Original Article

Prevalence and Intensity of Musculoskeletal (Joint) Pain in Patients with Hyperuricemia Among Population of Lahore

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ABSTRACT

Background: Hyperuricemia is a prevalent metabolic abnormality and may be associated with clinically relevant musculoskeletal symptoms even before classical gout becomes apparent; however, local data describing joint-site patterns and pain severity among hyperuricemic adults in Lahore remain limited. **Objective:** To determine the prevalence and intensity of musculoskeletal (joint) pain and assess its relationship with serum uric acid (SUA) levels among hyperuricemic adults presenting to tertiary-care hospitals in Lahore. **Methods:** A descriptive cross-sectional study was conducted over six months at Gulab Devi Hospital and Pakistan Air Force (PAF) Hospital, Lahore, enrolling 102 adults aged 20–60 years with elevated SUA levels. After informed consent, SUA values were recorded from laboratory reports and musculoskeletal pain was characterized by site (including MTP, ankle, knee, shoulder, wrist, MCP, cervical spine, and lumbar spine) and intensity using a Visual Analogue Scale (VAS). Associations between SUA and pain intensity and between SUA and joint-site involvement were evaluated using Pearson correlation (two-tailed). **Results:** Of 102 participants, 57 (55.9%) were male and 45 (44.1%) were female; 83 (81.4%) were from middle socioeconomic status. Knee (27.5%) and ankle (25.5%) were the most prevalent pain sites. SUA correlated positively with pain intensity ($r = 0.564$; $p < 0.001$). The metatarsophalangeal joint showed the strongest association with SUA among joint sites ($r = 0.302$; $p = 0.002$). **Conclusion:** Higher SUA levels were associated with greater musculoskeletal pain intensity, and MTP involvement demonstrated the most specific biochemical relationship, supporting focused joint assessment in hyperuricemic patients.

Keywords: Hyperuricemia; Serum uric acid; Musculoskeletal pain; Joint involvement; Visual analogue scale; Metatarsophalangeal joint; Cross-sectional study.

INTRODUCTION

Hyperuricemia, traditionally described as the “disease of kings,” is a metabolic condition characterized by elevated serum uric acid (SUA) levels and represents a well-recognized precursor to gout and urate crystal deposition disease (1). Beyond its classical association with acute inflammatory arthritis, accumulating evidence suggests that hyperuricemia may exert systemic effects, contributing to cardiovascular disease, renal dysfunction, metabolic syndrome, and subclinical inflammatory states (2,7,26). Despite its high prevalence and expanding pathophysiological implications, hyperuricemia often remains clinically underrecognized in the absence of overt gouty attacks, particularly in low- and middle-income settings where biochemical abnormalities may not prompt musculoskeletal evaluation (2,6).

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Uric acid is the end product of purine metabolism and, under physiological conditions, is maintained within a tightly regulated range through renal and gastrointestinal excretion (9). However, sustained elevation of SUA beyond saturation thresholds facilitates monosodium urate crystal formation, particularly in peripheral joints exposed to lower temperatures and mechanical stress (11,14). The metatarsophalangeal (MTP) joint has been historically recognized as a predilection site for urate crystal deposition, although contemporary imaging studies indicate that asymptomatic crystal deposition may occur in multiple anatomical regions (18). Increasing attention has therefore shifted toward understanding whether hyperuricemia—even in the absence of clinically diagnosed gout—may be associated with measurable musculoskeletal pain or joint discomfort (19,29).

Epidemiological studies have demonstrated variable patterns of joint involvement among hyperuricemic individuals. For instance, cross-sectional analyses have identified significant associations between elevated SUA and musculoskeletal pain across several joints, although the strength and anatomical distribution of these associations differ by population and study design (19,24). Furthermore, sedentary behavior, obesity, and dietary patterns have been implicated in modulating both SUA levels and musculoskeletal symptomatology, suggesting potential interaction between metabolic and biomechanical factors (15,25). In Pakistan, hyperuricemia has been reported with considerable frequency, and diabetes and other metabolic comorbidities are commonly observed in affected individuals (6). However, local data specifically examining the intensity and anatomical distribution of joint pain in hyperuricemic adults remain limited.

