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Associations of body mass index and remnant cholesterol with hyperuricemia in patients with hypertension

Wei Zhou[1,2,3,4], Tao Wang[1,2,3,4], Lingjuan Zhu[1,2,3,4], Yumeng Shi[1*], Chao Yu[1,2,3,4*], Huihui Bao[1,2,3,4] and Xiaoshu Cheng[1,2,3,4]

Abstract

Background There is a paucity of prior research on residual cholesterol (RC) and hyperuricemia, and it remains unclear whether body mass index (BMI) functions as a mediating factor between them or intensifies lipid metabolic dysregulation, thereby elevating the risk of hyperuricemia. This study aims to investigate whether BMI mediates the association between RC and hyperuricemia, as well as the interaction or joint effect of BMI and RC on hyperuricemia.

Methods This is a cross-sectional study, involving a total of 14,218 hypertensive patients. Exposure factors include RC and BMI. The outcome was Hyperuricemia, defined as serum uric acid (SUA) ≥ 420 μmol/L. Multivariable logistic regression models and causal mediation analysis were used to examine the association between RC and BMI and the prevalence of hyperuricemia.

Results A total of 14,218 hypertensive patients were enrolled in this cross-sectional study, comprising 6,713 (47.2%) males, with a mean age of 63.8 (9.36) years. The prevalence of diabetes mellitus was found to be 10.4% (1,473), while hyperuricemia accounted for approximately 44.4% (6,319). The results show that there is a linear positive correlation between RC and hyperuricemia (P for trend < 0.01). RC and BMI only had significant additive interaction on hyperuricemia, but there was no multiplicative interaction (Additive: RERI = 0.45, 95%CI: 0.13–0.78; Multiplicative, OR = 1.09, 95% CI 0.92–1.3, *P* = 0.308). There are direct and indirect effects between RC and hyperuricemia [estimate (95% CI): DE = 0.063 (0.048, 0.070), IE = 0.005 (0.003, 0.001)]. In the aforementioned causal mediation analysis, among the hyperuricemia caused by RC, BMI mediates 7.1%.

Conclusion The intermediary role of BMI and its interaction with RC play a pivotal role in augmenting the prevalence of hyperuricemia.

Trial registration Registered prospectively in the Chinese Clinical Trial Registry (ChiCTR1800017274) on July 20, 2018. Access at https://www.chictr.org.cn/showproj.html?proj=28262.

Keywords Remnant cholesterol, Hyperuricemia, Hypertension, Body mass index, Lipid metabolism

*Correspondence:
Yumeng Shi
shiyumeng666@126.com
Chao Yu
yuchao9@gmail.com

Full list of author information is available at the end of the article

Introduction

As a new risk factor for cardiovascular events, remnant cholesterol (RC) has attracted the attention of many scholars. In the past, we advocated intensive lipid-lowering therapy to reduce the level of low-density lipoprotein (LDL-C) as much as possible in order to prevent the occurrence and development of cardiovascular diseases (CVD) [1, 2]. However, when we reduce LDL-C to the level recommended by the guidelines, there is still a considerable risk of residual atherosclerotic cardiovascular events (CVEs) [3]. Furthermore, a plethora of studies have consistently demonstrated that an elevated level of RC is strongly associated with a substantial increase in the incidence of cardiovascular events [4, 5, 6, 7]. Therefore, it is imperative to incorporate RC into the blood lipid spectrum as a pivotal component in the prevention and management of CVD. Currently, the accumulation of other metabolic risk factors, such as obesity and dyslipidemia, contributes to the increased incidence of cardiovascular diseases. Therefore, researchers have proposed the concept of metabolic cardiovascular diseases as an intermediary link between metabolic disorders and future cardiovascular events in order to effectively manage these conditions and reduce the associated risks [8]. In addition, the metabolic index concerning serum uric acid level has garnered significant attention from researchers. Previously, it was believed that the collective influence of elevated serum uric acid levels, age, gender, hypertension, hypertriglyceridemia, and insulin resistance contributed to the development of metabolic syndrome and ultimately served as a risk factor for CVD [9].However, recent studies have revealed that hyperuricemia constitutes an autonomous risk factor for hypertension and cardiovascular diseases, including ischemic heart disease and heart failure [10].Therefore, hyperuricemia should be regarded as a pivotal intermediary event warranting meticulous attention, as it not only exerts regulatory influence on metabolic alterations in vivo but also independently precipitates cardiovascular events.

