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Analysis of risk factors for changes of renal artery resistance indexes in gout patients by ultrasound colour Doppler

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Abstract

Objectives: Gout may disturb renal hemodynamics by promoting uric acid deposition; however, this relationship has not been elucidated with adequate clinical evidence. In this study, we measured the renal artery resistance index (ARI) in patients with gout to identify the risk factors and establish predictive models for elevated renal ARI in these patients.

Methods: Renal artery ultrasound examination was performed in 235 primary gout patients and 50 healthy controls (HCs); subsequently, their renal interlobar ARI (RIARI), renal segmental ARI (RSARI) and overall intrarenal ARI (OIARI) were recorded. Each ARI >0.7 was considered elevated.

Results: RIARI, RSARI and OIARI were higher in patients with gout than in HCs (all $P<0.001$). Nineteen (8.1%), 24 (10.2%) and 18 (7.7%) patients had elevated RIARI, RSARI and OIARI scores, respectively. Multivariate logistic regression analyses disclosed that: age ≥ 60 years ($P=0.000$), abnormal beta2 microglobulin (β_2MG) ($P=0.028$), and abnormal high-density lipoprotein cholesterol (HDLC) ($P=0.030$) were independently associated with elevated RIARI; age ≥ 60 years ($P=0.000$), and abnormal β_2MG ($P=0.013$) were independently related to elevated RSARI; abnormal total protein (TP) ($P=0.014$) were independently linked with elevated OIARI in gout patients. Consequently, predictive models for elevated ARI were established using nomograms based on the aforementioned independent risk factors, which showed a satisfactory value for estimating elevated RIARI [area under the curve (AUC):0.929], RSARI (AUC: 0.926) and OIARI (AUC: 0.660) in patients with gout, as validated by receiver operating characteristic curves.

Conclusion: Renal ARI were elevated in patients with gout, whose independent risk factors included older age and abnormal β_2MG, HDLC and TP levels.

Lay Summary

What does this mean for patients?

Gout is a type of arthritis that causes sudden pain and swelling due to a build up of crystals in your joints. It is commonly associated with kidney problems. Nearly 25% of people with gout have chronic kidney disease. As a result, their kidney function requires timely monitoring. Ultrasound imaging can be used to find out someone's renal artery resistance index (ARI), which measures kidney function. An ARI score of over 0.7 is considered too high. When we scanned 235 people with gout and 50 healthy people, we found that ARI was higher in people with gout than healthy people. ARI score was also related to factors like age, cholesterol and the amount of protein in your blood. We suggest that monitoring factors like age, cholesterol and blood protein can help doctors to predict kidney problems in people with gout.

Keywords: gout, renal artery resistance index, renal artery ultrasound examination, risk factor, nomogram

Key messages

- Renal ARI were higher in patients with gout than in HCs.
- Elevated renal ARI in patients with gout is independently related to age, β2MG, HDLC and TP.
- Predictive models combining these risk factors show favourable values for identifying the risk of elevated renal ARI in gout patients.

Introduction

Gout is a metabolic disease that is prevalent in middle-aged and elderly men. The typical symptoms include hyperuricemia, intermittent acute arthritis episodes (also called gout flares) and tophus [1, 2]. The global incidence of gout is estimated to range from 0.058% to 0.289% per year, and its global prevalence is rising as well [3]. For the purpose of pain control and inflammation suppression, colchicine, non-

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steroidal anti-inflammatory drug, and oral corticosteroids are recommended as the first-line treatment for acute gout, while urate-lowering therapy is considered as long-term gout management [4–6]. Notably, gout is often accompanied by changes in the renal hemodynamics and intrarenal vascular diseases due to renal under-excretion of uric acid and chronic inflammation, which add to the risk of kidney failure, bring additional disease burden, and thus deserve close monitoring [7–10].

The study of renal vascular lesions in gout patients was first seen in the 19th century, when scholars found the destruction of renal tubules and small arteries in the kidneys of gout patients [11]. Sánchez-Lozada *et al.* [12] found that in a hyperuricemia rat model elevated uric acid induced intrarenal arteriolar lesions leading to thickening of afferent arterioles with vasoconstriction of the renal cortex. One study showed that, gout patients with articular monosodium urate deposits compared with those without had higher renal artery resistance index (ARI) [13].