From a PICO perspective, the population of interest comprises adults with laboratory-confirmed hyperuricemia; the exposure variable is elevated SUA level; the comparator implicitly includes lower SUA levels within the hyperuricemic spectrum; and the primary outcome is musculoskeletal pain intensity and site-specific involvement. While the pathophysiological link between urate crystal deposition and acute gout is well established, less is known about the graded relationship between SUA concentration and pain intensity in patients without classical gouty arthritis. Moreover, whether certain joints demonstrate stronger biochemical associations than others has not been adequately explored in regional hospital-based cohorts. This represents a clinically relevant knowledge gap, as early identification of urate-related musculoskeletal symptoms could inform preventive strategies and lifestyle modification approaches before irreversible joint damage occurs (5,20,21).

The present study was therefore designed to quantify the prevalence and intensity of musculoskeletal joint pain among hyperuricemic adults presenting to tertiary care hospitals in Lahore and to evaluate the association between SUA levels and both pain intensity and joint-site involvement. We hypothesized that higher SUA levels would be significantly associated with greater pain intensity and that distal lower-limb joints, particularly the metatarsophalangeal joint, would demonstrate stronger biochemical correlations compared with other anatomical sites.

MATERIALS AND METHODS

This descriptive cross-sectional observational study was conducted over a six-month period at Gulab Devi Hospital and Pakistan Air Force (PAF) Hospital, Lahore, Pakistan. The cross-sectional design was selected to estimate the prevalence and intensity of musculoskeletal pain among hyperuricemic adults and to examine the contemporaneous association between serum uric acid (SUA) levels and pain characteristics within a defined clinical population, consistent with STROBE recommendations for observational research (31). The

study period encompassed consecutive patient presentations during routine inpatient and outpatient clinical services..

Eligible participants were adults aged 20–60 years with laboratory-confirmed hyperuricemia. Hyperuricemia was operationally defined as SUA levels exceeding the upper limit of normal according to hospital laboratory reference standards (>7.0 mg/dL in males and >6.0 mg/dL in females), based on established clinical thresholds (9,32). Patients were excluded if they were pregnant, had recent trauma or postoperative status, congenital musculoskeletal disorders, severe physical disability precluding pain assessment, documented psychiatric illness impairing consent, or were currently receiving urate-lowering therapy. A non-probability convenience sampling approach was used, and consecutive eligible patients presenting during the study period were invited to participate to minimize selection bias within the accessible sampling frame. Recruitment was performed by trained investigators who explained the study objectives, procedures, risks, and benefits in the local language. Written informed consent was obtained prior to enrollment. Data were collected using a structured case-record form designed to standardize variable definitions and ensure uniform data capture. Demographic variables included age, sex, and socioeconomic status categorized into low, middle, and high strata based on self-reported income and occupation. Laboratory SUA values were extracted directly from authenticated hospital laboratory reports generated using standardized enzymatic colorimetric assays. To reduce measurement error, only values obtained within the same clinical encounter as pain assessment were recorded. The primary outcome variable was musculoskeletal pain intensity, measured using a 10-cm Visual Analogue Scale (VAS), where 0 represented “no pain” and 10 represented “worst imaginable pain.” Participants rated their current pain intensity at the time of assessment. Secondary outcome variables included the presence or absence of pain at specific anatomical sites: metatarsophalangeal (MTP), ankle, knee, shoulder, wrist, metacarpophalangeal (MCP), cervical spine, and lumbar spine. Joint involvement was recorded as binary (present/absent), and multiple sites per participant were permitted. The primary exposure variable was SUA level expressed as a continuous variable (mg/dL). To address potential sources of bias, standardized instructions were provided prior to VAS administration to reduce information bias, and all investigators underwent protocol training to ensure consistent assessment procedures. Data entry was performed using double-entry verification to prevent transcription errors. Although the cross-sectional design does not permit causal inference, potential confounding by age and sex was evaluated analytically, given their known associations with both SUA levels and musculoskeletal pain (2, 26).

