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ORIGINAL RESEARCH

Subclinical Cardiac Dysfunction in Hyperuricemic Gout and Asymptomatic Hyperuricemia: A Comparative Echocardiographic Study

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Objective: This study examines subclinical cardiac dysfunction in hyperuricemic populations by comparing echocardiographic parameters—including left ventricular diastolic dysfunction (LVDD), which reflects impaired ventricular relaxation, and left ventricular strain parameters, sensitive markers of early systolic dysfunction—between gout patients and asymptomatic hyperuricemic controls, and identifying echocardiographic and clinical predictors of cardiac impairment.

Methods: A cross-sectional study was conducted involving 202 hyperuricemic patients (100 gout and 102 asymptomatic hyperuricemic patients) from a tertiary rheumatology center (June 2021-May 2023). A comprehensive evaluation included conventional echocardiography and two-dimensional speckle-tracking. Biochemical indexes such as serum uric acid (SUA), lipid profiles, and renal function markers, along with demographic data, were collected. Group comparisons, univariate and multivariate regression analyses, and receiver operating characteristic (ROC) analyses assessed associations between echocardiographic parameters and clinical variables.

Results: Gout patients had a significantly higher prevalence of LVDD (31.00% vs 10.78%, $P<0.001$), a lower left ventricular ejection fraction (LVEF) (60.01±3.92 vs 65.85±3.78, $P<0.001$), and a lower absolute value of global longitudinal strain (GLS) (17.72 vs 19.68, $P<0.001$) compared to asymptomatic hyperuricemic patients. SUA positively correlated with GLS (β=0.236, $P<0.05$) in gout patients, while body mass index (BMI) and total cholesterol negatively correlated with LVEF (β=−0.248; −0.280, respectively, both $P<0.05$). In asymptomatic patients, triglycerides positively correlated with GLS and global circumferential strain (β=0.355; 0.310, both $P<0.05$). Risk factors for LVDD differed: gout patients showed BMI, creatinine, and course-dependent risks, while asymptomatic patients exhibited BMI and β2 microglobulin-related risks.

Conclusion: Distinct echocardiographic profiles and risk factor patterns in gout versus asymptomatic hyperuricemia suggest differing mechanisms of cardiac involvement, underscoring the need for tailored cardiovascular monitoring in hyperuricemic populations.

Keywords: gout, hyperuricemia, echocardiography, ventricular function, comparative study

Introduction

In the context of globalized health research, elevated uric acid levels have emerged as a significant public health concern. Epidemiological data clearly indicate a substantial rise in the prevalence of hyperuricemia in recent years. In developed countries such as the United States, approximately 20.1% of adults are affected by hyperuricemia, while in China, the prevalence among adult residents is around 13.3%, with high incidence rates observed across different age groups and regions.[1] The detrimental effects of elevated uric acid levels should not be overlooked, the most common being gouty arthritis, which severely disrupts daily life during acute attacks and, in advanced stages, may lead to joint deformities.[2]

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More critically, hyperuricemia is closely linked to the onset of various chronic diseases, particularly cardiovascular disorders.

Gout is primarily caused by elevated serum uric acid, often due to reduced renal excretion. Genetic variants, especially in urate transporters such as ABCG2 and SLC2A9, contribute significantly to hyperuricemia. Uric acid crystals trigger innate immune responses, activating the NLRP3 inflammasome and promoting pro-inflammatory cytokines such as IL-1β, leading to joint inflammation. At the same time, uric acid also modulates anti-inflammatory cytokines, including IL-37, which exerts protective effects in inflammatory diseases, reflecting its dual immunomodulatory role. In addition, epigenetic reprogramming (trained immunity) and microRNAs represent further regulatory layers in gout pathogenesis, influencing inflammatory pathways through mechanisms involving chromatin remodeling and NLRP3 activation.[3,4] Beyond joint involvement, extensive research has established a strong association between elevated uric acid levels and cardiac pathology, highlighting the systemic impact of hyperuricemia and underscoring the importance of cardiovascular assessment in these patients.[5] The adverse effects of hyperuricemia on cardiac structure and function are mediated through multiple mechanisms.[6] For example, hyperuricemia impairs endothelial function, disrupts normal vascular relaxation and contraction, and exacerbates oxidative stress, resulting in excessive free radical production that further damages vascular and myocardial tissues.[7] Over time, these pathological processes gradually weaken the heart's pumping capacity, increasing the risk of severe complications such as arrhythmias and heart failure.

Asymptomatic patients are often diagnosed with elevated serum uric acid (SUA) levels during routine medical examinations, despite the absence of overt clinical symptoms.[8] While numerous studies have established a direct link between gout and hyperuricemia, not all patients with elevated uric acid levels develop gout. In fact, some may remain asymptomatic throughout their lifetime.[9] Gout patients, due to recurrent chronic inflammation, experience more severe structural and functional cardiac impairments. Clinically, they often exhibit reduced myocardial strain capacity, increased myocardial stiffness, and left ventricular diastolic dysfunction (LVDD), all of which compromise overall cardiac performance.[10] In contrast, cardiac dysfunction in asymptomatic patients tends to be subtle in the early stages, making detection challenging.[11] While echocardiographic abnormalities have been described in patients with gout, few studies have systematically compared multiple echocardiographic parameters—including conventional indices and advanced strain imaging—between gout and asymptomatic hyperuricemia populations. A comprehensive comparative analysis of left ventricular systolic and diastolic function between asymptomatic patients and gout patients is crucial for the early identification of hyperuricemia-related cardiac abnormalities. Early detection enables timely and effective intervention, preventing disease progression and mitigating cardiovascular risks. One such method of early detection is two-dimensional speckle tracking imaging—a sophisticated ultrasound technique that tracks natural acoustic speckles within the myocardium to quantitatively assess myocardial deformation, allowing for the identification of subclinical myocardial dysfunction before overt changes become apparent on conventional echocardiography.

Consequently, this study aims to assess and compare left ventricular function parameters in asymptomatic patients and gout patients using echocardiography and two-dimensional speckle tracking imaging technology. Furthermore, it explores the correlation between these parameters and clinical indicators, providing essential clinical evidence for the early diagnosis and treatment of cardiovascular diseases in both patient groups. By improving early detection and intervention strategies, this research seeks to enhance patient survival outcomes and overall quality of life.