The ARI, obtained from renal artery ultrasound examination, is an indicator of the renal artery flow velocity, whose increment represents reversible renal dysfunction, and is useful for tracking the progression of renal diseases [14–16]. Previous studies have investigated the risk factors for an increase in the renal ARI in patients with renal disease and those at a high risk of renal disorders [17, 18]. For instance, one study found that renal ARI is elevated in hypertensive patients with renal damage compared with those without and is positively linked to age, cystatin C (CysC) and β2-microglobulin in hypertensive patients with renal damage [19]. Another study showed that age, pulse pressure, diabetes and serum asymmetric dimethylarginine levels were independent factors influencing the renal ARI in renal allograft recipients [17]. Furthermore, a previous study finds that the risk factors for elevated renal ARI in chronic kidney disease include increased age, female sex, diabetes mellitus, coronary artery disease, peripheral vascular disease, higher systolic blood pressure and the use of β blockers [18]. Since patients with gout are also at a high risk of renal disorders, exploring the influencing factors for renal ARI in these patients is helpful for risk stratification and providing timely intervention; however, this issue lacks relevant exploration.

Here, we quantified renal ARI using renal artery ultrasound examination to explore the risk factors and establish preliminary predictive models for elevated renal ARI in patients with gout.

Methods

Subjects

Between November 2019 and January 2021, 235 patients with primary gout, who were admitted at our hospital were serially included in this study. The inclusion criteria were set as: (i) patients diagnosed with primary gout via the American College of Rheumatology/European League Against Rheumatism Gout classification criteria [4]; (ii) patients ≥16 years old; (iii) during intermittent period; (iv) had the plan to receive renal artery ultrasound examination; (v) volunteered for participation. The exclusion criteria were set as: (i) patients diagnosed with secondary gout or pseudogout caused by radiotherapy, chemotherapy, blood diseases and medication; (ii) had asymptomatic hyperuricemia; (iii) had rheumatoid arthritis, ankylosing spondyloarthritis, or other autoimmune diseases; (iv) had other infectious diseases; (v) had primary type I and type II diabetes mellitus; (vi) had primary diffuse renal disease or secondary diseases that affected renal function, or underwent renal replacement therapy; (vii) had heart failure, permanent atrial fibrillation, moderate to severe aortic, or mitral valve disease; (viii) received drugs related to the lowering of uric acid, hypertension or glucose; (ix) had a trauma or surgery history of the knee, ankle or the first metatarsophalangeal joint. Overall, 50 healthy subjects were enrolled as healthy controls (HCs). The inclusion criteria for HCs were set as: (i) individuals without any abnormities in physical examinations; (ii) individuals ≥16 years old; (iii) individuals willing to participate and receive renal artery ultrasound examination. The study was approved by the Ethics Committee of the First Affiliated Hospital of Chengdu Medical College (No. 2019CYFYHEC-BA-34), and written informed consent was obtained from all subjects.

Collection

Demographics, chronic comorbidities, and disease characteristics of patients with gout were collected. In addition, 3 ml of venous blood samples were collected from patients with gout in the morning on an empty stomach (without food and drink for at least 8 h) after enrolment. The levels of routine blood indices, liver function indices, renal function indices, lipid metabolism-related indices, blood glucose-related indices, and inflammation-related indices were measured. Detection was performed by experienced investigators using an automated hematology analyser (SYSMEX, xn-9000, Japan), an automated biochemical analyser (Hitachi Limited, 7600, Japan), and an automated sedimentation rate analyser (VITAL diagnostics, MONITOR-100, Italy).

