

ORIGINAL RESEARCH

Association between hyperuricaemia
and hand osteoarthritis: data from the
Xiangya Osteoarthritis Study

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ABSTRACT

Objective The pathogenesis of hand osteoarthritis (OA) remains unknown. Hyperuricaemia, which is related to inflammation, may play a role in hand OA, but evidence is lacking. In a large population-based study, we examined the association between hyperuricaemia and hand OA.

Methods Participants were from the Xiangya OA Study, a community-based observational study. Hyperuricaemia was defined as serum urate >416 µmol/L in men and >357 µmol/L in women. Radiographic hand OA (RHOA) was defined as presence of the modified Kellgren-Lawrence grade ≥2 in any hand joint. Symptomatic hand OA (SHOA) was defined as presence of both self-reported symptoms and RHOA in the same hand. The associations of hyperuricaemia with RHOA or SHOA were examined using generalised estimating equations.

Results Among 3628 participants, the prevalence of RHOA was higher in participants with hyperuricaemia than those with normouricaemia (26.9% vs 20.9%), with an adjusted OR (aOR) of 1.34 (95% CI 1.11 to 1.61). The associations were consistent in men (aOR 1.33, 95% CI 1.01 to 1.74) and women (aOR 1.35, 95% CI 1.05 to 1.74). Hyperuricaemia was mainly associated with bilateral RHOA (aOR 1.54, 95% CI 1.18 to 2.01) but not unilateral RHOA (aOR 1.13, 95% CI 0.89 to 1.45). Prevalence of SHOA was higher, although statistically insignificant, in participants with hyperuricaemia (aOR 1.39, 95% CI 0.94 to 2.07).

Conclusion In this population-based study, hyperuricaemia was associated with a higher prevalence of hand OA. Future prospective studies are required to investigate the temporal relationship.

Trial registration number NCT04033757.

INTRODUCTION

Osteoarthritis (OA) is a highly prevalent condition, and the hand is one of the most commonly affected sites, with the prevalence of radiographic hand OA (RHOA) ranging from 18.9% to 61.7%,^{1–5} and the prevalence of symptomatic hand OA (SHOA) ranging from 3% to 16% globally.¹⁶ Hand OA can cause pain, decreased grip and pinch strength, impaired function and reduced quality of life.¹⁶ To date,

WHAT IS ALREADY KNOWN ON THIS TOPIC

- ⇒ The pathogenesis of hand osteoarthritis (OA) remains largely unknown, and there is a need for more data to help understand the aetiology and inform the development of prevention and treatment strategies for hand OA.
- ⇒ Previous studies have demonstrated that hyperuricaemia can induce a pro-inflammatory response, which may play a role in the pathogenesis of OA.
- ⇒ While most previous studies have reported positive associations between hyperuricaemia and knee OA, few epidemiological studies have examined the association between hyperuricaemia and hand OA.

WHAT THIS STUDY ADDS

- ⇒ Using data from a community-based cohort study conducted in rural area of China, we found that participants with hyperuricaemia had a higher prevalence of radiographic hand OA than those with normouricaemia.
- ⇒ Hyperuricaemia was also associated with bilateral radiographic hand OA but not unilateral hand OA, indicating that hyperuricaemia was associated with more widespread radiographic hand OA.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

- ⇒ The findings suggest that hyperuricaemia was associated with a higher prevalence of hand OA.
- ⇒ Further prospective studies of their temporal relationship are required to investigate causality.

the pathogenesis of hand OA remains largely unknown, and there is a need for more data to help understand the aetiology and inform the development of prevention and treatment strategies for hand OA.⁷

Hyperuricaemia is a common metabolic disorder caused by abnormally high serum urate levels.⁸ Previous studies have demonstrated that hyperuricaemia can induce a pro-inflammatory response, which may play a role in the pathogenesis of OA.^{9–12} In addition,

monosodium urate crystal deposition from hyperuricaemia may create local mechanical and inflammatory damage to joint tissues leading to the development or progression of OA.¹³ However, few epidemiological studies have examined the association between hyperuricaemia and hand OA. Since hand OA is common and hyperuricaemia is a modifiable condition, elucidating their relation has potential clinical and public health implications.