Methods

Subjects

This study enrolled gout patients with hyperuricemia who attended the Rheumatology and Immunology Department, both as outpatients and inpatients, at the First Affiliated Hospital of Chengdu Medical College between June 2021 and May 2023. The inclusion criteria were as follows: 1) Age between 18 and 65 years; 2) No gender restrictions; 3) body mass index (BMI) ranging from 18.5 to 30; 4) The asymptomatic hyperuricemia group met the diagnostic criteria for asymptomatic hyperuricemia as outlined in the 2020 American College of Rheumatology (ACR) guidelines for gout management;[12] 5) The gout group met the 2015 classification criteria for gout established by the ACR and the European League Against Rheumatism (EULAR).[13] The exclusion criteria were delineated as follows: 1) Patients with pre-existing

cardiac conditions, including hypertension, coronary artery disease, ischemic heart disease, dilated or hypertrophic cardiomyopathy, valvular heart disease, congenital heart disease, or a history of cardiac surgery or pacemaker implantation; 2) Patients diagnosed with autoimmune diseases such as rheumatoid arthritis and systemic lupus erythematosus, as well as those with chronic kidney disease or diabetes; 3) Patients on urate-lowering therapy; 4) Patients with poor compliance, making it difficult to cooperate throughout the study; 5) Patients with poor ultrasound imaging quality due to severe thoracic gas interference. The asymptomatic hyperuricemia group was selected from individuals who underwent routine health examinations at our hospital's physical examination center. These individuals were matched with gout patients in terms of age and sex. According to inclusion and exclusion criteria, patients were categorized into a gout group (n = 100) and an asymptomatic group (n = 102). This study was a retrospective cross-sectional study. The data were obtained from existing medical records at the First Affiliated Hospital of Chengdu Medical College. All data were anonymized prior to analysis, and no identifiable patient information was used. The study was approved by the Ethics Committee of the First Affiliated Hospital of Chengdu Medical College (Approval No.: 2019CYFYHEC-BA-34). As the data were anonymized and retrospectively collected, the requirement for informed consent was waived by the ethics committee. This study was conducted in accordance with the principles of the Declaration of Helsinki.

Clinical Diagnostic Criteria for Primary Gout and Asymptomatic Hyperuricemia

According to the 2015 gout classification criteria proposed by the ACR and the EULAR,[13] a suspected gout diagnosis requires at least one episode of swelling, pain, or tenderness in a peripheral joint or bursa. If monosodium urate crystals are identified in the affected joint, bursa, or tophus, gout can be directly diagnosed. For patients who do not meet this gold standard, a classification-based diagnostic approach is used, comprising three domains with a total of eight items, scoring up to 23 points. A score of ≥8 confirms a gout diagnosis.

According to the 2020 ACR guidelines for gout management,[12] asymptomatic hyperuricemia is defined as a SUA concentration of ≥420 μmol/L in the absence of a history of gout attacks or subcutaneous tophi. If a patient has no prior gout attacks or tophi, but advanced imaging detects monosodium urate crystal deposition, they can still be classified as asymptomatic.

Clinical Data Collection

Before performing transthoracic echocardiography, the general information of sex, age, height, weight, and course were collected and BMI was calculated according to the body mass index formula. Within eight hours following the echocardiographic examination, blood samples were obtained from all participants in the hospital's laboratory for biochemical analysis. The collected parameters included serum creatinine (CREA), blood urea nitrogen (BUN), SUA, cystatin C (CysC), β2-microglobulin (β2MG), low-density lipoprotein cholesterol (LDLC), high-density lipoprotein cholesterol (HDLC), triglycerides (TG), and total cholesterol (TC).

Acquisition of Ultrasound Data

The echocardiographic examinations were conducted by two experienced cardiovascular ultrasound physicians with over five years of expertise, who were blinded to the patients' clinical conditions and group assignments prior to the assessment. The examinations were performed using the EPIQ 7C ultrasound diagnostic system (PHILIPS, Netherlands) with an S5-1 transducer (1 MHz–5 MHz), equipped with two-dimensional speckle tracking analysis software. All participants were positioned in the left lateral decubitus position with the precordial region fully exposed. Electrocardiographic leads were correctly attached before initiating the ultrasound examination. All imaging measurements were strictly performed in standard echocardiographic planes in accordance with the guidelines of the American Society of Echocardiography (ASE).[14] The normal reference ranges for echocardiographic parameters were also defined based on ASE-recommended standards to ensure consistency and comparability in interpretation.

In the standard apical four-chamber view, pulsed-wave Doppler was utilized to measure the peak early mitral inflow velocity (MV Peak E Vel) and peak late mitral inflow velocity (MV Peak A Vel), followed by the calculation of the E/A ratio (Figure 1a). In the same view, tissue Doppler imaging was applied to measure the septal mitral annular early diastolic motion velocity (Sepe') (Figure 1b) and lateral mitral annular early diastolic motion velocity (Late'), from