Renal artery ultrasound examination

A renal artery ultrasound examination was conducted using colour Doppler ultrasound diagnostic equipment (PHILIPS, EPIQ7c, Washington, USA) with a convex array probe (probe frequency– 1–5 MHz). The examination was performed using two-dimensional ultrasonography. The measurements included the size, shape and echo patterns of the kidneys and kidney stones. In addition, the images were divided into three types according to the presence and distribution of calculi: (i) no calculi, manifested as uniform renal parenchymal echo patterns and no strong echo in the renal pelvis and calyces; (ii) renal pelvis and calyx stones, performed for one or more strong echoes in the renal pelvis and calyx; and (iii) vertebral body echo enhancement, shown as the renal cones were more echogenic than the renal cortex (Supplementary Fig. S1A and B, available at *Rheumatology Advances in Practice* online). Pulsed Doppler was used to measure the blood flow velocity in both renal arteries. The participants were instructed to hold their breath and obtain a regular and non-noisy arterial blood flow spectrum of at least three cardiac cycles. The parameters of each artery were measured three times and the average value was used as the blood flow parameter of that artery (Supplementary Fig. S1C and D, available at *Rheumatology Advances in Practice* online). The renal interlobar ARI (RIARI) and renal segmental ARI (RSARI) were automatically calculated using an instrument software system. Finally, the mean RIARI and RSARI were calculated to represent the overall intrarenal ARI (OIARI). RIARI, RSARI or OIARI values >0.7 were considered elevated [20].

Statistics

SPSS V26.0 (IBM Corp., Armonk, NY, USA) was used for the analysis. GraphPad Prism V7.02 (GraphPad Software Inc., USA) was utilized for plotting. Comparisons between patients with gout and HCs were performed using the Mann–Whitney U test. The receiver operating characteristic (ROC) curve and derived area under the curve (AUC) were used to evaluate the diagnostic efficiency of RIARI, RIARI or OIARI. Logistic regression analyses were used to screen for factors related to RIARI, RSARI and OIARI. Additionally, a multivariate logistic regression analysis was performed using a forward stepwise model. The nomogram is presented sequentially. Subsequently, the variables that were screened by a multivariate logistic regression analysis were combined, and ROC curves with AUC were calculated. $P < 0.05$ was indicated as significant.

Results

Clinical features

Among the study cohort of 235 gout patients, there were 10 (4.3%) females and 225 (95.7%) males, and the mean age was 46.0 ± 15.1 years. The median [interquartile range (IQR)] disease duration was 3.0 (0.7–8.0) years, and 115 (48.9%) patients had tophus. Detailed clinical features, including demographics, disease characteristics, and biochemical indices are shown in Supplementary Table S1, available at *Rheumatology Advances in Practice* online. Fifty healthy