Previous studies have demonstrated a significant association between serum uric acid levels and lipid metabolism, suggesting a potential link with lipids implicated in the development of atherosclerosis [11, 12, 13]. RC, in addition to LDL-C, is considered a residual risk factor that may contribute to increased cardiovascular events and could potentially be associated with serum uric acid levels. However, at present, reports on RC and hyperuricemia are limited. Moreover, the study was carried out among American adults. Wang et al. [14]. conducted a cross - sectional study on RC and hyperuricemia in 14,568 American adults. The results showed that elevated RC was positively correlated with an increased risk of hyperuricemia. In addition, obesity also affects lipid metabolism. Furthermore, multiple epidemiological studies have indicated that obesity significantly increases the prevalence of hyperuricemia [15, 16, 17].

Therefore, the objective of this study is to assess the association between RC and hyperuricemia in hypertensive patients, while additionally investigating the mediating and interactive effects of RC combined with BMI level on hyperuricemia.

Methods

Study population

This cross-sectional study was based on the China H-type Hypertension Registry Study (ChiCTR1800017274), which was conducted in China from March 2018 to August 2018. This real-world, observational study was designed to enroll and monitor cohorts of the hypertensive population with a high prevalence of hyperhomocysteinemia in China. The design and methods of the trial have been described in detail elsewhere [18]. The inclusion criteria of this study are hypertension patients over 18 years old. Hypertension is defined as the screening period of systolic blood pressure (SBP) ≥ 140mmHg and diastolic blood pressure (DBP) ≥ 90mmHg after resting for 10 min or taking hypertension drugs within two weeks. Exclusion criteria include the abnormal nervous system, inability to follow up according to the research plan or planned short-term resettlement, and patients not suitable for inclusion or long-term follow-up as assessed by the research doctor. All participants signed informed consent, with the anonymity of the information obtained being assured. The study protocol was approved by the ethics committee of the institute of Second Affiliated Hospital of Nanchang University.

A total of 14,234 hypertensive patients met the above criteria. After excluding 16 participants due to missing data, this cross-sectional analysis finally included 14,218 hypertensive patients with complete data. See the flow chart of this study for details (Fig. 1).

Data collection

All participants were required to complete a structured modified screening questionnaire to determine their demographic characteristics, including age, sex, smoking history, drinking history, medical history, and medications. Anthropometric indices included body height, weight. Body mass index (BMI) was defined as body weight / height ^{2} (kg/m^2). Training medical staff assessed blood pressure (BP) to limit the interobserver variability in measurement. After the participants had rested for 5 min, seated BP was measured using an electronic sphygmomanometer (Omron; Dalian, China), following the standard method and appropriately sized cuffs. Three measurements on the right arm were performed with one-minute intervals between successive readings, and the mean value was calculated. Alcohol consumption