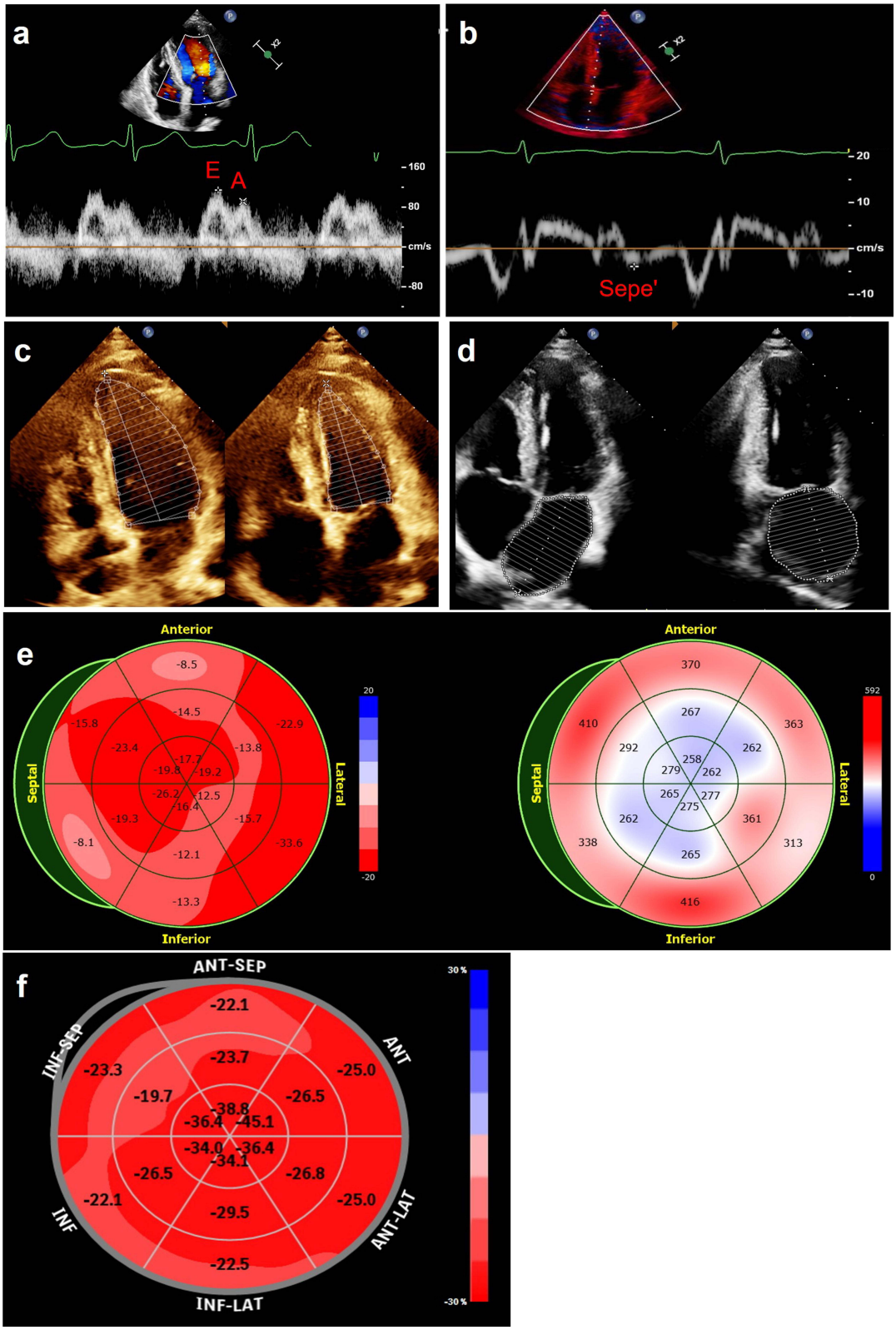

Figure 1 Transthoracic echocardiography in patients with gout. (**a**) Peak mitral valve velocities in early diastole E and late diastole A were measured using a pulsed Doppler. (**b**) Doppler measurements of septal mitral annular early diastolic motion velocity (Sepe'). (**c**) Measurement of left ventricular related parameters by apical biplane Simpson. (**d**) Left atrial volume and volume index. (**e**) global longitudinal strain (GLS). (**f**) Global circumferential strain (GCS). These images were directly exported from the Philips ultrasound system; minor text overlap is inherent to the system-generated output and does not affect measurement accuracy or interpretation.

which the average e′ value was derived, enabling the computation of the E/e′ ratio. In the standard apical four-chamber view, color Doppler flow imaging and continuous-wave Doppler were used to acquire the tricuspid regurgitation spectrum, and the maximum tricuspid regurgitation velocity was measured during systole. For left ventricular function assessment using the biplane Simpson's method, the standard apical four-chamber view was first obtained, and the endocardial border of the left ventricle was carefully traced at end-diastole and end-systole (Figure 1c). The same procedure was then performed in the standard apical two-chamber view. Upon completion, the ultrasound diagnostic system automatically calculated key parameters, including left ventricular ejection fraction (LVEF), left ventricular end-diastolic diameter (LVEDD), left ventricular mass index (LVMI), and left atrial volume index (LAVI) (Figure 1d). For strain analysis, high-quality dynamic images with optimal imaging conditions were selected. The Qlab analysis software embedded in the ultrasound system was used to process the stored image data. The software automatically divided the left ventricle into 18 segments and tracked the spatial movement of myocardial speckle echoes. If the automatic contouring was unsatisfactory, manual adjustments were performed to ensure the region of interest accurately followed the ventricular wall, fully covering the myocardium. The software then generated a bullseye plot for global longitudinal strain (GLS) and, using the same method, a bullseye plot for global circumferential strain (GCS) (Figure 1e and f). Normal values were defined as GLS $\leq -20\%$ and GCS $\leq -30\%$.[15]

Diagnosis of Left Ventricular Diastolic Insufficiency

According to the guidelines issued by the British Society of Echocardiography in 2024,[16] the primary reference parameters for patients with normal LVEF include five key indices: mitral inflow Doppler spectrum, average E/e′ ratio, septal or lateral e′ velocity, peak tricuspid regurgitation velocity, and left atrial volume index. Based on these criteria, the following conditions can be used to diagnose LVDD: 1. diastolic dysfunction is considered if $E/A \leq 0.8$ with $E \leq 50$ cm/s, or $E/A \leq 0.8$ with $E > 50$ cm/s, or if $0.8 < E/A < 2$ while meeting no more than one of the following conditions—average $E/e' > 14$, left atrial volume index > 34 mL/m^2, or peak tricuspid regurgitation velocity > 2.8 m/s; 2. diastolic dysfunction is indicated if $E/A \leq 0.8$ with $E > 50$ cm/s or $0.8 < E/A < 2$ while meeting at least two of the aforementioned conditions; 3. diastolic dysfunction is diagnosed if $E/A > 2$ or if a single peak E wave is observed.[17]

Statistics

Statistical analyses were performed using SPSS 26.0. Normality testing was conducted for continuous variables. If all data followed a normal distribution, they were expressed as mean ± standard deviation (**eq**) and analyzed using an independent sample *t*-test. If the data did not conform to a normal distribution, they were presented as median ± interquartile range (IQR) and analyzed using nonparametric tests. For correlation analysis, Pearson's correlation coefficient was used to assess linear relationships between continuous variables if the data followed a normal distribution, whereas Spearman correlation coefficient was employed for non-normally distributed data. Univariate and multivariate logistic regression analyses were performed to identify independent risk factors for LVDD in both gout and asymptomatic hyperuricemic patients. Variables with statistical significance in univariate analysis were included in the multivariate model. The results were reported as odds ratios with 95% confidence intervals. Receiver operating characteristic (ROC) curve analysis was performed to assess the diagnostic efficacy of course, CREA, and SUA for LVDD in both asymptomatic and gout patients. A difference with two-tailed P-value <0.05 was considered significant. Additionally, a post hoc power analysis was performed using G*Power 3.1 to evaluate the adequacy of the sample size for detecting differences in key echocardiographic parameters and LVDD prevalence.

Results

Comparison of General Information and Laboratory Indicators Between the Hyperuricemic Gout Group and the Asymptomatic Hyperuricemia Group

This study included a total of 100 gout patients, comprising 6 females (6.00%) and 94 males (94.00%), with a median age of 34.00 years (IQR: 29.25–40.00) and a median BMI of 27.12 (IQR: 24.20–28.60). In the asymptomatic hyperuricemia group, 4 females (3.92%) and 98 males (96.08%) were enrolled, with a median age of 34.00 years

(IQR: 30.00–42.00) and a median BMI of 24.51 (IQR: 23.00–26.71). Among gout patients, 60 (60.00%) had a history of smoking, and 71 (71.00%) reported alcohol consumption. In the asymptomatic group, 65 patients (63.73%) had a history of smoking, and 74 (72.55%) consumed alcohol. Regarding laboratory indices, SUA and CysC levels were significantly higher in gout patients compared to asymptomatic patients ($P < 0.05$). However, no statistically significant differences were observed between the two groups in CREA, BUN, TG, TC, LDLC, HDLC, or β2MG levels (Table 1).