To fill this knowledge gap, using data collected from the Xiangya OA (XO) Study,^{14–17} we conducted a population-based observational study to investigate the association between hyperuricaemia and hand OA.

METHODS

Study design and population

Participants were from the XO Study, a community-based longitudinal study conducted in Longshan County, Hunan Province, China (NCT04033757), primarily focused on the natural history and risk factors of OA. Details of the XO Study have been described previously.^{14–17} Briefly, it comprises three subcohorts (ie, subcohorts I, II and III), and participants of each subcohort were recruited in 2015, 2018 and 2019, respectively. First, we adopted a probability proportionate to size sampling method to select 14 communities in Longshan County. Then, all villages in the selected communities were listed in random order. Beginning with the first village in the first community, residents aged 50 years or older were invited to participate, and a total of 25 rural villages of Longshan County were eventually included in the XO Study. Using the baseline data of the XO Study, we conducted a cross-sectional study to evaluate the association between hyperuricaemia and hand OA. Individuals who did not undergo an X-ray assessment or provide a blood sample were excluded from the study. Additionally, participants with the self-reported rheumatic disease were excluded to differentiate the impact of other rheumatic conditions.

Assessment of hand OA

Participants underwent a posterior-anterior radiograph of both hands. The second to fifth distal interphalangeal, second to fifth proximal interphalangeal, first to fifth metacarpophalangeal, thumb interphalangeal and thumb base (first carpometacarpal) joints in each hand

were graded using the Kellgren-Lawrence (KL) grade for RHOA.^{18–20} In this scale, 0=normal; 1=questionable osteophytes and/or joint space narrowing; 2=definite small osteophytes and/or mild joint space narrowing; 3=moderate osteophytes and/or moderate joint space narrowing, sclerosis and the potential presence of central erosions; 4=large osteophytes and/or severe joint space narrowing, sclerosis and the potential presence of central erosions (figure 1). Even without osteophytes, a diagnosis of definite OA (grade 2 or higher) can be made solely based on joint space narrowing. RHOA was defined as a KL grade ≥ 2 in any of the joints listed above in each hand. The intra-rater and inter-rater reliabilities for RHOA as a dichotomous variable expressed by the kappa statistic were 0.91 (95% CI 0.83 to 0.99) and 0.71 (95% CI 0.45 to 0.96), respectively.¹⁴

Hand symptoms were ascertained by a ‘yes’ response to the question, “On most days, do you have pain, aching, or stiffness in your left/right hand?” We employed a clinical doctor to conduct the questionnaire interview; only symptoms deemed relevant to OA were recorded as hand pain. A hand would be diagnosed with SHOA if both self-reported symptoms and radiographic OA were presented in the same hand.^{14 21}

Assessment of hyperuricaemia

Blood samples were taken in the morning after at least 12 hours of fasting and stored at 4°C until analysis. Blood was aspirated into a vacutainer tube containing EDTA and allowed to clot before being centrifuged at 3000 rpm for 15 min for serum separation. Serum urate was analysed at the Clinical Laboratory of Xiangya Hospital and detected on a Beckman Coulter AU 5800 (Beckman Coulter, Brea, California, USA). The inter-assay and intra-assay coefficients of variation for serum urate were tested for low (118 $\mu\text{mol/L}$) and high concentrations (472 $\mu\text{mol/L}$). The intra-assay coefficients of variation were 1.39% (118 $\mu\text{mol/L}$) and 0.41% (472 $\mu\text{mol/L}$), respectively, and inter-assay coefficients of variation were 1.40% (118 $\mu\text{mol/L}$) and 1.23% (472 $\mu\text{mol/L}$), respectively. A participant was defined as having hyperuricaemia if the serum urate level was $>416 \mu\text{mol/L}$ (7.0 mg/dL) in men and $>357 \mu\text{mol/L}$ (6.0 mg/dL) in women, which is above the accepted normal range in the Chinese population.^{22 23}