Sample size adequacy was assessed using correlation-based power estimation methods. Assuming a moderate correlation ($r = 0.30$) between SUA and pain intensity, with a two-sided α of 0.05 and 80% statistical power, a minimum sample of 84 participants was required; the final sample size of 102 exceeded this threshold, providing sufficient precision for primary association estimates (33). Statistical analyses were conducted using SPSS version 23.0 (IBM Corp., Armonk, NY, USA). Continuous variables were assessed for normality using the Shapiro–Wilk test and graphical inspection. Descriptive statistics were reported as mean ± standard deviation for normally distributed variables and frequency (percentage) for categorical variables. The association between SUA and VAS pain intensity was evaluated using Pearson’s correlation coefficient (two-tailed). Correlations between SUA and binary joint-site variables were interpreted using point-biserial correlation equivalents. Ninety-five percent confidence intervals (95% CI) were calculated for all correlation coefficients. Statistical significance was set at $p < 0.05$. Missing data were minimal (<5%) and handled using complete-case analysis. Sex-stratified analyses were

conducted to explore potential effect modification. The study protocol was reviewed and approved by the institutional ethics committee of Gulab Devi Hospital, and all procedures were conducted in accordance with the Declaration of Helsinki principles for medical research involving human subjects (34). Participant confidentiality was maintained through anonymized coding, secure storage of electronic datasets, and restricted access to research personnel. The study dataset, coding framework, and statistical analysis plan were archived to ensure transparency, reproducibility, and independent verification of results

RESULTS

A total of 102 hyperuricemic adults were included in the final analysis. There were no missing data for the primary exposure (serum uric acid, SUA) or primary outcome (VAS pain score). All quantitative findings are presented in numbered tables with corresponding inferential statistics.

Table 1. Baseline Demographic and Clinical Characteristics (n = 102)

Variable	n (%) or Mean ± SD	95% CI
Sex		
Male	57 (55.9%)	46.1–65.2
Female	45 (44.1%)	34.8–53.9
Age Group		
20–40 years	22 (21.6%)	14.8–30.4
40–60 years	80 (78.4%)	69.6–85.2
Socioeconomic Status		
Low	7 (6.9%)	3.4–13.6
Middle	83 (81.4%)	72.7–87.8
High	12 (11.8%)	6.8–19.7
VAS Pain Score	3.18 ± 0.65	3.05–3.31
SUA (coded value)	1.43 ± 0.64	1.31–1.55
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Low	7 (6.9%)	3.4–13.6
Middle	83 (81.4%)	72.7–87.8

Table 2. Distribution of Pain Intensity Categories (n = 102)

Pain Category	n (%)
Moderate–Severe	66 (64.7%)
Severe	28 (27.5%)
Worst	8 (7.8%)
Pain Category	n (%)
Moderate–Severe	66 (64.7%)
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Pain Category	n (%)
Moderate–Severe	66 (64.7%)
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Worst	8 (7.8%)
Pain Category	n (%)

Table 1 presents the baseline profile of the 102 hyperuricemic participants and shows a modest male predominance, with 57/102 (55.9%) males and 45/102 (44.1%) females. The cohort was concentrated in the older eligibility band: 80/102 (78.4%) were aged 40–60 years,

whereas 22/102 (21.6%) were aged 20–40 years, indicating that nearly four in five participants were in late adulthood. Socioeconomic status was strongly skewed toward the middle category, with 83/102 (81.4%) from middle socioeconomic strata, compared with 12/102 (11.8%) high and 7/102 (6.9%) low socioeconomic status.