Comparison of Ultrasound Parameters Between the Hyperuricemic Gout Group and the Asymptomatic Hyperuricemia Group

To assess the reproducibility of echocardiographic measurements, 30 patients were randomly selected. Echocardiographic images were analyzed offline by two independent sonographers and re-evaluated by the same sonographer at different time points. Intra-observer and inter-observer variability was assessed. The intraclass correlation coefficients (ICCs) for all parameters were greater than 0.9, indicating excellent repeatability and reliability of the measurements (Supplementary Table 1).

Compared to gout patients, asymptomatic patients exhibited significantly higher LVEF, E/A ratio, and e′ values ($P < 0.001$), while their LAVI, GLS, and GCS were significantly lower ($P < 0.05$). However, no statistically significant differences were observed between the two groups in terms of LVEDD, LVMI, and E/e′. Among gout patients, 31 patients (31.00%) were diagnosed with LVDD, whereas 11 patients (10.78%) in the asymptomatic group had LVDD. The incidence of LVDD was significantly higher in gout patients compared to asymptomatic patients ($P < 0.001$). (Table 2).

Analysis of Indicators Related to Systolic Function in the Gout Group and the Asymptomatic Hyperuricemia Group

The analysis of the correlation between left ventricular systolic function parameters and general data as well as laboratory indicators in gout patients revealed a positive correlation between SUA and GLS (β = 0.236, 95% CI = 0.000–0.007)

Table 1 Comparison of General Data and Laboratory Indexes Between Primary Gout Group and Asymptomatic Hyperuricemia Group

Variables	Gout Patients (n=100)	AHU Patients (n=102)	*P*
General information			
Age, years, median (IQR)	34.00(29.25–40.00)	34.00(30.00–42.00)	0.659
Gender, n, %	94(94.00%)	98(96.08%)	0.496
Course, years, median (IQR)	4.00(2.00–7.00)	4.00(2.00–5.00)	0.545
BMI, Kg/m^2, median (IQR)	27.12(24.20–28.60)	24.51(23.00–26.71)	<0.001
Smoking, n, %	60(60.00%)	65(63.73%)	0.583
Alcohol consumption, n,%	71(71.00%)	74(72.55%)	0.807
Laboratory indexes			
CREA, umol/L, mean (SD)	86.27(14.31)	85.92(14.04)	0.859
BUN, mmol/L, median (IQR)	5.06(4.13–6.09)	5.09(4.15–6.08)	0.971
SUA, umol/L, median (IQR)	526.00(476.75–592.75)	508.00(454.25–552.25)	0.016
β2MG, mg/L, median (IQR)	2.20(1.73–2.80)	2.20(1.78–2.80)	0.937
CysC, mg/L, median (IQR)	1.03(0.90–1.16)	0.99(0.88–1.07)	0.037
LDLC, mmol/L, median (IQR)	2.76(2.33–3.64)	2.74(2.27–3.59)	0.860
HDLC, mmol/L, median (IQR)	1.17(0.93–1.51)	1.17(0.96–1.52)	0.862
TG, mmol/L, median (IQR)	1.92(1.46–2.69)	1.90(1.46–2.68)	0.915
TC, mmol/L, median (IQR)	4.73(4.09–5.31)	4.68(4.08–5.26)	0.872

Notes: Data are presented as mean ± SD or median (IQR) and frequency (%).
Abbreviations: AHU, asymptomatic hyperuricemia; IQR, interquartile range; SD, standard deviation; BMI, body mass index; CREA, creatinine; BUN, blood urea nitrogen; SUA, serum uric acid; β2MG, β2-microglobulin; CysC, cystatin C; LDLC, low-density lipoprotein cholesterol; HDLC, high-density lipoprotein cholesterol; TG, triglycerides; TC, total cholesterol.

Table 2 Comparison of Ultrasonic Parameters Between Primary Gout Group and Asymptomatic Hyperuricemia Group

Variables	Gout Patients (n=100)	AHU Patients (n=102)	*P*
LVEF, %, mean (SD)	60.01(3.92)	65.85(3.78)	<0.001
LVEDD, mm, mean (SD)	46.37(3.74)	46.37(3.70)	0.999
LVMI, g/m^2, median (IQR)	0.31(0.24–0.39)	0.31(0.24–0.39)	0.971
E/A, median (IQR)	0.69(0.63–0.73)	1.22(0.80–1.48)	<0.001
e′, cm/s, median (IQR)	−12.00(−13.07- (−11.50))	8.57(7.73–10.05)	<0.001
E/e′, median (IQR)	9.00(7.58–11.84)	8.75(7.24–11.54)	0.373
LAVI, mL/m^2, median (IQR)	31.95(28.50–34.65)	30.00(26.67–32.30)	0.001
GLS, %, mean (SD)	−17.72(1.40)	−19.68(1.46)	<0.001
GCS, %, median (IQR)	−24.90(−26.90- (−23.98))	−28.45(−29.30- (−27.05))	<0.001
Left ventricular diastolic insufficiency	31(31.00%)	11(10.78%)	<0.001

Notes: Data are presented as mean ± SD or median (IQR) and frequency (%).
Abbreviations: AHU, asymptomatic hyperuricemia; IQR, interquartile range; SD, standard deviation; LVEF, left ventricular ejection fraction; LVEDD, left ventricular end-diastolic diameter; LVMI, left ventricular mass index; LAVI, left atrial volume index; GLS, global longitudinal strain; GCS, global circumferential strain.