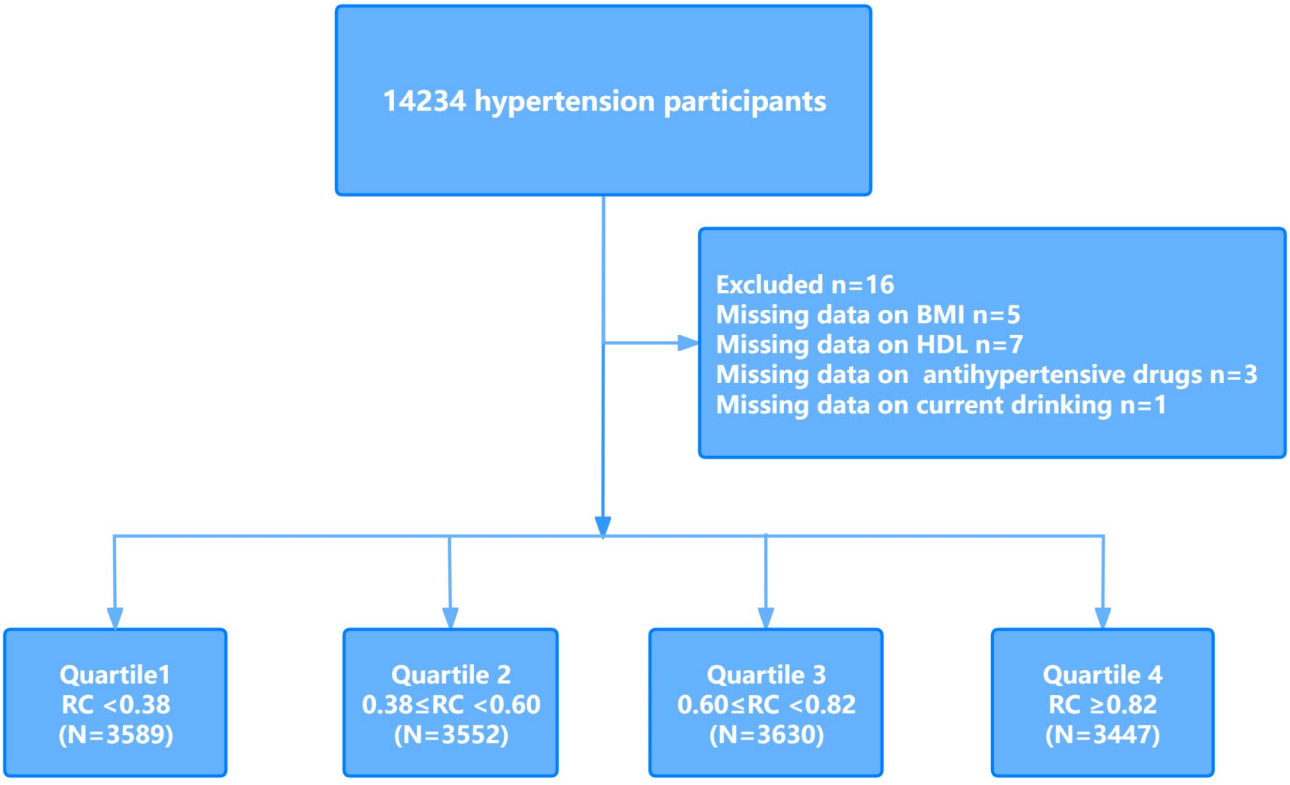

Fig. 1 Flow chart of research population

was defined as drinking an average of two or more times per week over a year. There are two types of drinking: occasional and regular. Occasional drinking was defined as drinking alcohol monthly or less, and regular drinking was defined as drinking alcohol at least twice a month. Current smoking was defined as smoking ≥ 1 cigarette per day for one year or more or a cumulative smoking amount ≥ of 360 cigarettes per year.

Blood samples were collected after fasting for 8–12 h. Those samples were quickly processed to obtain serum and stored at -80℃ until they were sent to Biaojia Biotechnology in Shenzhen in Guangdong Province, China, for analysis. Furthermore, automatic clinical analyzers (Beckman Coulter, USA) were used to measure all biochemical parameters, which included fasting plasma glucose (FPG), homocysteine (Hcy), triglycerides (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and serum uric acid (SUA). The estimated glomerular filtration rate (eGFR) was estimated according to CKD-EPI equation [19]. Diabetes mellitus was defined as self-reported physician diagnosis of diabetes or FBG concentration ≥ 7.0 mmol/L or use of glucose-lowering drugs.

Definition of the RC, BMI and hyperuricemia

RC is calculated by total cholesterol, high density lipoprotein and low density lipoprotein, and the specific formula is as follows: RC = TC-(HDL-C)-(LDL-C) [2]. According to China's BMI classification, the normal BMI is 18.5–24 kg/m^2, and the overweight BMI is 24–28 kg/m^2 [20]. Thus, in this cross-sectional analysis, we take 24 as the BMI tangent point. As the average age of the population in this study is 64 years old and women are basically in menopause, the definition of hyperuricemia is SUA ≥ 420 μmol/L [21, 22].

Statistical analysis

A two-tailed $P < 0.05$ was regarded as statistically significant. The statistical packages R (http://www.r-project.org) and Empower (R) (www.empowerstats.com) were used to perform all statistical analyses.

Baseline characteristics are presented as means (SDs) or medians (interquartile ranges) (IQRs) for continuous variables and proportions for categorical variables according to RC quartiles. ANOVA, the Mann-Whitney test of nonparametric or Chi-squared tests were used to compare the significant differences in population characteristics. Multivariable logistic regression models were used to examine the association between RC and BMI and the prevalence of hyperuricemia. There are three models: model 1was crude model; model 2 was adjusted

for age, BMI, SBP, DBP; model 3 was further adjusted for diabetes mellitus, antihypertensive drugs, lipoprotein-lowering drugs, glucose-lowering drugs, current smoking, current drinking, Hcy, FPG, eGFR based on model 2.

Utilizing the "mediation" R package, we conducted a causal mediation analysis to examine the mediating role of BMI in the association between RC and the prevalence of hyperuricemia. In order to quantify the interaction of addition and multiplication, we added the product term of BMI (< 24.0 kg/m^2, ≥ 24.0 kg/m^2) and RC (< 0.82, ≥ 0.82) to the model. The OR with its 95% CI of the product term was the measure of interaction on the multiplicative scale. Furthermore, relative excess risk due to interaction (RERI), proportion attributable to interaction (AP), and synergy index (SI) are used to evaluate the effect of additive interaction. We computed the corresponding 95% CIs for these three metrics using the delta method. To evaluate the combined effect of two exposure variables, participants were divided into four groups according to their BMI (< 24.0 kg/m^2,≥24.0 kg/m^2) and RC (< 0.82,≥0.82). Taking the study population with BMI < 24 and RC < 0.82 as the reference group, we calculated the OR value of the prevalence of hyperuricemia in the other three groups.