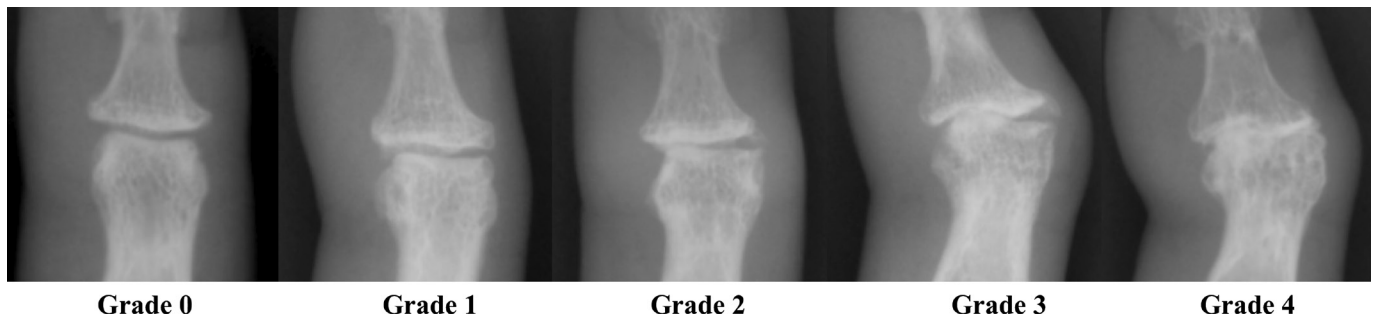


Figure 1 Representative examples of KL grade of RHOA. KL, Kellgren-Lawrence; RHOA, radiographic hand osteoarthritis.

Assessment of covariates

Data were collected by trained health professional researchers using standard questionnaires in a face-to-face interview. Age, sex, smoking status, alcohol consumption, history of hand injury, diabetes and hypertension were recorded. Height and weight were measured, and body mass index (BMI) was calculated as weight (kg) divided by the square of height (m^2). Blood pressure was checked on an electronic sphygmomanometer. The fasting blood glucose was also detected on a Beckman Coulter AU 5800 (Beckman Coulter). Hypertension was defined as systolic blood pressure ≥ 140 mm Hg or diastolic blood pressure ≥ 90 mm Hg or if the participant was taking anti-hypertensive treatment.²⁴ Diabetes was diagnosed as a fasting glucose level ≥ 7.0 mmol/L or if the participant was receiving drug treatment to control blood glucose.²⁵

Statistical analysis

In the analysis, continuous variables were presented as mean with SD, and categorical variables were expressed as percentages. We calculated the prevalence of hand OA for participants according to hyperuricaemia status. We compared the baseline characteristics between hyperuricaemia and normouricaemia and presented the standardised mean difference.²⁶ We performed a hand-based analysis to examine the association between hyperuricaemia and hand OA in both hands using the generalised estimating equations.²⁷ This approach could adjust for hand-specific confounder (ie, history of hand injury) to minimise misclassification of confounder measurement and, in general, improve the precision of the effect estimate when compared with a person-based analysis. The binary distribution and the logit linkage of the PROC GENMOD program in SAS were used to calculate the OR and its 95% CI of RHOA or SHOA according to the status

of hyperuricaemia. The adjusted OR (aOR) was adjusted for age (<60 years, ≥ 60 years), sex (men, women), BMI (<24 kg/ m^2 , 24 – 28 kg/ m^2 , ≥ 28 kg/ m^2), smoking status (never, past, current), alcohol consumption (never, past, current), history of hand injury (yes, no), diabetes (yes, no) and hypertension (yes, no).

Several additional analyses were performed to test the robustness of the findings. First, we tested whether the associations between hyperuricaemia and hand OA differed between men and women through sex-specific subgroup analyses. Second, we evaluated the association of hyperuricaemia with the laterality of hand OA using the nominal logistic regression model. Third, we excluded participants with self-reported gout to differentiate the effects of crystal-induced inflammation from hyperuricaemia. Fourth, we performed person-based analyses using logistic regression analysis to evaluate the association between hyperuricaemia and hand OA. Fifth, we conducted sensitivity analyses by restricting the OA diagnosis to the distal interphalangeal joints, as changes in the distal interphalangeal joints were the most common features of hand OA.²⁸

Statistical analyses were conducted using SAS V.9.4 (SAS Institute, Cary, North Carolina, USA). All p values were two-sided, and $p < 0.05$ was considered statistically significant.

