC/23/666). Written informed consent was obtained from all participants prior to enrollment. A total of 100 patients aged between 40 and 70 years with clinically and radiologically confirmed OA of the knee were included in the study. Participants were recruited consecutively from the Outpatient and Inpatient Departments of Orthopaedics using a non probability convenience sampling technique.

Sample size calculation: The sample size was calculated using the standard formula for cross-sectional studies:

$$n=Z^2\times\frac{PQ}{d^2}$$

Where, n=required sample size;

Z=standard normal deviate at 95% confidence level (1.96);

p=estimated prevalence of knee OA, assumed to be 50% based on previous literature [13];

q=1-p;

d=allowable error (10%).

Using these values, the calculated minimum sample size was 96 participants. Considering feasibility and possible incomplete data, a total of 100 participants were included in the study.

Inclusion criteria: Patients aged 40-70 years presenting with chronic knee pain of at least six weeks duration and clinically as well as radiologically confirmed OA of the knee were included in the study. Participants of either gender who provided written informed consent were enrolled.

Exclusion criteria: Patients receiving medications known to alter SUA levels, including diuretics, allopurinol, losartan, oestrogens, glucocorticoids, salicylates, levodopa, theophylline, pyrazinamide, ethambutol and cyclosporine, were excluded. Patients who had received methotrexate within the previous three months were also excluded. In addition, individuals with systemic or musculoskeletal conditions known to influence OA or uric acid levels, such as rheumatoid arthritis, metabolic disorders, major knee trauma, limb deformities, congenital abnormalities, sports injuries, or bone infections around the knee joint, were excluded from the study.

Study Procedure

Data were collected using a structured data collection form. Information recorded included demographic details such as age and gender, anthropometric parameters including BMI, clinical history of knee pain and findings from physical examination. BMI, calculated by dividing weight in kilograms by height in metres squared, is classified into four categories: underweight (less than 18.5), normal weight (18.5-24.9), overweight (25.0-29.9) and obesity (30.0 or higher) [14].

Each participant underwent a detailed clinical examination of the knee joint. Clinical parameters assessed included joint tenderness, swelling, crepitus and range of motion. Pain severity was measured using the VAS, a 10-cm scale ranging from 0 (no pain) to 10 (worst possible pain) [15].

Functional status was evaluated using the WOMAC index, which assesses pain, stiffness and physical function in OA patients. It has 24 items across three domains: pain (5 items, score 0-20), stiffness (2 items, score 0-8) and physical function (17 items, score 0-68). The total score ranges from 0 to 96, with higher scores indicating greater symptom severity and functional disability [16]. Radiological assessment was performed using bilateral weight-bearing anteroposterior and lateral knee radiographs. The severity of OA was graded according to the KL grading system, which classifies disease severity from Grade 0 (no radiographic features of OA) to Grade 4 (severe OA with marked joint space narrowing, osteophytes, sclerosis and deformity) [17].

The BMI was calculated using the formula weight in kilograms divided by height in metres squared (kg/m^2) and categorised according to World Health Organisation criteria [18].

Measurement of Serum Uric Acid (SUA): Venous blood samples were collected from all participants under aseptic conditions. SUA levels were measured using the Uricase-Peroxidase enzymatic method in the hospital biochemistry laboratory. Based on the measured values, participants were classified into three groups: normal (<6 mg/dL), moderately elevated (6-8 mg/dL) and high (>8 mg/dL) [19].

Data management and statistical analysis: All collected data were entered into a structured database and verified using a double-entry method to minimise transcription errors. Statistical analysis was performed using Stata version 17.0. Continuous variables such as age, BMI and SUA levels were summarised using mean and standard deviation. Categorical variables were presented as frequencies and percentages. The association between SUA levels and radiological severity of OA was assessed using the Pearson's Chi-square. The association between SUA levels and pain severity was also analysed using the Pearson's Chi-square. Correlation between SUA levels and WOMAC functional scores was evaluated using Pearson's correlation coefficient and ANOVA. A p-value of less than 0.05 was considered statistically significant and 95% confidence intervals were reported where appropriate.

RESULTS

A total of 100 participants with clinically and radiologically confirmed OA of the knee were included in the present study. The mean SUA level in the study population was 6.6±2.3 mg/dL, with values ranging from 3.2 to 10.4 mg/dL.

The baseline characteristics of the study is shown in [Table/Fig-1]. The majority of participants belonged to the 51-60 years age group (51%), followed by the 61-70 years group (34%) and the 41-50 years group (15%). The mean age of the participants was 57.65±5.65 years, with an age range of 48-68 years. Male participants constituted 75% of the study population, while 25% were female. With regard to BMI, 39% of participants were classified as obese, 31% were overweight and 30% had normal BMI.