(Figure 2a). As SUA levels increase, GLS also increases, suggesting that myocardial longitudinal contraction function may be progressively impaired. This indicates a risk of myocardial injury in gout patients, with the severity of myocardial damage potentially correlating with higher uric acid levels. Additionally, BMI and TC were negatively correlated with LVEF (β = −0.248, 95% CI = −0.576 to −0.064; β = −0.280, 95% CI = −2.175 to −0.063) (Figure 2b and c). Among asymptomatic patients, TG exhibited positive correlations with both GLS and GCS (β = 0.355, 95% CI = 0.205–0.732; β = 0.310, 95% CI = 0.209–0.975, respectively). As TG levels rise, both GLS and GCS increase, indicating

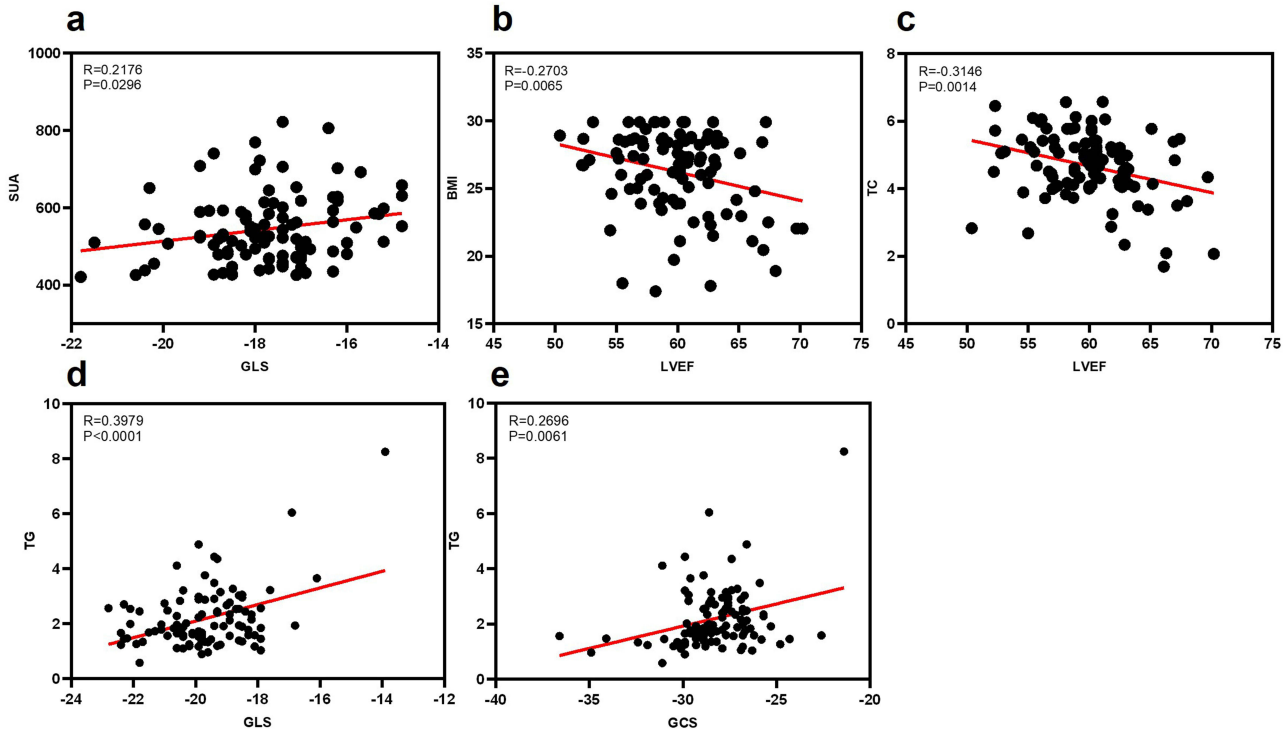

Figure 2 Correlation analysis of left ventricular systolic function parameters with general information and laboratory indicators. (**a**) Correlation between GLS and SUA in gout patients. (**b**) Correlation between LVEF and BMI in gout patients. (**c**) Correlation between LVEF and TC in gout patients. (**d**) Correlation between GLS and TG in asymptomatic hyperuricemia patients. (**e**) Correlation between GLS and TG in asymptomatic hyperuricemia patients. No statistical values are shown in these panels; all p-values are reported in the Results section.

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a potential deterioration of myocardial function in both longitudinal and circumferential dimensions. This suggests an elevated risk of myocardial systolic dysfunction in asymptomatic patients, with the risk appearing to be more pronounced at higher TG levels (Figure 2d and e).

Analysis of the Correlation Between General Data, Laboratory Indicators and the Occurrence of LVDD

Based on both univariate and multivariate logistic regression analyses, and after adjusting for potential confounding factors, the following variables were identified as independent risk factors for LVDD. In patients with gout, BMI (OR = 1.202, 95% CI: 1.033–1.479), serum creatinine (CREA) (OR = 1.407, 95% CI: 1.004–1.091), and disease duration (OR = 1.187, 95% CI: 1.049–1.342) were independently associated with the presence of LVDD (Figure 3a). In asymptomatic hyperuricemic individuals, BMI (OR = 1.747, 95% CI: 1.117–2.730) and β2-microglobulin (β2MG) (OR = 3.836, 95% CI: 1.136–12.949) were independently associated with an increased risk of LVDD (Figure 3b).

ROC Analysis of BMI, Course, CREA, and β2MG for LVDD

ROC curve analysis was conducted to evaluate the diagnostic performance of BMI, course, and CREA in identifying LVDD in gout patients, as well as the diagnostic efficacy of BMI and β2MG in asymptomatic patients with respect to LVDD. In gout patients, the combination of BMI (AUC = 0.645, 95% CI = 0.533–0.758), course (AUC = 0.677, 95% CI = 0.565–0.790), and CREA (AUC = 0.598, 95% CI = 0.476–0.719) demonstrated a relatively robust diagnostic performance for LVDD (AUC = 0.758, 95% CI = 0.657–0.859) (Figure 4a). This suggests that gout patients with higher BMI, prolonged course, and elevated CREA levels are more likely to develop LVDD. In asymptomatic patients, the combination of BMI (AUC = 0.769, 95% CI = 0.615–0.922) and β2MG (AUC = 0.670, 95% CI = 0.504–0.836) exhibited