RESULTS

Of 4080 participants from the XO Study, we excluded participants who did not undergo hand radiographs ($n=45$), or did not provide blood samples ($n=235$), or reported a history of rheumatic diseases ($n=172$). The final sample comprised 3628 participants. The selection process of study participants is shown in figure 2. The

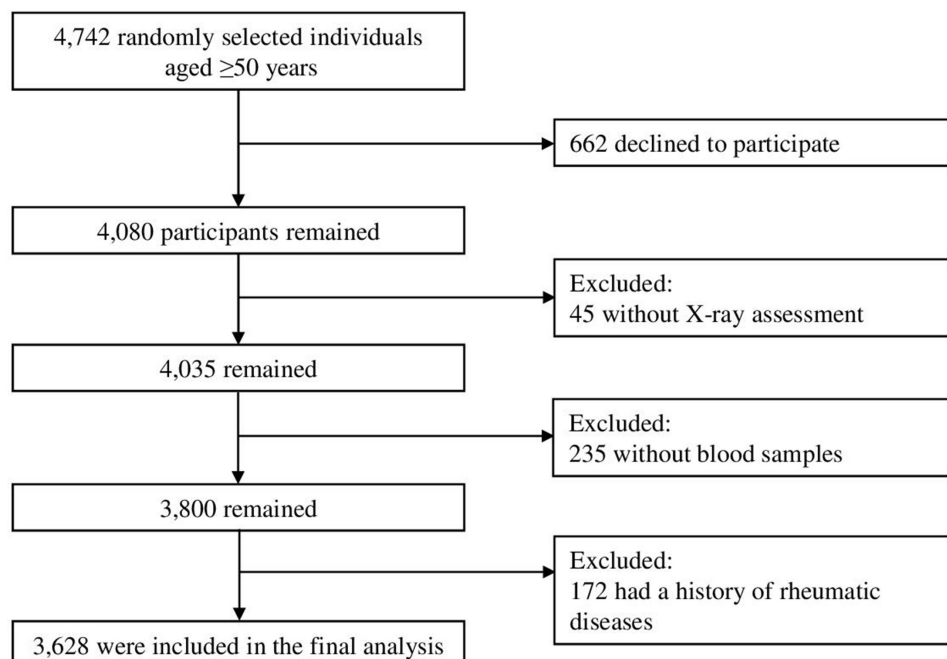


Figure 2 The selection process of included participants in the study.

Table 1 Characteristics of the study participants at baseline

	Total (n=3628)	Hyperuricaemia (n=649)	Normouricaemia (n=2979)	SMD for hyperuricaemia versus normouricaemia
Sex				0.223
Women, n (%)	2024 (55.8)	303 (46.7)	1721 (57.8)	
Men, n (%)	1604 (44.2)	346 (53.3)	1258 (42.2)	
Age, years (mean±SD)	63.7±9.3	65.8±9.8	63.2±9.1	0.274
BMI, kg/m ² (mean±SD)	23.9±3.5	24.6±4.0	23.8±3.4	0.224
Serum urate, µmol/L (mean±SD)	315.2±89.7	455.2±71.7	284.6±59.0	2.810
Smoking status (%)				0.227
Non-smoker	64.5	55.8	66.3	
Ex-smoker	4.5	6.9	4.0	
Current smoker	31.0	37.3	29.7	
Alcohol drinking (%)				0.163
Non-drinker	54.2	49.1	55.2	
Ex-drinker	11.3	15.4	10.5	
Current drinker	34.5	35.5	34.3	
History of hand injury (%)	6.6	5.4	6.9	0.064
Diabetes (%)	7.5	10.0	7.0	0.109
Hypertension (%)	49.4	59.5	47.2	0.248

BMI, body mass index; SMD, standardised mean difference.

characteristics of the participants are shown in [table 1](#). The mean age of included participants was 63.7±9.3 years, 55.8% were women, the mean BMI was 23.9±3.5 kg/m² and the prevalence of hyperuricaemia was 17.9%. The mean serum urate levels in the groups with hyperuricaemia and normouricaemia were 455.2±71.7 µmol/L and 284.6±59.0 µmol/L, respectively. The standardised mean differences of covariates except hand injury were >0.1 between population with hyperuricaemia and normouricaemia.