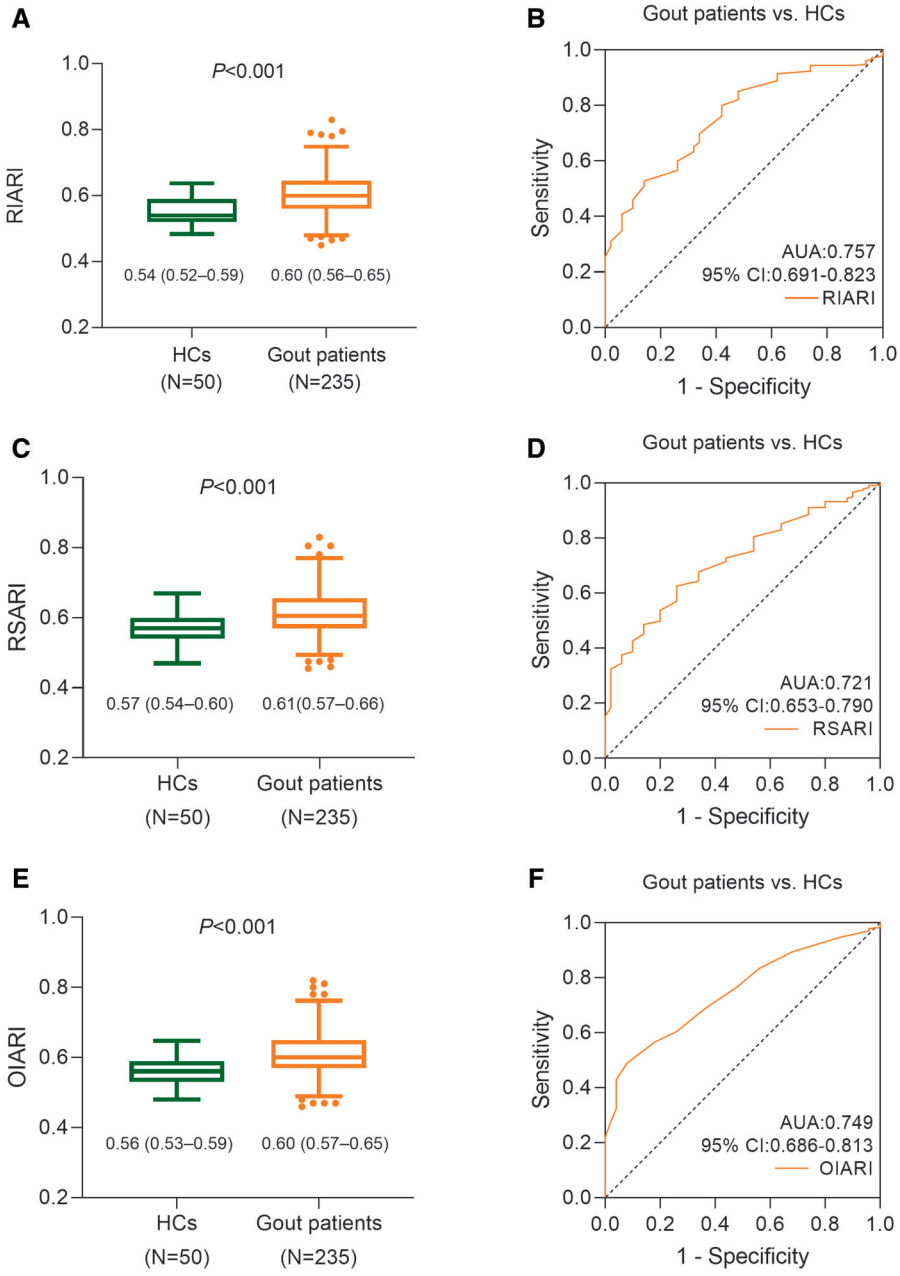

Figure 1. Comparisons between patients with gout and HCs, and ROC curve. Elevated RIARI, RSARI and OIARI in gout patients versuss HCs (A); ROC-evaluated efficiency of RIARI for identifying gout patients from HCs (B). Comparison of RSARI between gout patients and HCs (C), as well as its ability (evaluated by ROC) for differentiating gout patients from HCs (D). Comparison of OIARI between gout patients and HCs (E), and its ROC curve in distinguishing gout patients from HCs (F)

subjects were enroled as HCs, there were 4 (8.0%) females and 46 (92.0%) males, and the mean age was 38.5 ± 14.5 years, the mean BMI was 24.2 ± 2.8 kg/m^2.

Comparison of RIARI, RSARI and OIARI between gout patients and HCs

Nineteen (8.1%), 24 (10.2%) and 18 (7.7%) patients had elevated RIARI, RSARI and OIARI scores, respectively. RIARI was higher in gout patients than that in HCs [median (IQR):0.6 (0.56–0.65) *vs* 0.54 (0.52–0.59), $P < 0.001$] (Fig. 1A), which disclosed a good value on distinguishing gout patients from HCs [AUC: 0.757, 95% CI: 0.691–0.823] (Fig. 1B). Similarly, RSARI was higher in gout patients compared with HCs [median (IQR):0.61 (0.57–0.66) *vs* 0.57 (0.54–0.60), $P < 0.001$] (Fig. 1C), and it also possessed the potency to differentiate gout patients from HCs (AUC: 0.721, 95% CI: 0.653–0.790) (Fig. 1D). Additionally, OIARI was enhanced in gout patients versuss HCs [median (IQR):0.60 (0.57–0.65) *vs* 0.56 (0.53–0.59), $P < 0.001$] (Fig. 1E), with a good ability to identify gout patients from HCs (AUC: 0.749, 95% CI: 0.686–0.813) (Fig. 1F).

