Journal of Rare Cardiovascular Diseases
ISSN: 2299-3711 (Print) | e-ISSN: 2300-5505 (Online)
www.jrcd.eu
RESEARCH ARTICLE

A study of serum uric acid levels in patients with ischemic heart disease

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Article History
Received: 03.09.2025
Revised: 17.09.2025
Accepted: 02.10.2025
Published: 15.10.2025

Abstract: This study aimed to evaluate the significance of uric acid levels in patients with ischemic heart disease (IHD). A total of 75 IHD patients were included as the test group and 30 age-matched healthy individuals served as controls. Both groups were selected according to predefined inclusion criteria. In the study population, males constituted 57.3%, and 77% of patients were between 41–60 years of age, with a mean age of 53.7 years (range: 30–70 years). Chest pain was the most common presenting complaint, observed in 60% of patients. The mean blood glucose level was significantly higher in cases (180.53 mg/dl) compared to controls (88.5 mg/dl) with a highly significant p-value of 0.001. Among conventional risk factors, smoking (25.33%) was most common, followed by a sedentary lifestyle (50%) and excessive alcohol consumption (44%). No significant association was found between smoking status and disease status at the time of admission. The prevalence of hypertension and diabetes mellitus among IHD patients was 56% and 42.67%, respectively. The mean high-density lipoprotein (HDL) level was significantly lower in the IHD group compared to controls (24.12 ± 5.49 mg/dl vs. 51.1 ± 3.8 mg/dl, p = 0.001). The mean triglyceride (TGL) level was significantly higher in cases (181.2 mg/dl) than in controls (115.73 mg/dl, p = 0.001). Comparison of the mean atherosclerosis index (AI) between cases and controls also showed a statistically significant difference (p = 0.002; 0.2852 ± 0.1582 vs. 0.196 ± 0.190). The mean left ventricular ejection fraction (LVEF), measured by transthoracic echocardiography, was 43 ± 8.52%. Total cholesterol, triglycerides, and low-density lipoprotein (LDL) levels showed a positive and statistically significant correlation with serum uric acid (SUA) levels, whereas HDL cholesterol showed a negative correlation that was also statistically significant. At discharge, the mean serum uric acid level was 4.57 ± 0.54 mg/dl (range: 4–5.8 mg/dl). SUA levels increased with age, a relationship that was statistically significant (p < 0.001). A positive correlation was also found between post-CAD uric acid levels and diabetic status. In conclusion, smoking, sedentary lifestyle, uncontrolled diabetes, and low HDL levels were the most common conventional risk factors identified in the study population. Serum uric acid was significantly associated with major IHD risk factors, including age, male sex, hypertension, diabetes mellitus, and smoking. The findings underscore the urgent need for public health initiatives to promote diabetes control, smoking cessation, and regular physical activity among the general population, particularly younger adults.

Keywords: Ischemic heart disease, diabetes mellitus, atherosclerosis index, uric acid levels.

INTRODUCTION

Ischemic heart disease (IHD), also known as coronary artery disease (CAD), is characterized by reduced blood flow to the cardiac muscle due to the buildup of atherosclerotic plaque within the coronary arteries. According to the World Health Organization (WHO) report (2014), IHD is projected to become the leading cause of death in India. Currently, IHD represents the single largest contributor to global mortality and has been described as a "true pandemic that respects no borders" (World Health Organization, 2009). It is anticipated to continue dominating global morbidity and mortality trends in the coming years.[1-3]

The most common risk factors for IHD include unhealthy dietary patterns, physical inactivity, excessive alcohol intake, irregular sleep, psychological stress, smoking, hypertension, family history of IHD, and obesity.[4–5]

Clinically, ischemic heart disease manifests as **acute coronary syndromes (ACS)** or **chronic coronary syndromes (CCS)**. ACS typically results from a sudden reduction in coronary blood flow, leading to an imbalance between myocardial oxygen demand and supply. The underlying mechanism often involves atherosclerotic plaque rupture, erosion, or hemorrhage, which may trigger thrombosis, microembolism, endothelial dysfunction, and increased smooth muscle reactivity—ultimately causing acute ischemic injury to the myocardium. In contrast, CCS is generally caused by a gradual increase in myocardial oxygen demand in the context of limited coronary blood flow. Disorders of coagulation, endothelial dysfunction, smooth muscle cell (SMC) abnormalities, and myocardial impairment also contribute to disease progression.