Results

Characteristics of participants

A total of 14,218 hypertensive patients were enrolled in this cross-sectional study, comprising 6,713 (47.2%) males, with a mean age of 63.8 (9.36) years. The prevalence of diabetes mellitus was found to be 10.4% (1,473), while hyperuricemia accounted for approximately 44.4% (6,319). The baseline characteristics of the research population according to the RC quartile grouping are presented in Table 1. Patients in the highest Q4 group (RC ≥ 0.82) exhibit a lower proportion of males and tend to be younger in age. Compared to participants in Q1, those in Q4 demonstrate higher values for BMI, SBP, DBP, FPG, TC, TG, LDL-C and serum uric acid levels as well as a higher prevalence of diabetes mellitus and rate of glucose-lowering drugs usage. Currently, there is a lower rate of smoking and alcohol consumption among these individuals along with lower Hcy levels, HDL levels, eGFR values and rates of antihypertensive drug usage and lipid-lowering drug intake.

Association of RC and BMI with hyperuricemia

When RC levels were examined as a continuous variable in model 3, for one unit increase in the RC adjusted odds ratio (OR) for hyperuricemia were 1.40 (95% CI:1.29, 1.52). Consistently, when RC was assessed as quartiles, compared with participants in quartiles 1 (<0.38), the

Table 1 Baseline characteristics of the study population according to RC quartile

Variable[a]	All	RC				*P* value
		Q1 (< 0.38)	Q2 (0.38–0.60)	Q3 (0.60–0.82)	Q4 (≥ 0.82)	
Participants, n	14,218	3589	3552	3630	3447	
Male, n (%)	6713 (47.2%)	1888 (52.6%)	1846 (52.0%)	1635 (45.0%)	1344 (39.0%)	<0.001
Age, year	63.8 ± 9.36	64.0 ± 9.06	63.7 ± 9.52	64.1 ± 9.31	63.4 ± 9.55	0.013
BMI, kg/m^2	23.6 ± 3.74	23.4 ± 4.40	23.4 ± 3.50	23.7 ± 3.47	24.0 ± 3.48	<0.001
SBP, mmHg	148 ± 17.9	149 ± 17.8	147 ± 17.8	148 (17.6)	149 ± 18.1	<0.001
DBP, mmHg	88.9 ± 10.8	88.6 ± 10.6	88.8 ± 10.6	88.9 (10.8)	89.5 ± 11.0	0.003
Current smoking	3659 (25.7%)	1025 (28.6%)	951 (26.8%)	892 (24.6%)	791 (22.9%)	<0.001
Current drinking	3063 (21.5%)	879 (24.5%)	758 (21.3%)	737 (20.3%)	689 (20.0%)	<0.001
diabetes mellitus[$]	1473 (10.4%)	381 (10.6%)	333 (9.38%)	358 (9.86%)	401 (11.6%)	0.012
Antihypertensive drugs	9220 (64.8%)	2473 (68.9%)	2293 (64.6%)	2258 (62.2%)	2196 (63.7%)	<0.001
Glucose-lowering drugs	754 (5.30%)	184 (5.13%)	165 (4.65%)	194 (5.34%)	211 (6.12%)	0.048
Lipoprotein-lowering drugs	546 (3.84%)	161 (4.49%)	165 (4.65%)	123 (3.39%)	97 (2.81%)	0.001
Hcy,μmol/L	15.0 [12.5–19.1]	15.4 [12.7–19.6]	14.8 [12.4–19.1]	14.8 [12.4–19.0]	15.0 [12.5–18.7]	<0.001
FPG, mmol/L	6.18 ± 1.61	6.02 ± 1.41	5.99 ± 1.33	6.17 ± 1.58	6.57 ± 1.99	<0.001
TC, mmol/L	5.16 ± 1.12	4.64 ± 1.10	4.80 ± 0.93	5.24 ± 0.87	5.97 ± 1.06	<0.001
TG, mmol/L	1.81 ± 1.26	1.28 ± 0.68	1.50 ± 0.82	1.83 ± 1.02	2.65 ± 1.80	<0.001
HDL-C, mmol/L	1.57 ± 0.43	1.72 ± 0.47	1.55 ± 0.42	1.51 ± 0.39	1.48 ± 0.38	<0.001
serum uric acid, μmol/L	419 ± 121	399 ± 114	414 ± 117	421 ± 123	443 ± 124	<0.001
LDL-C, mmol/L	2.98 ± 0.81	2.83 ± 0.85	2.75 ± 0.72	3.03 ± 0.69	3.33 ± 0.86	<0.001
eGFR, mL/min/1.73 m^2	88.2 ± 20.2	90.9 ± 21.1	88.6 ± 19.7	87.2 ± 19.4	85.9 ± 20.3	<0.001