Variables	Category	n (%)
Age group (years)	41-50	15 (15.00)
	51-60	51 (51.00)
	61-70	34 (34.00)
Age (years)	Mean±SD	57.65±5.65
	Min-Max	48-68
Gender	Female	25 (25.00)
	Male	75 (75.00)
BMI category (kg/m^2)	Normal (18.5-24.9)	30 (30.00)
	Overweight (25.0-29.9)	31 (31.00)
	Obese (30.0 or Higher)	39 (39.00)

[Table/Fig-1]: Characteristics of participants (N=100).

The association between SUA levels and radiological severity of knee OA based on KL grading is shown in [Table/Fig-2]. Among participants with normal SUA levels (<6 mg/dL), most cases were classified as mild OA (32.43%), no OA (29.73%), or doubtful OA (27.03%). In contrast, participants with high SUA levels (>8 mg/dL)

OA knee severity (KL Grade 0-4)	Normal (<6 mg/dL) n (%)	Moderate (6-8 mg/dL) n (%)	High (>8 mg/dL) n (%)	Total n (%)
No OA (Grade 0)	11 (29.73)	9 (25.71)	1 (3.57)	21 (21.00)
Doubtful (Grade 1)	10 (27.03)	4 (11.43)	5 (17.86)	19 (19.00)
Mild (Grade 2)	12 (32.43)	11 (31.43)	1 (3.57)	24 (24.00)
Moderate (Grade 3)	2 (5.41)	5 (14.29)	11 (39.29)	18 (18.00)
Severe (Grade 4)	2 (5.41)	6 (17.14)	10 (35.71)	18 (18.00)
Total	37 (100)	35 (100)	28 (100)	100 (100)

[Table/Fig-2]: Association of Serum Uric Acid (SUA) and OA knee score.
Pearson's χ^2=26.21; p-value=0.006

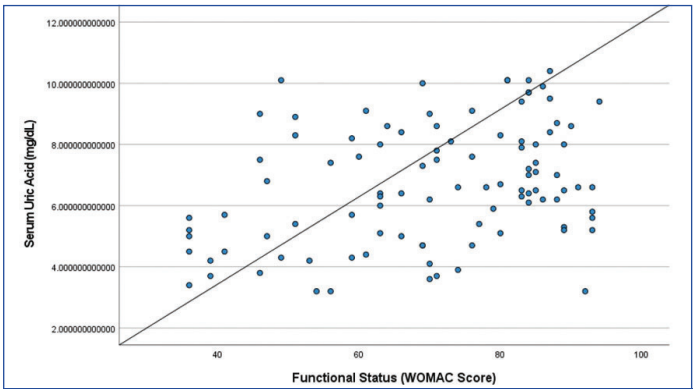
showed higher proportions of moderate OA (39.29%) and severe OA (35.71%). The association between SUA levels and KL grading was statistically significant (χ^2=26.21, p=0.006).

The distribution of pain severity according to SUA levels using the VAS is shown in [Table/Fig-3]. Among participants with normal SUA levels, the most frequent pain score was VAS 2-3 (32.43%), indicating mild pain. In the moderately elevated SUA group (6-8 mg/dL), 48.57% of participants had a VAS score of 6-7, while in the high SUA group (>8 mg/dL), 35.71% of participants also reported a VAS score of 6-7, indicating moderate pain intensity. The association between SUA levels and pain severity was statistically significant (χ^2=16.15, p=0.013).

Pain severity (VAS Score)	Normal (<6 mg/dL) n (%)	Moderate (6-8 mg/dL) n (%)	High (>8 mg/dL) n (%)	Total n (%)
2-3	12 (32.43)	2 (5.71)	2 (7.14)	16 (16.00)
4-5	11 (29.73)	8 (22.86)	7 (25.00)	26 (26.00)
6-7	7 (18.92)	17 (48.57)	10 (35.71)	34 (34.00)
8-10	7 (18.92)	8 (22.86)	9 (32.14)	24 (24.00)
Total	37 (100)	35 (100)	28 (100)	100 (100)

[Table/Fig-3]: Association of Serum Uric Acid (SUA) and pain score.
Pearson's χ^2=16.15, p-value=0.013

A strong correlation was observed between SUA levels and total WOMAC scores (r=0.67, p<0.001), indicating that higher SUA levels were associated with greater functional impairment in patients with knee OA. The Scatterplot showing the distribution of uric acid across various WOMAC total scores is plotted in [Table/Fig-4].



[Table/Fig-4]: Scatterplot of WOMAC functional (total) scores and Serum Uric Acid (SUA) levels.

The mean WOMAC scores across different SUA categories is summarised in [Table/Fig-5]. The mean WOMAC pain score increased from 12.47 in the normal SUA group to 15.19 in the moderately elevated group and 15.06 in the high SUA group. Similarly, the mean WOMAC stiffness score increased from 5.89 to 6.87 and 6.71, respectively. The mean WOMAC function score also increased progressively with rising SUA levels (43.50, 54.23 and 53.13, respectively). The total WOMAC functional status score showed a similar trend, with values of 61.87, 76.29 and 74.90 across the three SUA groups. When the authors applied ANOVA, WOMAC pain and stiffness scores were found to be significantly affected by serum uric acid levels.