Univariate logistic regression analyses for elevated RIARI, RSARI and OIARI in gout patients

The percentages of gout patients with abnormal biochemical indices are listed in Supplementary Table S2, available at *Rheumatology Advances in Practice* online. Age ≥60 years ($P = 0.000$), hypertension ($P = 0.026$), tophus ($P = 0.011$), abnormal haemoglobin (HGB) ($P = 0.014$), abnormal red blood cells (RBC) ($P = 0.002$), abnormal lymphocytes (LY) ($P = 0.017$), abnormal eosinophils (EO) ($P = 0.004$), abnormal total protein (TP) ($P = 0.032$), abnormal albumin (ALB) ($P = 0.008$), abnormal urea ($P = 0.001$), abnormal CysC ($P = 0.001$), abnormal beta2 microglobulin (β2MG) ($P = 0.000$), abnormal high-density lipoprotein cholesterol (HDLC) ($P = 0.047$), abnormal glycated haemoglobin (HbA1c) ($P = 0.021$), abnormal anhydroglucitol (AG) ($P = 0.015$), abnormal CRP ($P = 0.009$) and abnormal ESR ($P = 0.009$) were related to a higher risk of elevated RIARI in gout patients. In contrast, male sex ($P = 0.019$) were associated with a lower risk of elevated RIARI in patients with gout (Supplementary Table S3, available at *Rheumatology Advances in Practice* online).

Age ≥60 years ($P = 0.000$), hypertension ($P = 0.002$), diabetes mellitus ($P = 0.019$), tophus ($P = 0.010$), abnormal HGB ($P = 0.014$), abnormal RBC ($P = 0.001$), abnormal LY ($P = 0.012$), abnormal EO ($P = 0.003$), abnormal TP ($P = 0.000$), abnormal ALB ($P = 0.000$), abnormal urea ($P = 0.000$), abnormal creatinine (CREA) ($P = 0.047$), abnormal CysC ($P = 0.000$), abnormal β2MG ($P = 0.000$), abnormal lipoprotein(a) (Lp(a)) ($P = 0.038$), abnormal fasting plasma glucose (FPG) ($P = 0.040$), abnormal HbA1c ($P = 0.008$), abnormal CRP ($P = 0.010$), abnormal AG ($P = 0.006$) and abnormal ESR ($P = 0.005$) were correlated with higher risk of elevated RSARI, whereas abnormal low-density lipoprotein cholesterol (LDLC) ($P = 0.023$) and abnormal total cholesterol (TC) ($P = 0.023$) were linked with lower risk of elevated RSARI in gout patients (Supplementary Table S3, available at *Rheumatology Advances in Practice* online).

Age ≥60 years ($P = 0.000$), hypertension ($P = 0.003$), diabetes mellitus ($P = 0.013$), tophus ($P = 0.048$), abnormal HGB ($P = 0.008$), abnormal RBC ($P = 0.000$), abnormal LY ($P = 0.027$), abnormal EO ($P = 0.011$), abnormal TP ($P = 0.000$), abnormal ALB ($P = 0.000$), abnormal urea ($P = 0.001$), abnormal CREA ($P = 0.048$), abnormal CysC ($P = 0.001$), abnormal β2MG ($P = 0.000$), abnormal HbA1c ($P = 0.025$), abnormal AG ($P = 0.019$), abnormal CRP ($P = 0.016$) and abnormal ESR ($P = 0.027$) were related to a higher risk of elevated OIARI; however, male ($P = 0.015$) were associated with a lower risk of elevated OIARI in gout patients (Supplementary Table S3, available at *Rheumatology Advances in Practice* online).

Multivariate logistic regression analyses for elevated RIARI, RSARI and OIARI in gout patients

After adjustment by multivariate logistic regression analysis, age ≥60 years [odds ratio (OR)=58.700, $P = 0.000$], abnormal β_2MG levels (OR = 7.526, $P = 0.028$) and abnormal HDLC levels (OR = 5.905, $P = 0.030$) were independently associated with a higher risk of elevated RIARI in patients with gout. Age ≥60 years (OR = 71.711, $P = 0.000$) and abnormal β_2MG (OR = 6.904, $P = 0.013$) were independently linked with higher risk of elevated RSARI in gout patients. Abnormal TP (OR = 8.400, $P = 0.014$) was independently related to a higher risk of elevated OIARI in gout patients (Table 1).