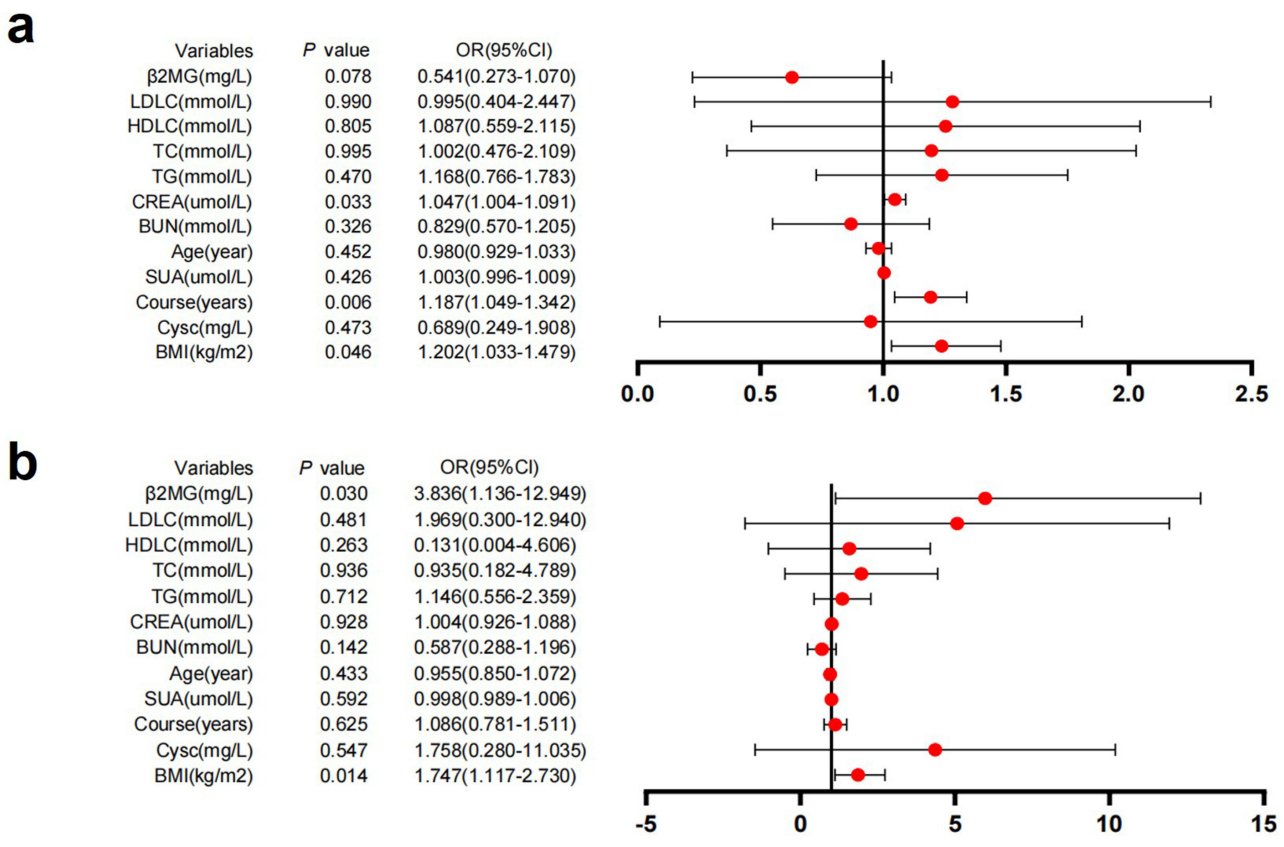

Figure 3 Analysis of the correlation between general data, laboratory indicators and the occurrence of left ventricular diastolic dysfunction. (**a**) Correlation between general data, laboratory indicators and the occurrence of left ventricular diastolic dysfunction in gout patients. (**b**) Correlation between general data, laboratory indicators and the occurrence of left ventricular diastolic dysfunction in patients with asymptomatic hyperuricemia.

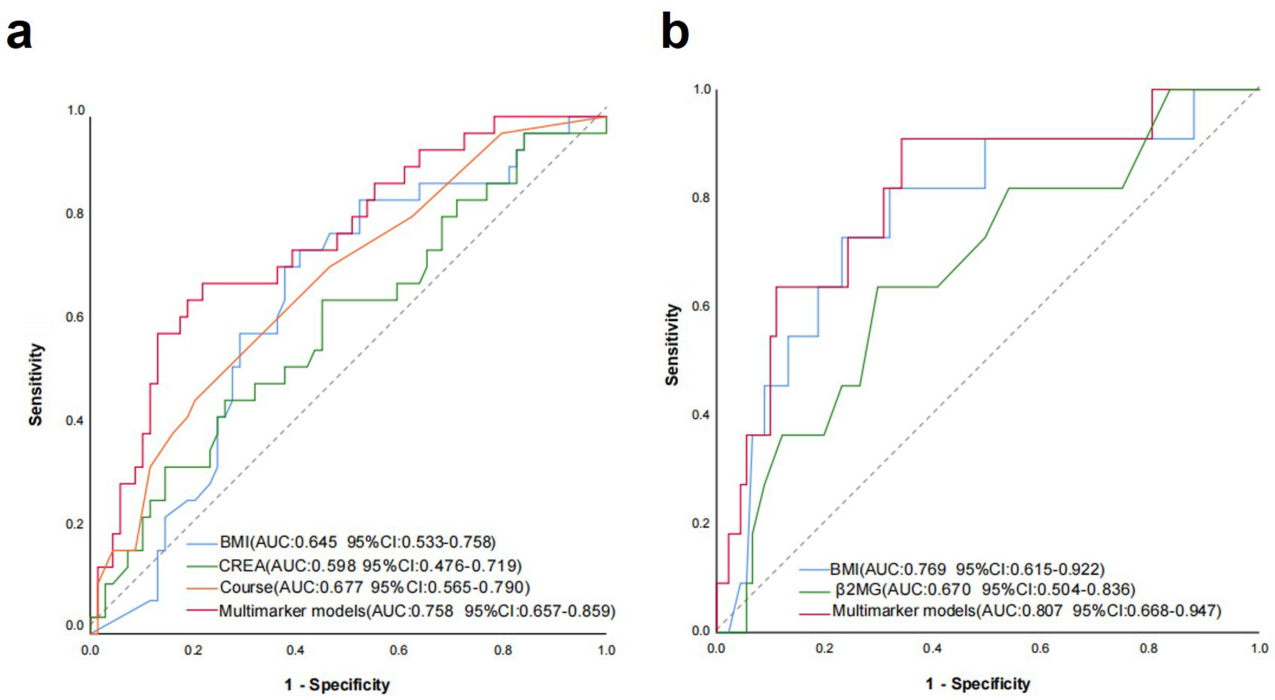

Figure 4 ROC curves for gout patients and patients with asymptomatic hyperuricemia. (**a**) The ROC was used to evaluate the diagnostic efficacy of body mass index BMI, CREA, Course, and their combination in diagnosing left ventricular diastolic dysfunction in gout patients. (**b**) The ROC was also used to evaluate the diagnostic efficacy of BMI, β2MG, and their combination in diagnosing left ventricular diastolic dysfunction in patients with asymptomatic hyperuricemia.

strong diagnostic potential for LVDD (AUC = 0.807, 95% CI = 0.668–0.947) (Figure 4b). This indicates that asymptomatic patients with higher BMI and β2MG levels are more predisposed to LVDD.