[a]:Continuous variables: mean ± standard deviation for normal distribution, median [Quartiles1-Quartiles3]; Categorical variables: number (percentage)

Abbreviation: RC, remnant cholesterol; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; Hcy, homocysteine; FPG: fasting plasma glucose; TC, total cholesterol; TG, triglycerides; HDL, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; eGFR, estimated glomerular filtration rate

Table 2 Association between RC and hyperuricemia in different models

Variable	Event (%)	hyperuricemia, OR (95%CI)		
		Model 1	Model 2	Model 3
RC				
Per 1 mmol/L increse	6319 (44.4%)	1.47 (1.36, 1.58)	1.44 (1.34, 1.55)	1.40 (1.29, 1.52)
Quartiles				
Q1 (<0.38)	1360 (37.9%)	Reference	Reference	Reference
Q2 (0.38 to < 0.60)	1522 (42.8%)	1.23 (1.12, 1.35)	1.20 (1.09, 1.32)	1.17 (1.05, 1.31)
Q3 (0.60 to < 0.82)	1644 (45.3%)	1.36 (1.24, 1.49)	1.30 (1.18, 1.43)	1.25 (1.13, 1.39)
Q4 (≥ 0.82)	1793 (52.0%)	1.78 (1.62, 1.95)	1.73 (1.57, 1.9)	1.60 (1.43, 1.78)
P for trend		< 0.001	< 0.001	< 0.001
BMI, kg/m^2	6319 (44.4%)			
< 24	3187 (39.9%)	Reference	Reference	Reference
≥ 24	3132 (50.3%)	1.52 (1.43, 1.63)	1.33 (1.19, 1.48)	1.28 (1.14, 1.45)

Model 1: crude model

Model 2: adjusted for age, BMI, SBP, DBP

Model 3: adjusted for age, BMI, SBP, DBP, diabetes mellitus, antihypertensive drugs, lipoprotein-lowering drugs, glucose-lowering drugs, current smoking, current drinking, Hcy, FPG, eGFR

Table 3 Interactive effects of remnant cholesterol and body mass index on hyperuricemia

Interactive items	Interactive effects (95% CI)*, *P* value
Additive effects	
RERI	0.45 (0.13, 0.78), *P* < 0.0001
AP	0.18 (0.05, 0.29), *P* < 0.0001
SI	1.44 (1.11, 1.86), *P* < 0.0001
Multiplicative scale	1.09 (0.92, 1.3), *P* = 0.308

AP, proportion attributable to interaction; CI, confdence interval; RERI, relative excess risk due to interaction; SI, synergy index

*Model was adjusted for age, BMI, SBP, DBP, diabetes mellitus, antihypertensive drugs, lipoprotein-lowering drugs, glucose-lowering drugs, current smoking, current drinking, Hcy, FPG, eGFR

adjusted ORs (95% CI) for hyperuricemia in quartile 2 (0.38 to < 0.60), quartile 3 (0.60 to < 0.82), and quartile 4 (≥ 0.82) were1.17 (1.05, 1.31), 1.25 (1.13, 1.39), 1.60 (1.43, 1.78), respectively (Table 2). The results show that there is a linear positive correlation between RC and hyperuricemia (*P* for trend < 0.01). Likewise, in the fully adjusted model 3, we observed a significant increase in the prevalence of hyperuricemia (OR = 1.28, 95% CI 1.14–1.45) among participants with BMI ≥ 24 compared to those with BMI < 24 kg/m^2.

Interaction and joint associations of RC and BMI on hyperuricemia

Through the multivariate logistic regression analysis, we can know that both higher RC value and higher BMI value can increase the prevalence of hyperuricemia, so we define RC ≥ 0.82 as elevated RC and BMI ≥ 24 kg/m^2 as elevated BMI. And calculate the comprehensive response of both to hyperuricemia. This study found that RC and BMI only had significant additive interaction on hyperuricemia, but there was no multiplicative interaction (Additive: RERI = 0.45, 95%CI: 0.13–0.78; Multiplicative, OR = 1.09, 95% CI 0.92–1.3) (Table 3). The joint associations of RC and BMI on hyperuricemia is shown in Fig. 2. After adjusting for confounders, compared with the participants with RC < 0.82 and BMI < 24 kg/m^2, the prevalence of hyperuricemia in participants with RC ≥ 0.82 and BMI ≥ 24 kg/m^2 increased significantly (OR = 2.48, 95% CI: 2.19–2.82).