Table 3. Prevalence of Joint-Site Pain

Joint Site	n (%)
Knee	28 (27.5%)
Ankle	26 (25.5%)
Shoulder	18 (17.6%)
MTP	16 (15.7%)
Lumbar spine	6 (5.9%)
Wrist	4 (3.9%)
Cervical spine	4 (3.9%)
MCP	1 (1.0%)
Joint Site	n (%)

Table 4. Correlation Between Serum Uric Acid and Pain Intensity

Variables	Pearson r	95% CI	r^2	p-value	Variables
SUA vs VAS Pain Score	0.564	0.415–0.684	0.318	<0.001	SUA vs VAS Pain Score
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Variables	Pearson r	95% CI	r^2	p-value	Variables
SUA vs VAS Pain Score	0.564	0.415–0.684	0.318	<0.001	SUA vs VAS Pain Score

Table 5. Association Between Serum Uric Acid and Joint-Site Pain)

Joint Site	Pearson r	95% CI	p-value
MTP	0.302	0.114–0.469	0.002
Knee	-0.107	-0.295–0.089	0.286
Ankle	-0.028	-0.221–0.167	0.779
Shoulder	-0.072	-0.263–0.124	0.475
Lumbar spine	-0.104	-0.293–0.092	0.297
Wrist	0.022	-0.173–0.216	0.827

Table 6. Sex-Stratified Correlation Between SUA and Pain Intensity

Sex	n	Pearson r	95% CI
Male	57	0.591	0.381–0.742
Female	45	0.524	0.272–0.705
Sex	n	Pearson r	95% CI
Male	57	0.591	0.381–0.742

The mean pain intensity was VAS 3.18 ± 0.65, with a narrow precision band (95% CI 3.05–3.31), suggesting relatively consistent pain scoring across participants. The reported "SUA coded value" averaged 1.43 ± 0.64 (95% CI 1.31–1.55), reflecting that the dataset stored uric acid in a coded form rather than directly presenting mg/dL values in the results table. Table 2 describes pain intensity categories and demonstrates that pain burden was predominantly moderate-to-severe. Specifically, 66/102 (64.7%) fell into the moderate–severe category (95% CI 55.0–73.4), while 28/102 (27.5%) reported severe pain (95% CI 19.7–36.8), and 8/102 (7.8%) experienced worst pain (95% CI 3.9–14.9). Taken together, 36/102 (35.3%) were in the severe-to-worst spectrum, meaning over one-third of hyperuricemic patients had high-intensity pain categories, reinforcing clinical relevance beyond mild discomfort.

Table 3 quantifies the anatomical distribution of joint-site pain and shows a clear dominance of lower-limb involvement. The knee was the most frequently reported site

(28/102; 27.5%, 95% CI 19.7–36.8), closely followed by the ankle (26/102; 25.5%, 95% CI 18.0–34.7). Upper-limb involvement was lower but still notable at the shoulder (18/102; 17.6%, 95% CI 11.5–26.2), whereas distal upper-limb sites were uncommon (wrist 4/102; 3.9%, cervical spine 4/102; 3.9%). The metatarsophalangeal (MTP) joint—clinically important in urate-related arthropathy—was involved in 16/102 (15.7%) (95% CI 9.9–24.0), making it less prevalent than knee/ankle but still affecting roughly one in six participants. Axial symptoms were comparatively rare (lumbar spine 6/102; 5.9%, 95% CI 2.7–12.2), and MCP involvement was exceptionally infrequent (1/102; 1.0%, 95% CI 0.17–5.35). Overall, knee plus ankle involvement alone accounted for 54/102 site reports (53.0%), demonstrating that more than half of observed joint-site pain clustered in the lower limb.

Table 4 reports the primary association between SUA and pain intensity and shows a moderate positive biochemical–symptom relationship. Serum uric acid correlated with VAS pain score at $r = 0.564$ with a tight 95% CI of 0.415–0.684 and $p < 0.001$, indicating strong statistical evidence of association. The corresponding explained variance ($r^2 = 0.318$) implies that approximately 31.8% of variation in pain intensity was associated with SUA levels, a clinically meaningful proportion for a single metabolic biomarker in an observational design.

Table 5 examines site-specific SUA associations and demonstrates that the biochemical relationship was joint-selective rather than uniform across all painful sites. The MTP joint showed the strongest and statistically significant positive correlation with SUA ($r = 0.302$; 95% CI 0.114–0.469; $p = 0.002$), supporting anatomical specificity consistent with urate-linked pathology. In contrast, the most prevalent joints—knee ($r = −0.107$; $p = 0.286$) and ankle ($r = −0.028$; $p = 0.779$)—showed weak, non-significant associations with SUA, suggesting that their high prevalence may reflect additional contributors such as mechanical load or degenerative disease beyond urate level alone. Other joints similarly showed small and statistically non-significant correlations, including shoulder $r = −0.072$ ($p = 0.475$) and lumbar spine $r = −0.104$ ($p = 0.297$).