Discussion

In this study, we compared left ventricular systolic and diastolic functions between gout patients and asymptomatic patients, investigating the impact of general data and laboratory indicators on cardiac function, as well as their diagnostic value for LVDD. The results indicated that, compared to gout patients, asymptomatic patients exhibited higher LVEF, E/A ratio, and e′, while LAVI, GLS, and GCS were lower. In gout patients, GLS was positively correlated with SUA, while LVEF showed a negative correlation with BMI and TC, and the proportion of LVDD increases. LVDD was positively correlated with BMI, CREA, and course, and ROC curve analysis demonstrated that the combination of BMI, CREA, and course provided superior diagnostic efficacy for LVDD. In asymptomatic patients, GLS and GCS were positively correlated with TG, and LVDD was positively correlated with BMI and β2MG. ROC curve analysis further revealed that the combination of BMI and β2MG offered better diagnostic performance for LVDD. Our research group has previously conducted a series of studies on echocardiographic parameters in patients with gout.[18–20] The present study is a continuation of these previous investigations. This study, for the first time, conducted a comparative analysis of echocardiographic indices in hyperuricemic gout and asymptomatic patients, aiming to provide valuable clinical insights to help identify cardiovascular risks, guide treatment, and monitor disease progression, ultimately improving patient prognosis and quality of life.

Uric acid, the end product of purine metabolism, is catalyzed by xanthine oxidase (XO) and plays a crucial role in chronic inflammation.[21,22] This study revealed a positive correlation between GLS and SUA in gout patients, suggesting that elevated uric acid levels may be closely linked to impaired cardiac function. On the one hand, gout patients experience a sustained inflammatory state, on the other hand, uric acid crystals activate immune cells (such as monocytes and macrophages), triggering the release of various inflammatory cytokines (eg, IL-1β, IL-6, TNF-α, NF-κB) and activating the NLRP3 inflammasome, thereby exacerbating the inflammatory response.[23,24] These cytokines directly suppress myocardial cell contraction, promote myocardial cell apoptosis, reduce the number of myocardial cells, and

subsequently impair overall cardiac function. The positive correlation between GLS and SUA in this study may reflect the direct damaging effect of uric acid on myocardial cells through inflammatory pathways. Furthermore, prolonged inflammatory stimulation can induce myocardial cell hypertrophy, alter cardiac structure, and impact both systolic and diastolic functions. In addition, the deposition of urate crystals within myocardial cells or interstitial myocardium can form microtophi, causing local inflammation and foreign body reactions, which further weaken the overall contractile strength of the myocardium and the heart's pumping capacity.[25] The reduction in GLS observed in this study may be related to these structural changes induced by uric acid, indicating that elevated uric acid levels could lead to myocardial fibrosis and scarring, further disrupting both cardiac structure and function.[26] Additionally, several lines of evidence suggest that the upregulation of XO is associated with left ventricular hypertrophy and heart failure in both humans and animal models.[27] The positive correlation between GLS and SUA in gout patients in this study may further support the notion that uric acid, via the XO pathway and inflammatory mechanisms, contributes to left ventricular dysfunction. The upregulation of XO not only increases uric acid production but also exacerbates myocardial injury through oxidative stress and inflammation, ultimately leading to left ventricular hypertrophy and heart failure. However, no correlation was observed between SUA and left ventricular systolic or diastolic function parameters in asymptomatic patients. The underlying mechanism for this discrepancy may lie in the fact that in gout patients, the deposition of uric acid crystals exacerbates the inflammatory response, directly damaging myocardial cells, which leads to a decline in both systolic and diastolic functions. In contrast, asymptomatic patients have a lower level of inflammation, and uric acid has yet to cause significant pathological changes.[28] From a clinical perspective, this finding suggests that for hyperuricemic patients, greater attention should be given to monitoring cardiac function in those with gout, particularly through the use of echocardiography to assess sensitive indicators like GLS, in order to detect potential myocardial damage at an early stage. In conclusion, regardless of whether gout has developed, hyperuricemic patients should undergo regular echocardiographic screening. This not only aids in assessing cardiovascular risk but also allows for timely adjustments to treatment plans, helping to delay disease progression and improve prognosis.

Furthermore, in asymptomatic hyperuricemic patients, TG exhibited positive correlations with both GLS and GCS. Previous studies have suggested that hyperuricemia may inhibit lipoprotein lipase activity, reducing the breakdown of TG, thereby resulting in elevated TG levels.[29] Simultaneously, hypertriglyceridemia may exacerbate hyperuricemia by promoting uric acid production (eg, through increased purine metabolism) or reducing uric acid excretion.[30] This bidirectional relationship may explain the observed correlation between TG and GLS, GCS. Additionally, hypertriglyceridemia is often associated with heightened systemic inflammation and oxidative stress. Inflammatory cytokines (eg, TNF-α, IL-6) and oxidative stress products can directly damage myocardial cells, leading to myocardial fibrosis and increased myocardial stiffness, which in turn affects GLS and GCS. In asymptomatic patients, although no overt clinical symptoms are present, the correlation between TG and GLS, GCS suggests the presence of early myocardial dysfunction. Such subclinical damage may serve as a critical predictor for future cardiovascular events, such as heart failure or arrhythmias.

Moreover, several other differential indicators, such as BMI, CERA, and β2MG, also exist between gout patients and asymptomatic patients. BMI, a key indicator for assessing obesity, significantly impacts left ventricular systolic and diastolic functions in gout patients by increasing cardiac workload, triggering myocardial hypertrophy, and promoting structural remodeling.[31] This study demonstrated a negative correlation between BMI and LVEF in gout patients. This suggests that obesity may impair left ventricular systolic function by increasing cardiac load and inducing myocardial hypertrophy in gout patients. Previous studies have shown that in obese patients, LVMI is significantly elevated and positively correlated with BMI, further supporting the negative impact of obesity on left ventricular systolic and diastolic functions.[32] This indicates that the synergistic effect of gout and obesity exacerbates myocardial injury and declines in cardiac function. In asymptomatic patients, increased BMI may lead to LVDD. During the asymptomatic phase of hyperuricemia, subclinical inflammation and oxidative stress may already cause myocardial cell damage, suggesting that even in asymptomatic patients who have not yet met the diagnostic criteria for gout, elevated BMI may still impair left ventricular diastolic function through subclinical inflammation, oxidative stress, and metabolic abnormalities. Additionally, studies have found that CERA levels are significantly higher in gout patients compared to asymptomatic patients.[33] Although no significant difference in CERA levels was observed between the two groups in this study, CERA

emerged as a risk factor for LVDD in gout patients. As course increases, renal uric acid excretion decreases, leading to uric acid accumulation, while kidney dysfunction causes elevated CREA levels. Together, these factors accelerate the development of LVDD. One study found that gout patients with tophi had significantly lower absolute GLS values in speckle tracking imaging, and even after adjusting for various confounding factors, gout still impacted the main parameters in cardiac speckle tracking.[34] This finding aligns with the results of the present study, suggesting that gout patients experience poorer left ventricular systolic function, potentially due to the chronic inflammation caused by tophi. This study also identified β2MG as a risk factor for LVDD in asymptomatic patients. β2MG is a small-molecule protein that is normally freely filtered by the glomerulus and largely reabsorbed by the renal tubules. However, when renal function is impaired or the body is in a pathological state such as inflammation or malignancy, β2MG levels increase. Early detection of β2MG levels may serve as a valuable marker for identifying the onset of LVDD.