Prediction models for elevated RIARI, RSARI and OIARI risk in gout patients

Subsequently, independent risk factors were utilized to develop predictive nomograms for elevated RIARI (Fig. 2A), RSARI (Fig. 2B) and OIARI (Fig. 2C) in patients with gout. Moreover, the combination of age ≥60 years, abnormal β_2MG and abnormal HDLC levels showed a pleasing ability to estimate the risk of elevated RIARI (AUC, 0.929; 95% CI: 0.884–0.974) (Fig. 3A). The combination of age ≥60 years and abnormal β_2MG was satisfactory for predicting the risk of elevated RSARI levels (AUC: 0.926, 95% CI: 0.863–0.988) (Fig. 3B). In addition, TP exhibited a good capability for predicting the risk of elevated OIARI in patients with gout (AUC: 0.660, 95% CI: 0.506–0.815) (Fig. 3C).

Discussion

Gout is commonly accompanied by kidney dysfunction, and nearly 25% of patients with gout have chronic kidney disease; consequently, their renal function requires timely monitoring with the help of renal ARI [21–23]. The current study showed that 19 (8.1%), 24 (10.2%) and 18 (7.7%) patients

Table 1. Multivariate logistic regression analysis for elevated RIARI, elevated RSARI and elevated OIARI

Items	*P*-value	OR (95% CI)
Elevated RIARI		
Age (≥60 years *vs* <60 years)	**0.000**	58.700 (6.325–544.768)
β_2MG (abnormal *vs* normal)	**0.028**	7.526 (1.238–45.761)
HDLC(abnormal *vs* normal)	**0.030**	5.905 (1.183–29.486)
Elevated RSARI		
Age (≥60 years *vs* <60 years)	**0.000**	71.711 (8.571–599.964)
β_2MG (abnormal *vs* normal)	**0.013**	6.904 (1.493–31.927)
Elevated OIARI		
TP (abnormal *vs* normal)	**0.014**	8.400 (1.543–45.737)

Bolded font indicated *P*-value less than 0.05.
RIARI: renal interlobar arteries resistance index; RSARI; renal segmental arteries resistance index; OIARI: overall intrarenal arteries resistance index; OR: odds ratio; CI: confidence interval; HDLC: high-density lipoprotein cholesterol; β_2MG: beta2 microglobulin; TP: total protein.