Table 6 provides sex-stratified correlations and shows that the SUA–pain intensity relationship persisted in both sexes, with slightly higher magnitude among males. In men ($n = 57$), SUA correlated with VAS pain intensity at $r = 0.591$ (95% CI 0.381–0.742; $p < 0.001$), while in women ($n = 45$) the correlation was $r = 0.524$ (95% CI 0.272–0.705; $p < 0.001$). The absolute difference in correlation ($\Delta r = 0.067$) indicates broadly similar effect strength across sexes, supporting that the urate–pain intensity gradient was not confined to one gender in this cohort

DISCUSSION

This cross-sectional observational study demonstrated a statistically significant and clinically meaningful association between serum uric acid (SUA) levels and musculoskeletal pain intensity among hyperuricemic adults. The observed moderate positive correlation ($r = 0.564$; $p < 0.001$) indicates that higher SUA concentrations were associated with greater pain severity, with approximately 31.8% of the variability in pain scores explained by SUA alone. This magnitude of association supports the growing recognition that hyperuricemia may exert symptomatic effects beyond classical acute gouty arthritis, potentially through low-grade inflammatory pathways and crystal-mediated synovial irritation even in the absence of overt flares (37,38). The findings therefore extend previous epidemiological observations linking elevated urate levels with musculoskeletal discomfort and subclinical joint pathology (19, 29).

A key observation was the divergence between anatomical prevalence and biochemical association. Although the knee (27.5%) and ankle (25.5%) were the most commonly reported sites of pain, neither demonstrated a statistically significant correlation with SUA. In contrast, the metatarsophalangeal (MTP) joint, which was involved in 15.7% of participants, exhibited the strongest and statistically significant association with SUA ($r = 0.302$; $p = 0.002$). This pattern aligns with established pathophysiological principles indicating that distal lower-limb joints, particularly the first MTP joint, are preferential sites for monosodium urate crystal deposition due to reduced temperature, biomechanical stress, and local microenvironmental factors (11,39). The results suggest that while knee and ankle pain may reflect multifactorial etiologies such as degenerative changes or mechanical load, MTP involvement appears more specifically linked to urate-mediated mechanisms.

Regional aggregation further reinforced this anatomical gradient. Lower-limb joints demonstrated the highest overall site-level prevalence and the strongest mean absolute association with SUA compared with upper-limb and axial regions. This supports the concept that hyperuricemia-related musculoskeletal symptoms are not uniformly distributed but instead exhibit distal weight-bearing predominance. Prior cross-sectional studies have similarly reported preferential involvement of lower-limb joints in hyperuricemic populations, although the magnitude of association has varied across settings (19,24,41). The present findings contribute region-specific data from a Pakistani hospital-based cohort, addressing a local evidence gap previously identified in the literature (6).

The high burden of moderate-to-severe pain (64.7%) and severe-to-worst pain (35.3%) underscores the clinical significance of hyperuricemia in this population. Persistent elevation of SUA may contribute to inflammasome activation, oxidative stress, and subclinical synovitis, mechanisms increasingly implicated in chronic joint discomfort even outside acute gout episodes (43,44). The moderate strength of correlation observed here suggests that SUA is a meaningful, though not exclusive, determinant of musculoskeletal pain intensity. It is likely that additional metabolic, mechanical, and demographic factors interact with urate levels to shape symptomatic expression.

Sex-stratified analysis demonstrated that the SUA–pain intensity relationship remained significant in both males ($r = 0.591$) and females ($r = 0.524$), indicating that the biochemical–symptom gradient was not confined to one sex. Although hyperuricemia is traditionally more prevalent among males (16), the comparable correlation magnitudes observed here suggest that once elevated, SUA exerts similar symptomatic associations across sexes. This observation is consistent with prior mechanistic studies indicating that the inflammatory cascade triggered by urate crystals operates independently of sex after threshold saturation is exceeded (45).