Mediation analysis of BMI on associations of RC with hyperuricemia

As shown in Fig. 3, there are direct and indirect effects between RC and hyperuricemia [estimate (95% CI): DE = 0.063 (0.048, 0.070), IE = 0.005 (0.003, 0.001)]. In the aforementioned causal mediation analysis, among the hyperuricemia caused by RC, BMI mediates 7.1%.

Discussion

In this cross-sectional study of a large-scale cohort of hypertensive patients, we observed an independent and positive correlation between the increase in RC and the development of hyperuricemia. Furthermore, our findings indicate that approximately 7.1% of this correlation is mediated by BMI. Additionally, when high RC and high BMI coexist, there is a significant increase in the prevalence of hyperuricemia.

Most previous studies explored the relationship between traditional lipid components and SUA [11, 23, 24, 25, 26]. Zhang et al. [23] conducted a cross-sectional study in 2482 to explore the relationship between lipid profile and uric acid. The results showed that TG, TC, LDL-C were positively correlated with uric acid, while HDL-c was negatively correlated with uric acid level. In another study, the relationship between TG and uric acid still exists after full adjustment in multiple logic model, indicating that TG is independently related to uric acid level. Interestingly, this association exists even in the normal range of serum triglyceride [27]. A retrospective

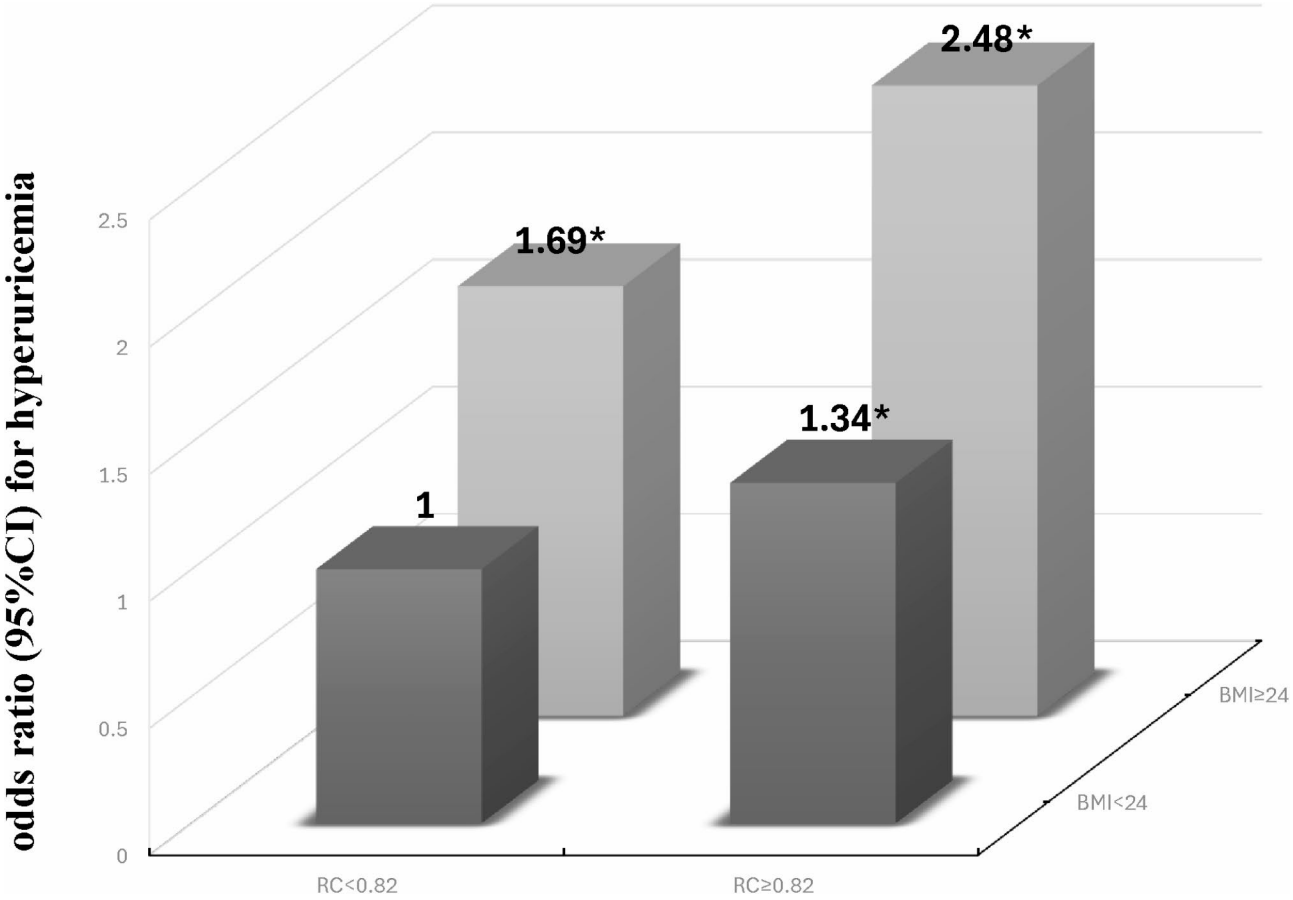