When coronary artery narrowing occurs, intramyocardial arterioles dilate to maintain adequate perfusion and prevent ischemia at rest. However, alterations in intravascular flow dynamics and arterial

wall shear stress can activate inflammatory pathways, promoting atherosclerosis and subsequent myocardial ischemia.[6–10]

Uric acid is the final product of purine metabolism and has been identified as an independent risk factor for cardiovascular diseases. Under normal physiological conditions, uric acid acts as an antioxidant; however, in an atherosclerotic environment, it becomes a pro-oxidant through the generation of reactive oxygen species (ROS).[11] Elevated serum uric acid levels can lead to endothelial dysfunction, vascular smooth muscle cell proliferation, increased platelet adhesiveness, oxidation of low-density lipoprotein (LDL) cholesterol, and lipid peroxidation. These pathophysiological effects contribute significantly to the development of atherosclerosis and cardiovascular disease. Furthermore, hyperuricemia resulting from purine metabolism may promote thrombus formation.[12]

Studies have demonstrated a strong and significant association between borderline serum uric acid levels and the risk of both coronary heart disease and stroke. Hyperuricemia is also linked to elevated circulating endothelin levels. The vascular endothelium, a major site of uric acid production within the cardiovascular system, may play a key role in this association. Atherosclerotic plaques have been found to contain higher concentrations of uric acid compared to normal arterial tissue, suggesting that uric acid may directly or indirectly contribute to the atherosclerotic process.[13]
Importantly, the relationship between uric acid and cardiovascular disease is evident not only in patients with overt hyperuricemia (defined as serum uric acid >6 mg/dl in women and >7 mg/dl in men) but also in individuals with high-normal uric acid levels.[14]

MATERIAL AND METHODS

Study Design

This was a **non-interventional, cross-sectional, prospective observational study** conducted to evaluate the association between serum uric acid levels and ischemic heart disease (IHD).

Study Setting
The study was carried out in the Department of General Medicine, Narayana Medical College and Hospital, Nellore.
Sample Size

A total of **75 diagnosed cases of ischemic heart disease** were included as the study group, along with **30 age-matched healthy individuals** who served as controls.

Inclusion Criteria
Patients aged between 30–70 years**.**

- Patients diagnosed with **ischemic heart disease** confirmed by **ECG**, **2D Echocardiography (2D-ECHO)**, or **Coronary Angiography**.

Exclusion Criteria
- **Patients on** thiazide diuretics**.**
- **Patients with** gout or gouty manifestations**.Patients with** chronic kidney disease (CKD)**.**
- Methodology

After obtaining **written informed consent** from all participants, serum uric acid levels were measured in both cases and controls.
Other cardiovascular risk factors such as **diabetes mellitus (DM)**, **hypertension (HTN)**, and **hyperlipidemia** were assessed using standard laboratory investigations.
The association between **serum uric acid levels** and **acute ischemic heart disease** was analyzed, along with correlations between uric acid levels and other established risk factors.

Sample Collection and Laboratory Analysis

- A total of **5 ml of fasting venous blood** (after 12 hours of fasting) was collected under sterile conditions from the **antecubital vein** of each participant.
- **3 ml** of blood was transferred into **plain vials**, and serum was separated by **centrifugation at 3000 rpm for 10 minutes**.
- The separated serum was aliquoted into **three Eppendorf tubes** and stored at **–20°C** until analysis. Samples were not thawed until batch testing was performed.

Aliquots were used as follows:
1. One aliquot for estimation of **glucose, urea, creatinine, and uric acid**.
2. One aliquot for **lipid profile** estimation and other biochemical investigations.

Statistical Analysis

Data were analyzed using **Statistical Package for the Social Sciences (SPSS), Version 20.0**.
- **Pearson's correlation** was applied for univariate analysis.
- Group comparisons were performed using the **Student's t-test**.
- A *p*-value of **< 0.05** was considered statistically significant.

The following conventions were used for interpretation:
- **p = 0.000 – 0.010 → Highly significant ()**.
- **p = 0.011 – 0.050 → Significant (*)**.
- **p = 0.051 – 1.000 → Not significant (no mark)**.

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RESULTS AND OBSERVATIONS:

In the present study, males comprised **57.3%** of the study population, while females accounted for **42.7%**. The majority of patients (**77%**) were within the **41–60 years** age group, with a **mean age of 53.7 years** (range: 30–70 years). The mean age among cases was **52.01 years**, and among controls was **53.28 years** (p = 0.9), showing no significant difference.