Although asymptomatic patients have not yet exhibited overt gout symptoms, their uric acid levels are already elevated beyond the normal range, which may lead to potential systemic damage through mechanisms such as chronic low-grade inflammation, oxidative stress, and metabolic abnormalities. A substantial body of evidence suggests that hyperuricemia itself may be associated with the progression of chronic kidney disease and its risk factors.[35] Additionally, hyperuricemia is commonly linked to insulin resistance and metabolic syndrome. Furthermore, Viggiano et al identified urate crystal deposition signs in the joints of asymptomatic patients, such as hyperechoic enhancement on the cartilage surface, double contour images of the cartilage, and high echogenicity clouds within the joints—signs that precede the onset of joint symptoms. Therefore, musculoskeletal ultrasound could potentially identify asymptomatic patient subgroups who may benefit from urate-lowering therapy.[36] This study explored the impact of asymptomatic hyperuricemia on the cardiovascular system through echocardiography, aiming to assess left ventricular function early and quantitatively in asymptomatic patients, thereby facilitating the timely identification of potential cardiac issues and providing a basis for early intervention. Echocardiography is capable of detecting cardiac structural changes and assessing cardiac function in gout patients. By measuring left ventricular wall thickness and calculating LVMI, echocardiography accurately evaluates the degree of left ventricular hypertrophy, offering crucial information for cardiovascular risk assessment. Furthermore, echocardiography can provide data on LVEF, left ventricular diastolic function parameters (such as E-wave, A-wave, E/A ratio, and deceleration time), and myocardial strain parameters (like GLS and GCS). Myocardial strain parameters are sensitive indicators of early myocardial dysfunction, as changes in GLS and GCS can reveal subtle alterations in myocardial contractility even when LVEF remains normal.[37] These parameters are crucial in the early diagnosis and intervention of both gout and asymptomatic hyperuricemia. The post hoc power analysis supports the robustness of our findings regarding LVDD prevalence and myocardial strain differences between gout and asymptomatic hyperuricemia patients. The adequate power strengthens confidence in the observed associations despite the moderate sample size. Moreover, the cardiac structural and functional data provided by echocardiography aid clinicians in developing comprehensive treatment strategies. For patients with significant cardiac structural changes or heart failure, special attention to cardiac protection is required during treatment. Appropriate drug selection is essential to avoid medications that may adversely affect cardiac function. Additionally, depending on the type and extent of cardiac abnormalities, multidisciplinary collaboration with cardiologists may be necessary to devise personalized treatment plans aimed at improving patients' quality of life and prognosis.

Recent epidemiological and clinical studies reinforce the growing recognition of hyperuricemia and gout as independent contributors to subclinical cardiac dysfunction. Clinical observations have demonstrated that gout and elevated serum uric acid levels are associated with increased risk of heart failure, myocardial infarction, and stroke, even after adjusting for traditional cardiovascular risk factors.[38] A comprehensive review presented updated mechanistic insights linking hyperuricemia to endothelial dysfunction, oxidative stress, inflammation, and metabolic dysregulation, highlighting its emerging role in cardiovascular injury.[39] Importantly, a recent echocardiographic study utilizing speckle tracking has shown that obesity combined with hyperuricemia significantly impairs left ventricular remodeling and myocardial function, which corresponds well with our findings that BMI and SUA correlate with GLS and GCS reduction.[40] Moreover, recent prospective data have suggested that serum uric acid is an independent predictor of right ventricular dysfunction and readmission in heart failure with preserved ejection fraction patients, emphasizing the broader impact of uric acid on biventricular myocardial performance even without overt systolic

dysfunction.[41] Taken together, these recent findings contextualize our results within the evolving body of literature, further supporting the clinical rationale for echocardiographic surveillance in hyperuricemic cohorts. Screening with measures such as GLS and GCS may identify subclinical myocardial dysfunction early, offering opportunities for risk stratification and timely intervention before overt heart failure develops. In summary, this study provides new insights into the early detection, treatment, and management of cardiac damage in asymptomatic patients with hyperuricemia by utilizing imaging techniques to compare the differences in left ventricular function between gout and asymptomatic hyperuricemia.

Although this study provides valuable findings from the comparison of echocardiographic parameters between gout patients and asymptomatic patients, there are several limitations. First, the relatively small sample size and the limited number of female participants precluded stratified demographic analyses in this study. This limitation may constrain the statistical power and generalizability of our findings across different population subgroups. Second, given the cross-sectional nature of this study, our findings indicate associations rather than causation, and further longitudinal studies are needed to establish temporal relationships and define clinical thresholds for intervention in asymptomatic hyperuricemic individuals. Besides We acknowledge that despite careful medical record review and face-to-face interviews, it is challenging to fully exclude the possibility of unreported or subclinical gout attacks in asymptomatic hyperuricemia patients, which may introduce recall bias or misclassification. This limitation should be considered when interpreting the findings related to asymptomatic hyperuricemia in our study. Finally, the limitations of a single-center study imply that the sample may lack broad representativeness, and the generalizability of the results may be constrained, particularly in different regions or healthcare settings where variations could exist. Therefore, future multi-center, large-sample prospective studies are necessary to include a broader range of subjects, reduce the impact of regional differences, and make the findings more universal and representative. This would allow for a more accurate understanding of the relationship between gout and cardiovascular diseases, clarify the causal links between uric acid levels and changes in cardiac structure and function, and provide more robust evidence for clinical interventions through long-term follow-up, enabling the dynamic observation of how variations in uric acid levels affect cardiac structure and function.

Conclusions

Distinct cardiac impairment patterns and risk factor profiles exist between gout and asymptomatic hyperuricemia. SUA-mediated systolic dysfunction and BMI/CREA-related diastolic impairment characterize gout patients, while lipid metabolism and β2-microglobulin emerge as key determinants in asymptomatic individuals. These findings support tailored cardiovascular monitoring strategies for hyperuricemic populations.

Data Sharing Statement

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

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Disclosure

The authors report no conflicts of interest in this work.

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