WOMAC domain	Normal (<6 mg/dL) Mean±SD	Moderate (6-7.99 mg/dL) Mean±SD	High (≥8 mg/dL) Mean±SD	Total score	One-way ANOVA (p-value)
WOMAC pain score	12.47±3.9	15.19±2.6	15.06±2.9	14.12±3.5	<0.001
WOMAC stiffness score	5.89±1.4	6.87±0.9	6.71±1.1	6.45±1.2	0.002
WOMAC function score	43.50±13.6	54.23±9.6	53.13±10.3	49.81±12.4	<0.001
Total WOMAC Score	61.87±18.8	76.29±12.5	74.90±13.7	70.38±16.8	<0.001

[Table/Fig-5]: Association of Serum Uric Acid (SUA) and mean WOMAC score.

DISCUSSION

The present study evaluated the association between SUA levels and the severity of knee OA in patients attending a tertiary care centre. Among the 100 participants included in the study, 37% had normal SUA levels (<6 mg/dL), 35% had moderately elevated levels (6-8 mg/dL) and 28% had high SUA levels (>8 mg/dL). The mean SUA level in the study population was 6.6±2.3 mg/dL, with values ranging from 3.2 to 10.4 mg/dL. In the present study, a statistically significant association was observed between SUA levels and radiological severity of knee OA based on the KL grading system (χ^2=26.21, p=0.006). Patients with higher SUA levels were more likely to have moderate or severe OA compared with those having normal SUA levels. These findings suggest that elevated uric acid levels may be associated with greater radiographic progression of knee OA.

Similar findings were reported by Krasnokutsky S et al., who observed that serum urate levels were associated with structural progression of knee OA. In their study, individuals with SUA levels ≥6.8 mg/dL showed greater joint space narrowing compared with those having lower SUA levels (0.90 mm vs 0.31 mm; p < 0.01). Furthermore, higher baseline SUA levels were found to predict progression of OA in non gout patients [12].

The present study also demonstrated a significant association between SUA levels and pain severity assessed using the VAS (χ^2=16.15, p=0.013). Participants with higher SUA levels reported greater pain intensity compared with those having normal uric acid levels. These findings indicate that hyperuricaemia may contribute to increased pain perception in patients with knee OA. These observations are consistent with the findings of Bassiouni S et al., who reported that patients with elevated SUA levels had significantly higher WOMAC pain, stiffness and functional scores compared with those having lower SUA levels. In their study, WOMAC pain, stiffness and function scores were significantly higher in patients with elevated SUA levels (p=0.004, p=0.019 and p=0.018, respectively), indicating a strong association between hyperuricaemia and clinical severity of OA [10].

In the present study, functional impairment assessed using the WOMAC index also increased with rising SUA levels. A strong positive correlation was observed between SUA levels and total WOMAC scores (r=0.67, p<0.001). Patients with higher SUA levels demonstrated greater pain, stiffness and functional limitation. These findings suggest that elevated uric acid levels may contribute to worsening functional status in patients with knee OA. The possible mechanism linking hyperuricaemia and OA may involve inflammatory and metabolic pathways. Elevated uric acid levels can promote activation of inflammatory mediators, oxidative stress and deposition of monosodium urate crystals within joint tissues, which may accelerate cartilage degeneration and synovial inflammation. Such mechanisms may contribute to the progression of OA and worsening of clinical symptoms.

However, not all studies have reported a similar association. Go DJ et al., conducted a longitudinal study among community residents

without gout and found no significant correlation between SUA levels and radiographic progression of knee OA after adjusting for age, sex and BMI [11]. Similarly, Lee YH and Song GG performed a Mendelian randomisation analysis and reported insufficient evidence to support a causal relationship between uric acid levels and the risk of OA [20]. In addition, Kim SK et al., reported that although women with OA initially appeared to have higher SUA levels than those without the disease, multivariate analysis did not demonstrate a statistically significant association between SUA levels and OA after adjustment for confounding factors [21]. Similar findings were also reported in earlier epidemiological investigations such as the Framingham Study and the Chingford Study, which did not observe a significant association between SUA levels and radiographic OA [22,23]. Despite these conflicting findings, the results of the present study support the hypothesis that elevated SUA levels may be associated with greater radiographic severity, increased pain and functional impairment in patients with knee OA.

Limitation(s)

The present study had several limitations. First, the cross-sectional design limits the ability to establish a causal relationship between SUA levels and OA severity. Second, the study was conducted at a single centre with a relatively small sample size, which may limit the generalisability of the findings. Third, longitudinal follow-up was not performed to evaluate disease progression over time. Further large-scale multicentric and prospective cohort studies are required to clarify the role of SUA in the pathogenesis and progression of knee OA and to determine whether urate-lowering therapies could influence disease outcomes in affected patients.

CONCLUSION(S)

The present study demonstrated a significant association between elevated SUA levels and the severity of knee OA. Higher SUA levels were associated with greater radiological severity, increased pain intensity and poorer functional status.

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