Results of the associations between hyperuricaemia and the prevalence of RHOA are presented in [table 2](#). The prevalence of RHOA was higher in participants with hyperuricaemia (26.9%) than in the population with normouricaemia (20.9%). After adjusting for potential confounders, the aOR of RHOA was 1.34 (95% CI 1.11 to 1.61) for hyperuricaemia compared with the population with normouricaemia. The results of the sex subgroup analyses were consistent with the main results, with aORs of 1.33 (95% CI 1.01 to 1.74) for men and 1.35 (95% CI 1.05 to 1.74) for women ([table 2](#)). The associations of hyperuricaemia with unilateral and bilateral RHOA are presented in [table 3](#). The prevalence of unilateral and bilateral RHOA were 19.6% and 17.6% in participants with hyperuricaemia, and 17.6% and 12.4% in those with normouricaemia, respectively. The corresponding aORs for unilateral and bilateral RHOA were 1.13 (95% CI 0.89 to 1.45) and 1.54 (95% CI 1.18 to 2.01), respectively, in participants with hyperuricaemia compared with those with normouricaemia (P for trend=0.005). We also observed more profound associations of hyperuricaemia

with bilateral RHOA in men (aOR 1.77, 95% CI 1.22 to 2.58), compared with the associations of hyperuricaemia with unilateral RHOA (aOR 0.90, 95% CI 0.63 to 1.29).

The prevalence of SHOA was also higher, although statistically insignificant, in the participants with hyperuricaemia (7.3%) than in the population with normouricaemia (5.5%), with an aOR of 1.39 (95% CI 0.94 to

Table 2 Association between hyperuricaemia and RHOA

	Hyperuricaemia	
	No	Yes
Total		
Number with RHOA, n (%)	1205 (20.9)	336 (26.9)
Crude OR (95% CI)	1.00 (reference)	1.39 (1.17 to 1.65)
Adjusted OR (95% CI)*	1.00 (reference)	1.34 (1.11 to 1.61)
Men		
Number with RHOA, n (%)	483 (19.9)	167 (25.3)
Crude OR (95% CI)	1.00 (reference)	1.36 (1.06 to 1.74)
Adjusted OR (95% CI)†	1.00 (reference)	1.33 (1.01 to 1.74)
Women		
Number with RHOA, n (%)	722 (21.6)	169 (28.7)
Crude OR (95% CI)	1.00 (reference)	1.46 (1.15 to 1.86)
Adjusted OR (95% CI)†	1.00 (reference)	1.35 (1.05 to 1.74)

*Adjusted for age, sex, body mass index, smoking status, alcohol consumption, history of hand injury, diabetes and hypertension.
†Adjusted for age, body mass index, smoking status, alcohol consumption, history of hand injury, diabetes and hypertension.
RHOA, radiographic hand osteoarthritis.

Table 3 Association of hyperuricaemia with unilateral and bilateral RHOA

	Non-RHOA	Unilateral RHOA	Bilateral RHOA	P for trend
Total				
Crude OR (95% CI)	1.00 (reference)	1.24 (0.99 to 1.56)	1.59 (1.25 to 2.02)	<0.001
Adjusted OR (95% CI)*	1.00 (reference)	1.13 (0.89 to 1.45)	1.54 (1.18 to 2.01)	0.005
Men				
Crude OR (95% CI)	1.00 (reference)	0.96 (0.68 to 1.34)	1.74 (1.23 to 2.46)	0.031
Adjusted OR (95% CI)†	1.00 (reference)	0.90 (0.63 to 1.29)	1.77 (1.22 to 2.58)	0.054
Women				
Crude OR (95% CI)	1.00 (reference)	1.58 (1.16 to 2.15)	1.52 (1.08 to 2.14)	0.001
Adjusted OR (95% CI)†	1.00 (reference)	1.39 (0.99 to 1.95)	1.38 (0.94 to 2.03)	0.048

*Adjusted for age, sex, body mass index, smoking status, alcohol consumption, history of hand injury, diabetes and hypertension.

†Adjusted for age, body mass index, smoking status, alcohol consumption, history of hand injury, diabetes and hypertension. RHOA, radiographic hand osteoarthritis.

2.07) (table 4). In addition, as shown in table 5, the aOR of unilateral and bilateral SHOA for hyperuricaemia was 1.06 (95% CI 0.63 to 1.77) and 1.73 (95% CI 1.00 to 2.97), respectively. Results from the sex subgroup analyses were similar to those of the total population (tables 4 and 5).