Fig. 2 Joint effects of BMI and RC on incident hyperuricemia risk among hypertensive[1]. [1]Models were adjusted for age, BMI, SBP, DBP, diabetes mellitus, antihypertensive drugs, lipoprotein-lowering drugs, glucose-lowering drugs, current smoking, current drinking, Hcy, FPG, eGFR (*$P < 0.05$)

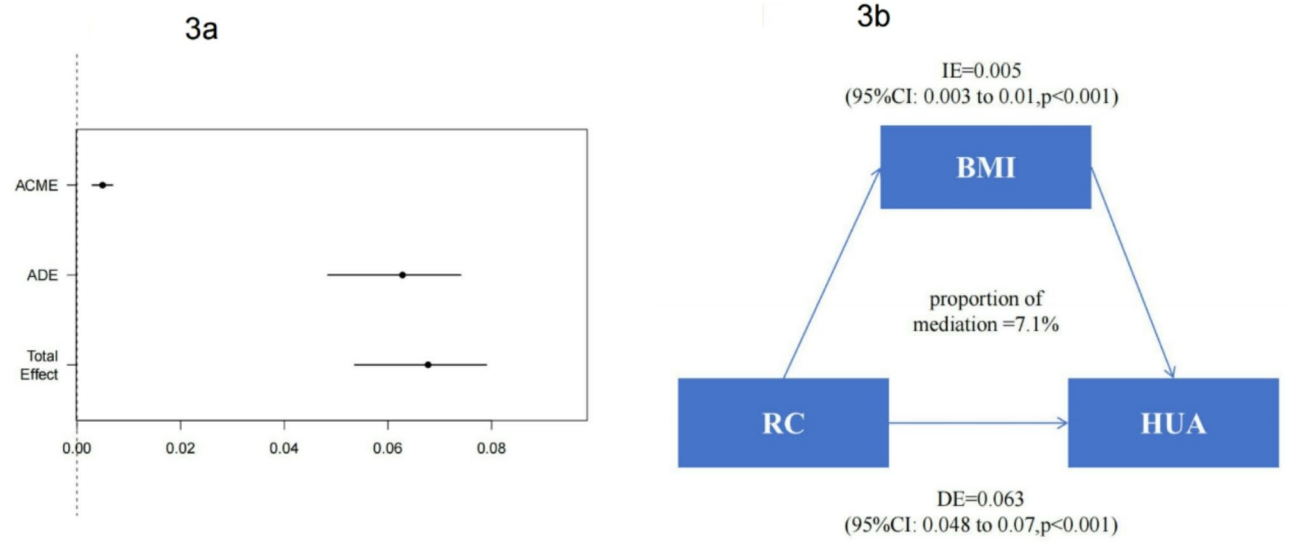

Fig. 3 Causal mediating effect of BMI on the association between RC and hyperuricemia. **(a)** Mediating effect regression (**b**) Mediated analysis directed acyclic graph. ACME stands for average causal mediation effects (indirect effects), that is, indirect effects, and ADE stands for average direct effects, that is, direct effects. Total effect is the total effect. Prop.mediated explains the percentage of correlation between RC and hyperuricemia for intermediary variables

cohort study showed that triglyceride, plasma atherogenic index (AIP), and total cholesterol /HDL-C were positively correlated with uric acid level independently in women and not in men [24]. Peng et al. [11]. explored the independent relationship between SUA and blood lipid profile in the American population by using the data of The Third National Health and Nutrition Examination Survey (NHANES III), and found that serum LDL-C, TG, TC, apolipoprotein -B level, TG/HDL, ratio of apolipoprotein -B to AI were closely related to SUA level. In contrast, HDL-C level was significantly negatively correlated with SUA level. Most of the above studies explored the relationship between different blood lipid profiles and SUA, but no study evaluated the relationship between RC and SUA.