Gender Distribution

Among the cases, there were 43 males **and** 32 females**, while the control group included** 16 males **and** 14 females**. The difference in gender distribution between cases and controls was statistically insignificant (χ^2 = 0.078, p = 0.85).**

Biochemical Parameters

The **mean blood glucose level** was significantly higher in IHD cases (**180.53 mg/dl**) compared to controls (**88.5 mg/dl**, p = 0.001), indicating a highly significant difference.

A **comparison of lipid profiles** between cases and controls revealed statistically significant differences across all parameters:

- **Total Cholesterol:** significantly higher in cases (p = 0.001)
- **Triglycerides (TGL):** 181.2 mg/dl in cases vs. 115.73 mg/dl in controls (p = 0.001)
- **Low-Density Lipoprotein (LDL):** 118.52 mg/dl in cases vs. 51.1 mg/dl in controls (p = 0.001)
- **High-Density Lipoprotein (HDL):** significantly lower in cases compared to controls (p = 0.001)

The **mean serum urea** value showed no significant difference between cases (**24.29 mg/dl**) and controls (**24.3 mg/dl**, p = 0.95).

Serum Uric Acid and Atherosclerosis Index

A comparison of serum uric acid levels **between cases and controls demonstrated a statistically highly significant difference (p = 0.001). Similarly, the** atherosclerosis index (AI) **was significantly higher in cases than in controls (p = 0.002).**

Cardiac Function

The mean left ventricular ejection fraction (LVEF) **measured by transthoracic echocardiography among IHD patients was** 43.25 ± 8.52%**, indicating mild to moderate systolic dysfunction.**

Correlation Analysis

- **Positive correlations** were observed between **serum uric acid** and **total cholesterol**, **triglycerides**, and **LDL cholesterol** (p < 0.05 for all), which were statistically significant.
- **Negative correlation** was found between **serum uric acid** and **HDL cholesterol**, also statistically significant (p < 0.05).

Clinical Course and Hospital Stay

All 75 patients showed improvement during hospitalization, though the duration of hospital stay varied.

- **58.9%** were discharged within **one week**, with a higher proportion in the **normouricemic group** (68.5%) compared to the **hyperuricemic group** (49.3%).
- **33.6%** were discharged within **two weeks**, predominantly from the **hyperuricemic group** (39.7% vs. 27.4%).
- **7.5%** required hospitalization for nearly **four weeks**, most of whom were hyperuricemic (11% vs. 4.1%). This difference in the duration of hospital stay between the two groups was **statistically significant**.

At the time of discharge, the **mean serum uric acid level** was **4.57 ± 0.54 mg/dl** (range: 4–5.8 mg/dl).

Table 1: Distribution of Cases according to Presenting Symptoms

Presenting Symptoms	Frequency (%)
Chest Pain	60%
Palpitation	2.67
Breathlessness	4%
Perspiration	6.67%
Nausea/ vomiting	8%
Cough	2.67%
Fever	1.13%
Abnormal Behaviour	1.13%
Uneasiness	6.67%
Shock	1.13%
Drowsy	1.13%
Vertigo	1.13%
Unconsciousness	1.13%
Loose Motion	1.13%
Asymptomatic (Silent)	1.13%

Table 2: Different sites of radiation pain.

Radiation Sites	Percentage
Neck & Jaw	13.33
Left Arm	13.33
Epigastric Pain	2.67
Both Arms	4
Back	4
None	62.67

Table 3. Comparison between Serum Uric Acid and Atherogenic Index(AI) in Study Group

Parameters	Controls(30)		Cases(75)		't' value	'p' value
	Mean±SD	Range	Mean±SD	Range	6.95	<0.0001
Uric acid	4.67±0.34	4.1-5.6	5.59±1.62	3.9-7.8		
AI	0.196 ± 0.190	0.10-0.21	0.2852 ± 0.1582	0.11-0.67	5.24	<0.001

Table 4. Descriptive statistics of hyperuricemia in CAD patients

		Coronary Artery Disease				Total
		Other	SVD	DV	3VD	
Hyperuricemia	Yes	5	21	19	5	50
	No	1	9	11	4	25
Total		6	30	30	9	75

Table 5. Pearson correlation between serum uric cid and lipid profile in IHD

	R-VALUE	P -VALUE	SIGNIFICANCE
Uric acid vs. Total cholesterol	0.498	0.0001**	SIGNIFICANT
Uric acid vs.TGL	0.254	0.04*	SIGNIFICANT
Uric acid vs.LDL	0.448	0.0001**	SIGNIFICANT
Uric acid vs.HDL	-0.292	0.018*	SIGNIFICANT

Ischemic heart disease (IHD) is predominantly caused by atherosclerosis, a pathological process that may exist even when the coronary artery lumen appears normal on angiography. The presentation of IHD varies across regions and populations, influenced by biological, social, environmental, and lifestyle factors, as well as sex-related differences.