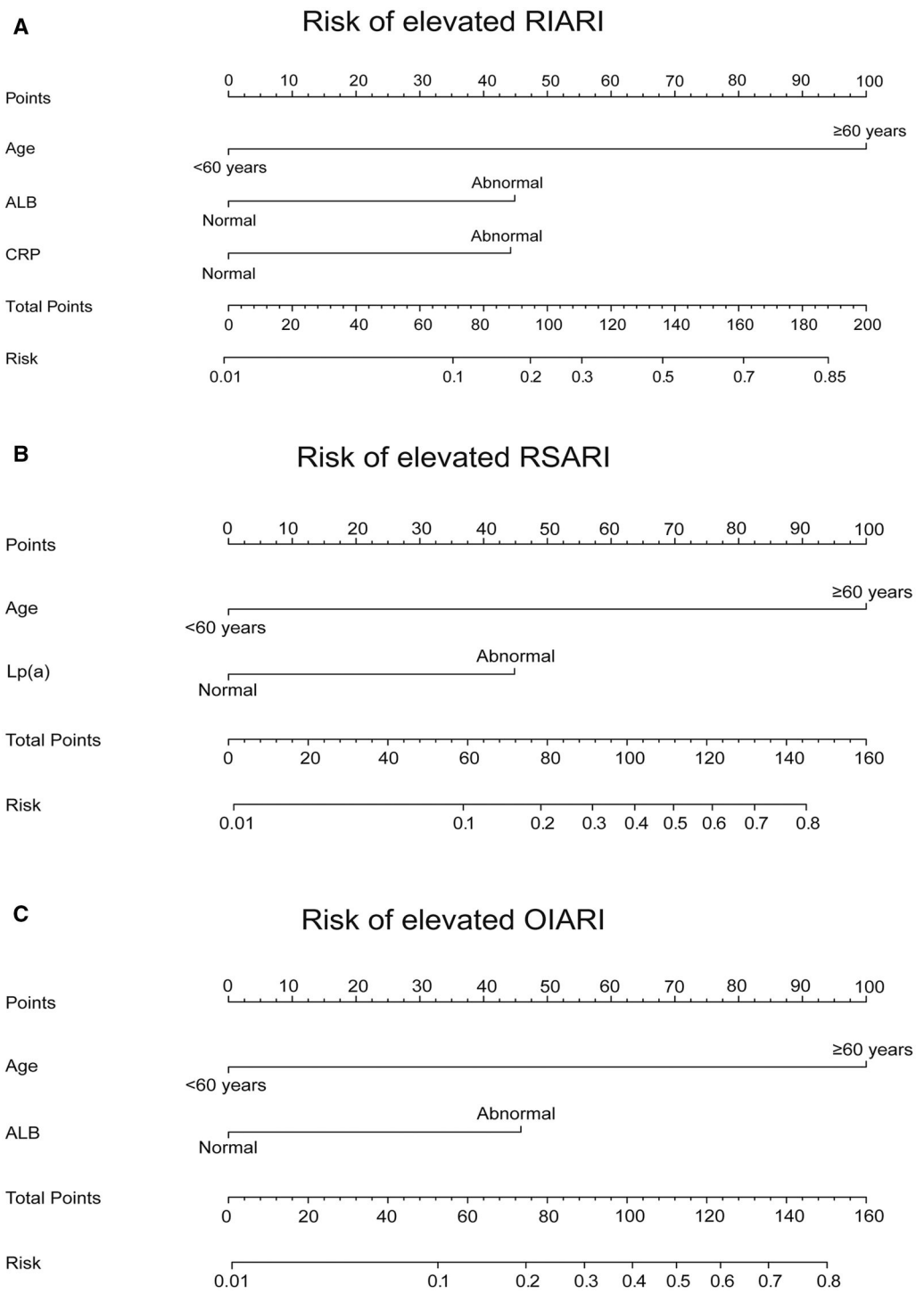

Figure 2. Nomograms. Nomogram for predicting the risk of elevated RIARI (A), RSARI (B) and OIARI (C) in gout patients

had elevated RIARI, RSARI and OIARI, respectively; meanwhile, RIARI, RSARI and OIARI were higher in gout patients compared with HCs. The probable explanation is that as a main manifestation of gout, hyperuricemia influences renal arterial tone and facilitates renal arterial damage [24]. Thus, the RIARI, RSARI and OIARI were higher in patients with gout than in HCs.

Considering the risk factors for elevated renal ARI, previous studies have shown that elevated age independently estimates the risk of elevated renal ARI [17, 25]. For example, one study discovered that an increased age of renal allograft recipients is independently related to elevated renal ARI [17]. Another study reported that renal ARI increments were independently predicted by age in systemic sclerosis patients [25]. Similarly, the present study found that age ≥60 years was an independent risk factor for elevated RIARI and RSARI scores in patients with gout. A possible reason is that the elderly population suffers from a declining glomerular filtration rate, leading to structural and functional changes in the kidneys, including renal hemodynamic disturbances [26]. As a result, age ≥60 years was independently correlated with higher risk of elevated RIARI, RSARI in gout patients. β2MG is a low-

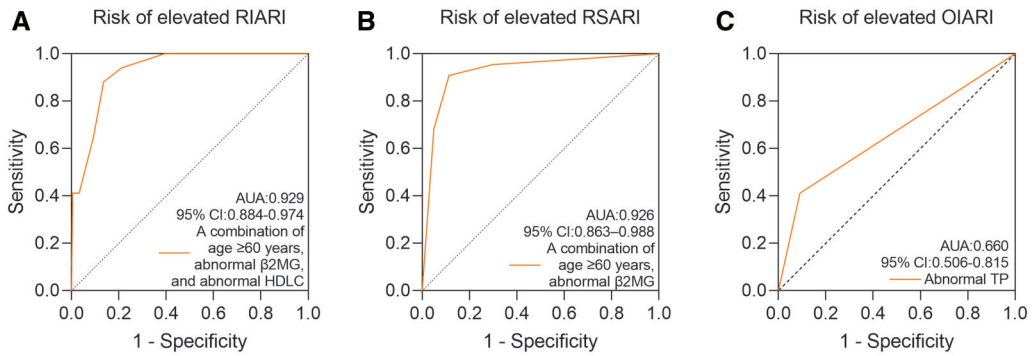

Figure 3. ROC curves. Value of the predictive models for elevated RIARI (A), RSARI (B) and OIARI (C) risk in gout patients