This cross-sectional study addresses the existing gaps in the emerging field of blood lipid indicators and hyperuricemia, providing a novel perspective on metabolic diseases. Firstly, we observed a significant association between the increase in RC and the development of hyperuricemia in hypertensive patients. This finding suggests that RC, similar to traditional blood lipids, may also contribute to metabolic disorders such as hyperuricemia. Secondly, our further analysis indicates that the potential biological mechanisms by which RC causes hyperuricemia in cross-sectional studies are partly attributed to an increase in BMI. Obesity is widely recognized as a significant risk factor for hyperuricemia, with an excessive BMI being associated with elevated serum uric acid levels. This relationship may be attributed to obesity-induced increased uric acid production or impaired uric acid excretion. Additionally, a synergistic effect of both RC and BMI was observed. In the presence of elevated RC, overweight and obese individuals exhibited a significantly higher prevalence of hyperuricemia compared to normal weight individuals without elevated RC.

The mechanism of RC increasing the prevalence of hyperuricemia is still unclear, and the main explanation may come from the following points: as we all know, RC is cholesterol-rich in TG, so it is the core particle of TG [28]. Lipoprotein lipase (LPL)-mediated RC lipolysis products promote atherosclerosis through pro-inflammatory, pro-coagulation, and pro-apoptosis genes [29]. Importantly, RC (also known as residual cholesterol), because of its small size, is directly swallowed by macrophages to form foam cells, while LDL-C will become atherosclerosis after being oxidized [30, 31, 32]. The increase of RC level in the body leads to renal arteriosclerosis, which leads to the impairment of renal function, the reduction of SUA excretion, and the accumulation of SUA in the body, which leads to hyperuricemia. The experiment proves that SUA can increase TG accumulation in cultured hepatocytes [33, 34]. Therefore, the further increase of SUA leads to the increase of TG, which further increases the risk of hyperuricemia. As we all know, abnormal lipid metabolism is closely related to non-alcoholic fatty liver disease (NAFLD). Free fatty acid are used as substrates to increase very low-density lipoproteins (VLDL-C) liver production. The abnormality and impaired metabolism of TRL lead to the increase of apolipoprotein B-rich concentration in NAFLD. The subsequent lipolysis of these lipoproteins leads to an increase in the production of dense small LDL-C particles that cause atherosclerosis [35], Moreover, the risk of NAFLD will be significantly reduced after lipid-lowering treatment [36]. At the same time, related clinical studies show that NAFLD is significantly related to the increased risk of chronic kidney disease (CKD) [37]; CKD will lead to the increase of SUA level and lead to hyperuricemia. Therefore, the increase of RC level can increase the prevalence of hyperuricemia, which is based on biology.

The current study has several limitations. First, as a result of the cross-sectional design, the causal relationship between RC and the prevalence of hyperuricemia cannot be determined. Second, although we have adjusted for most relevant confounders, we cannot rule out residual or unknown confounding factors. Lastly, our study only included Chinese hypertensive patients, whether the findings can be extrapolated to other populations requires further verification.

Conclusions

In this cross-sectional analysis, we identified RC as an independent risk factor for hyperuricemia. Furthermore, our research demonstrates that BMI not only partially mediates the detrimental effects of RC on hyperuricemia but also exhibits a synergistic effect with RC, thereby contributing to an increased prevalence of hyperuricemia.

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Author contributions

Wei Zhou wrote the manuscript and contributed to data analysis and interpretation. Tao Wang, Lingjuan Zhu, Yumeng Shi and Chao Yu extracted and collected data. Huihui Bao and Xiaoshu Cheng conceived of the study and provided critical revision. All authors reviewed the manuscript.

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Data availability
Data analyzed in the current study are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate
This research was approved by the Ethics Committees of the Biomedical Institute of Anhui Medical University (CH1059). The website of Chinese Clinical Trial Registry approved (2018-07-20) the current study with ChiCTR number: ChiCTR1800017274, URL:https://www.chictr.org.cn/showproj.html?proj=28262, and this study was conducted under the Declaration of Helsinki. After being informed of the benefits and risks of research, participants signed written consent.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

Author details
[1]Department of Cardiovascular Medicine, the Second Affiliated Hospital, Jiangxi Medical College, Nanchang University, No. 1 Minde Road, Nanchang 330006, China
[2]Center for Prevention and Treatment of Cardiovascular Diseases, the Second Affiliated Hospital, Jiangxi M edical College, Nanchang University, Nanchang, China
[3]Jiangxi Provincial Cardiovascular Disease Clinical Medical Research Center, Nanchang, China
[4]Jiangxi Sub-center of National Clinical Research Center for Cardiovascular Diseases, Nanchang, China

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