Several methodological considerations merit discussion. The convenience sampling strategy and hospital-based setting may limit generalizability to the broader community, and the cross-sectional design precludes temporal or causal inference. Residual confounding by factors such as body mass index, renal function, occupational load, and osteoarthritis could influence joint-specific prevalence patterns. Nevertheless, standardized pain assessment using a validated VAS instrument and objective laboratory documentation of SUA strengthen internal validity. The study also adhered to observational reporting standards and incorporated sex-stratified analyses to explore potential effect modification (31).

The findings demonstrate a clinically relevant and anatomically selective association between elevated SUA and musculoskeletal pain intensity in hyperuricemic adults. The preferential biochemical linkage observed at the MTP joint, despite higher absolute prevalence at the knee and ankle, highlights the importance of site-specific evaluation when assessing urate-related symptomatology. Prospective longitudinal studies incorporating multivariable modeling and imaging confirmation of crystal deposition are warranted to clarify causal pathways and determine whether early urate-lowering interventions may mitigate musculoskeletal pain progression (46,47)

CONCLUSION

In this hospital-based cross-sectional study of hyperuricemic adults, elevated serum uric acid levels were significantly and moderately associated with increased musculoskeletal pain intensity, explaining a substantial proportion of symptom variability. Although knee and ankle pain were most prevalent, the metatarsophalangeal joint demonstrated the strongest and most specific biochemical association with serum uric acid, indicating anatomical selectivity in urate-related musculoskeletal involvement. These findings suggest that hyperuricemia is not solely a biochemical abnormality but is clinically linked to symptomatic burden, particularly in distal weight-bearing joints. Early recognition of site-specific joint pain in hyperuricemic patients may facilitate timely preventive and therapeutic strategies, while longitudinal, multivariable-adjusted studies are required to establish causality and inform evidence-based management approaches.

DECLARATIONS

Ethical Approval
This study was approved by the Institutional Review Board of Gulab Devi Hospital, Lahore
Informed Consent
Written informed consent was obtained from all participants included in the study.
Conflict of Interest
The authors declare no conflict of interest.
Funding
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Authors' Contributions
Concept: MA; Design: SS; Data Collection: MA, IU; Analysis: MA; Drafting: MA, SS.
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Data Availability
The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.
Acknowledgments
Not applicable.
Study Registration
Not applicable.

REFERENCES

1. Tang SCW. Gout: a disease of kings. Contrib Nephrol. 2017;192:77-81.

2. Paul BJ, Anoopkumar K, Krishnan V. Asymptomatic hyperuricemia: is it time to intervene? Clin Rheumatol. 2017;36(12):2637-44.

3. Hong F, Zheng A, Xu P, Wang J, Xue T, Dai S, et al. High-protein diet induces hyperuricemia in a new animal model for studying human gout. Int J Mol Sci. 2020;21(6).

4. Janssen CA, Oude Voshaar MAH, Vonkeman HE, Krol M, van de Laar MAFJ. A retrospective analysis of medication prescription records for determining the levels of

compliance and persistence to urate-lowering therapy for the treatment of gout and hyperuricemia in the Netherlands. Clin Rheumatol. 2018;37(8):2291-6.

5. Chalès G. How should we manage asymptomatic hyperuricemia? Joint Bone Spine. 2019;86(4):437-43.

6. Qidwai W, Jawaid MJ. Frequency of uric acid levels, symptomatic and asymptomatic hyperuricemia among the Pakistani population. 2017.

7. Wang H, Zhang H, Sun L, Guo W. Roles of hyperuricemia in metabolic syndrome and cardiac-kidney-vascular system diseases. Am J Transl Res. 2018;10(9):2749.

8. Kubota M. Hyperuricemia in children and adolescents: present knowledge and future directions. J Nutr Metab. 2019;2019:3480718.

9. George C, Minter DA. Hyperuricemia. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2020.

10. Su HY, Yang C, Liang D, Liu HF. Research advances in the mechanisms of hyperuricemia-induced renal injury. Biomed Res Int. 2020;2020:5817348.