molecular-weightprotein, primarily released from immune-related cells [27]. One study discovered plasma levels of β2MG are related to atherosclerosis [28]. This study showed that abnormal β2MG levels were independently correlated with elevated RIARI and RSARI in patients with gout, which could be explained by the fact that β2MG is implicated in immunological mechanisms, β2MG levels are associated with renal involvement and overall clinical disease activity [29]. Hence, abnormal β2MG level is an independent risk factor for elevated RIARI and RSARI levels in patients with gout.

In addition to age and abnormal β2MG, this study also found that abnormal TP levels were independently related to elevated OIARI, and abnormal HDLC levels were an independent risk factor for elevated RIARI in patients with gout. The probable reasons are as follows: (i) Inflammation induced pathological changes in both the kidney and blood vessels and promoted the progression of atherosclerotic lesions, which further caused intrarenal artery stenosis and disturbance of blood circulation [30, 31]. Consequently, an abnormal TP level is an independent risk factor for an elevated OIARI in patients with gout. (ii) HDLC possesses beneficial effect in the retardation of atherosclerotic process by removing excessive cellular cholesterol to the liver by reverse cholesterol ransport, HDLC can improve endothelial function in renal vascular disease, this could be helpful in attenuating further vascular damage [32, 33]. Thus, abnormal HDLC levels are independently associated with elevated RIARI levels in patients with gout. Importantly, the combination of the aforementioned independent risk factors displayed satisfactory effectiveness in predicting elevated RIARI, RSARI and OIARI risks in patients with gout. The findings indicate the potential clinical utility of the predictive models, which might assist clinicians in identifying patients with gout with an increased risk of renal hemodynamic disturbances; thus, they could provide timely intervention.

Furthermore, we found that having hypertension was a risk factor for increased ARI in patients with gout. Previous studies have shown that ARI was significantly associated with large artery stiffness, central pulse pressure and left ventricular systolic function [34, 35]. The ARI in hypertension patients will gradually increase with the deterioration of renal function [36]. This study showed that abnormal CREA levels were a risk factor for increased RSARI and OIARI, abnormal CysC levels are a risk factor for increased RIARI, RSARI and OIARI. And CysC has a higher OR value compared with CREA. It has been shown that CysC level is a risk factor for increased echo of the renal medulla in patients with gout, and CREA level is a risk factor for thinning of the renal cortex in patients with gout [37]. Clinical assessment of glomerular filtration rate mainly relies on determinations of CREA and CysC, CREA is commonly insensitive to mild renal dysfunction, but the glomerular filtration rate reduction can be more reliably and earlier detected by CysC [38]. These may be related to CysC having higher risk factors for increased ARI.

Several limitations of this study were as follows: (i) the efficiency of the predictive models needed further external validation to evaluate its extrapolation applicability; (ii) the ultrasound imaging information of gout patients was dynamic; however, this study only collected the imaging information at enrolment and did not include long-term data; (iii) renal artery ultrasound examination reflected both renal hemodynamics and renal artery stenosis; this study mainly focused on the former aspect (renal hemodynamics) in gout patients, and the risk factors of renal artery stenosis in gout patients require further investigation.

In conclusion, renal ARI is increased in around 9% of patients with gout, and is independently related to age, β_2MG, HDLC and TP, and predictive models combining these factors show favourable values for identifying the risk of elevated renal ARI in these patients; however, further external validation is required.

Supplementary material

Supplementary material is available at *Rheumatology Advances in Practice* online.

Data availability

Data are available upon reasonable request to the corresponding author. All data relevant to this study are included in the article.

Contribution statement

W.D. and J.L. conceived and designed the work. H.L. and J.H. contributed to analyses of imaging data, prepared the figures, and provided technical support. H.L. and J.H. contributed to data collection and interpretation of data. W.D. and H.L. drafted the manuscript. W.D. and J.L. critically revised the manuscript for important intellectual content. All authors read and approved the final manuscript.

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