Results from sensitivity analyses that excluded participants with self-reported gout (n=35) were consistent with the primary results (online supplemental tables S1 and S2). Although the magnitude of associations from the hand-based analyses was similar to that from the person-based analyses, the SEs of ORs from the hand-based analyses were smaller than those from the person-based analyses, indicating the hand-based analyses provided more precise estimates than the person-based analyses (online supplemental table S3). Furthermore, the results by restricting the OA diagnosis to the distal interphalangeal joints remained consistent with the primary results (online supplemental table S4).

DISCUSSION

In this large community-based study, hyperuricaemia was associated with an increased prevalence of RHOA independent of some major confounding factors. Hyperuricaemia was also associated with bilateral RHOA, but not unilateral RHOA, indicating that hyperuricaemia was associated with more widespread RHOA. We also found that the prevalence of SHOA was higher in the population with hyperuricaemia than in the population with normouricaemia. Results from the subgroup analyses stratified by sex were consistent with the total population analyses.

Previous studies have focused on correlations between hyperuricaemia and OA in weight-bearing joints (eg, knee), and most have found positive associations between hyperuricaemia and knee OA.^{29–32} Ding *et al* found a positive association between asymptomatic hyperuricaemia and the presence of osteophytes on knee radiographs in women.²⁹ Musacchio *et al* observed

a significant correlation between hyperuricaemia and knee OA in older men.³⁰ Results from the third National Health and Nutrition Examination Survey also showed that older adults with asymptomatic hyperuricaemia had a 69% higher prevalence of knee OA than their counterparts.³¹ A similar finding was also reported in another cross-sectional study.³² To our knowledge, only one study has been conducted to examine the association between hyperuricaemia and prevalent hand OA, which reported a positive association between hyperuricaemia and hand OA in women but not in men.³⁰ However, the number of men (n=1182) in that study was smaller than in women (n=1789); thus, study may be lack of power to test hypothesis in men. In the current study, we observed that prevalence of RHOA was higher in participants with

Table 4 Association between hyperuricaemia and SHOA

	Hyperuricaemia	
	No	Yes
Total		
Number with SHOA, n (%)	228 (5.5)	63 (7.3)
Crude OR (95% CI)	1.00 (reference)	1.36 (0.94 to 1.97)
Adjusted OR (95% CI)*	1.00 (reference)	1.39 (0.94 to 2.07)
Men		
Number with SHOA, n (%)	58 (3.2)	27 (5.7)
Crude OR (95% CI)	1.00 (reference)	1.85 (1.00 to 3.43)
Adjusted OR (95% CI)†	1.00 (reference)	1.84 (0.98 to 3.45)
Women		
Number with SHOA, n (%)	170 (7.3)	36 (9.3)
Crude OR (95% CI)	1.00 (reference)	1.30 (0.81 to 2.08)
Adjusted OR (95% CI)†	1.00 (reference)	1.11 (0.66 to 1.86)

*Adjusted for age, sex, body mass index, smoking status, alcohol consumption, history of hand injury, diabetes and hypertension.

†Adjusted for age, body mass index, smoking status, alcohol consumption, history of hand injury, diabetes and hypertension. SHOA, symptomatic hand osteoarthritis.

Table 5 Association of hyperuricaemia with unilateral and bilateral SHOA

	Non-SHOA	Unilateral SHOA	Bilateral SHOA	P for trend
Total				
Crude OR (95% CI)	1.00 (reference)	1.06 (0.65 to 1.72)	1.66 (1.00 to 2.75)	0.142
Adjusted OR (95% CI)*	1.00 (reference)	1.06 (0.63 to 1.77)	1.73 (1.00 to 2.97)	0.152
Men				
Crude OR (95% CI)	1.00 (reference)	0.84 (0.32 to 2.24)	2.62 (1.21 to 5.68)	0.119
Adjusted OR (95% CI)†	1.00 (reference)	0.87 (0.32 to 2.36)	2.56 (1.15 to 5.70)	0.116
Women				
Crude OR (95% CI)	1.00 (reference)	1.33 (0.76 to 2.35)	1.35 (0.67 to 2.73)	0.211
Adjusted OR (95% CI)†	1.00 (reference)	1.10 (0.60 to 2.02)	1.18 (0.54 to 2.58)	0.656

*Adjusted for age, sex, body mass index, smoking status, alcohol consumption, history of hand injury, diabetes and hypertension.