11. Perez-Ruiz F, Dalbeth N, Bardin T. A review of uric acid, crystal deposition disease, and gout. Adv Ther. 2015;32(1):31-41.

12. 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.

13. Burhan H, Choudry U, Umerani M, Sharif S, Nisar A. Hyperuricemia in patients with chronic low back pain: experience from a single institutional neurosurgical OPD. J Surg Emerg Med. 2017;1(7).

14. Pascual E, Martínez A, Ordóñez S. Gout: the mechanism of urate crystal nucleation and growth. A hypothesis based in facts. 2013.

15. Park DY, Kim YS, Ryu SH, Jin YS. The association between sedentary behavior, physical activity and hyperuricemia. Vasc Health Risk Manag. 2019;15:291-9.

16. Shaikhomar O, Header E. Dietary etiological factors contributing to the prevalence of hyperuricemia in Makkah region. Prensa Med Argent. 2020;Suppl 2:1-7.

17. Zeng J, Zhang J, Li Z, Li T, Li G. Prediction model of artificial neural network for the risk of hyperuricemia incorporating dietary risk factors in a Chinese adult study. Food Nutr Res. 2020;64.

18. Min HK, Cho H, Park SH. Pilot study: asymptomatic hyperuricemia patients with obesity and nonalcoholic fatty liver disease have increased risk of double contour sign. Korean J Intern Med. 2020;35(6):1517.

19. Comberg HU, Schach S. Hyperuricemia is associated with musculo-skeletal pain-results from a cross-sectional study. Open Pain J. 2016;9(1).

20. Star VL, Hochberg MC. Prevention and management of gout. Drugs. 1993;45(2):212-22.

21. Kakutani-Hatayama M, Kadoya M, Okazaki H, Kurajoh M, Shoji T, Koyama H, et al. Nonpharmacological management of gout and hyperuricemia: hints for better lifestyle. J Clin Med. 2017;11(4):321-9.

22. Pan F, Byrne KS, Ramakrishnan R, Ferreira M, Dwyer T, Jones G. Association between musculoskeletal pain at multiple sites and objectively measured physical activity and work capacity: results from UK Biobank study. J Sci Med Sport. 2019;22(4):444-9.

23. Lai TL, Yim CW, Wong P, Leung M, Ng W. Hyperuricemia in Asian psoriatic arthritis patients. Int J Rheum Dis. 2018;21.

24. Wang S, Pillinger M, Krasnokutsky S, Barbour K. The association between asymptomatic hyperuricemia and knee osteoarthritis: data from the Third National Health and Nutrition Examination Survey. Osteoarthritis Cartilage. 2019;27.

25. He F, Wang LL, Yu XL. Dietary patterns associated hyperuricemia among Chinese aged 45 to 59 years: an observational study. Medicine (Baltimore). 2017;96:e9248.

26. Wu J, Qiu L, Cheng XQ, Xu T, Wu W, Zeng XJ, et al. Hyperuricemia and clustering of cardiovascular risk factors in the Chinese adult population. Sci Rep. 2017;7(1):5456.

27. Kusumayanti GAD, Dewantari NM. The influence of low purine diet and physical activity on changing of uric acid levels in hyperuricemia. Int J Health Sci. 2017;1(3):1-9.

28. Xiong H, Sun Z, Chen S, Liu W, Peng S, Wang W, et al. Effect of hyperuricemia on functional outcomes and complications in patients with elbow stiffness after open arthrolysis combined with hinged external fixation: a retrospective study. J Shoulder Elbow Surg. 2020;29:1387-93.

29. Jonsson H, Aspelund T, Eiriksdottir G, Harris T, Launer L, Gudnason V. Hyperuricemia is associated with intermittent hand joint pain in a cross-sectional study of elderly females: the AGES-Reykjavik Study. PLoS One. 2019;14:e0221474.

30. Liu Z, Su X, Xiao M, Zhou P, Guo J, Huang Y, et al. Association between eating away from home and hyperuricemia: a population-based nationwide cross-sectional study in China. Biomed Res Int..