†Adjusted for age, body mass index, smoking status, alcohol consumption, history of hand injury, diabetes and hypertension. SHOA, symptomatic hand osteoarthritis.

hyperuricaemia than those without hyperuricaemia after adjusting for potential confounders in the total population and in men and women separately. However, the association between hyperuricaemia and SHOA was stronger in men than in women, although statistically insignificant. The urate levels in men were much higher in men than in women,³³ whereas the prevalence of SHOA in men was generally lower than that in women.²⁰ As a result, one would expect a larger OR in men than in women if the association existed. Moreover, results from the sensitivity analyses by excluding participants with self-reported gout, person-based analyses or restricting the OA diagnosis to the distal interphalangeal joints further supported the robustness of our findings. In addition, the positive association between hyperuricaemia and bilateral RHOA underscored the systemic, rather than localised, role of hyperuricaemia.

The results indicated a positive association between hyperuricaemia and hand OA. One of the possible explanations is that hyperuricaemia could influence an inflammation response, which is involved in the pathogenesis of OA.^{34 35} Evidence suggests that OA may have a different profile of pathological mechanisms in different joints, with hand OA being more linked to systemic inflammation and knee OA more likely resulting more from excessive biomechanical joint loading and injury.^{1 36} Hyperuricaemia has been reported to promote low-level systemic inflammatory states.³⁷ On the one hand, soluble urate has pro-inflammatory and pro-oxidative effects in the intracellular environment.¹⁰ On the other hand, at higher concentrations, urate can crystallise as monosodium urate, stimulating the NLRP3 inflammasome and potentially promoting interleukin-1 β production and cartilage, bone and other tissue damage.⁹ Elevated serum urate has been associated with several markers of systemic inflammation, including white blood cells, C reactive protein and various serum levels of inflammatory cytokines (including interleukin-6, interleukin-18 and tumour necrosis factor- α).³⁸ Hyperuricaemia was only associated with bilateral

but not unilateral hand OA, indicating that urate may play a systemic rather than a local role in the development of OA. Several in vitro studies have also indicated that exposure to urate may have adverse effects on chondrocytes that mimic intrinsic OA processes.^{12 39} The underlying mechanisms linking hyperuricaemia to hand OA remain unclear. It has been postulated that the role of hyperuricaemia in OA can be contextualised within a broader framework of metabolic dysregulation.⁴⁰ Metabolic syndrome, a cluster of conditions including hyperuricaemia, has been increasingly recognised as a risk factor for OA.⁴¹ The shared inflammation pathways, oxidative stress and adipokine dysregulation also bridge metabolic disturbances with joint degeneration.

There are several strengths to this study. First, the XO cohort is a randomly selected sample from the general population in a rural area of China; thus, the findings are generalisable to the general population of the same kind in China. Second, with the comprehensive data collected in the study, we adjusted for many potential confounders to minimise confounding bias. However, the limitations of the present study should also be acknowledged. First, we tested the research hypothesis using across-sectional data; thus, we cannot establish the temporal relationship between hyperuricaemia and hand OA. Second, we excluded participants with self-reported rheumatic diseases; however, this approach may be susceptible to misclassification bias. Third, hand pain for reasons other than OA may also lead to misclassification of SHOA. We suspect such misclassification, if it existed, which is likely to be non-differential, and thus biases the association towards the null.

Hand OA is a highly prevalent, potentially disabling condition with a large individual and socioeconomic burden.^{42 43} How to prevent and manage it is an important unmet need in rheumatology and public health.⁴⁴ Our study found that hyperuricaemia was related to a higher prevalence of hand OA. Hyperuricaemia is very common and is potentially modifiable with urate-lowering treatment. Our analysis indicates that hyperuricaemia may be an important modifiable risk factor for hand OA, which

deserves more attention and future investigations at clinical and public health levels.

CONCLUSION

In this population-based study, hyperuricaemia was associated with a higher prevalence of hand OA. Future prospective studies are required to investigate the temporal relationship.

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