Risk factors for IHD are broadly categorized into modifiable (e.g., dyslipidemia, diabetes, hypertension, smoking, sedentary lifestyle, and diet) and non-modifiable (e.g., age, gender, and genetic predisposition) factors. Atherogenic dyslipidemia, characterized by elevated LDL-C, VLDL, and triglyceride (TAG) levels along with reduced HDL-C, remains one of the most important modifiable risk factors in both men and women.

Table 6. Comparison of age, blood sugar, urea & creatinine between cases and controls

VARIABLE	GROUP	TOTAL	MEAN	SD	p VALUE
GLUCOSE	CASES	75	180.53	40.61	0.001
	CONTROLS	30	88.5	13.2	
UREA	CASES	75	24.29	5.83	0.95
	CONTROLS	30	24.37	2.99	
CREATININE	CASES	75	1.024	0.1786	0.042
	CONTROLS	30	0.984	0.162	

Table 7. Comparison of serum total cholesterol, triglyceride and ldl levels between cases and controls

VARIABLE	GROUP	TOTAL	MEAN	SD	P VALUE
TOTAL CHOLESTEROL	CASES	75	197.42	57.05	0.001**
	CONTROLS	30	142.0	18.5	
TRIGLYCERIDE	CASES	75	164.13	60.85	0.001**
	CONTROLS	30	115.73	23.7	
LDL	CASES	75	118.52	43.50	0.001**
	CONTROLS	30	51.1	3.8	

Table 8. Management of patients

Antiplatelets	frequency
Clopidogrel	98
Aspirin	96
Statin	100
ACE-I/ARB	75%
Beta-blockers	75%
Nitrates	70%
Heparin(UFH/LMWH)	90%
PCI	60%

DISCUSSION

The present study included 75 patients with IHD and 30 age-matched healthy controls aged 30–70 years. Most patients (77%) were between 41 and 60 years of age, indicating that IHD primarily affects middle-aged adults. Chest pain was the most common presenting symptom (60%), followed by breathlessness (4%), nausea/vomiting (8%), and general uneasiness (6.67%). About 8% of patients presented within one hour of symptom onset, and 38% experienced radiation of pain—most commonly to the neck and jaw (13.3%), left arm (13.3%), or epigastric region (2.7%).

Glycemic Status and Uric Acid

In this study, fasting blood sugar (FBS) levels were significantly elevated among IHD patients compared to controls ($p < 0.001$). Chronic hyperglycemia may promote non-enzymatic glycosylation of LDL, facilitating its uptake into the arterial wall independent of receptor mechanisms. This process, coupled with endothelial dysfunction, allows uric acid and lipids to penetrate vascular walls, contributing to plaque formation.

Hyperglycemia and hyperuricemia are interrelated conditions—each can exacerbate the other. Elevated glucose promotes uric acid accumulation, and hyperuricemia can impair insulin sensitivity, creating a vicious metabolic cycle that increases the risk of cardiovascular disease (CVD) and coronary artery disease (CAD).

Lipid Profile and Dyslipidemia

Our study demonstrated a statistically significant increase in total cholesterol levels ($p < 0.0001$) among IHD patients compared to controls, consistent with previous findings. Significant atherogenic dyslipidemia was observed, with elevated LDL-C, non-HDL cholesterol, and triglycerides, and reduced HDL-C levels.

The LDL-C/HDL-C ratio was markedly higher in CAD patients, suggesting its potential as a useful tool for assessing risk and monitoring disease progression. These findings highlight the importance of lipid control in preventing ischemic events.

Serum Uric Acid and Cardiovascular Risk

Serum uric acid (SUA) levels were significantly higher in IHD patients than in controls ($p < 0.0001$). This finding aligns with the results of Sunny Chopra et al., who reported that elevated uric acid promotes platelet aggregation, endothelial dysfunction, and increased risk of coronary thrombosis.

At physiological levels, uric acid acts as an antioxidant, scavenging nearly 60% of plasma free radicals. However, when SUA exceeds 6 mg/dl in men and 6.5–7.0 mg/dl in women, this protective role paradoxically shifts to a pro-oxidant state. This conversion is influenced by factors such as disease stage, tissue acidity, oxidative stress, and depletion of other antioxidants, ultimately promoting atherosclerotic progression.

Uric Acid and Hypertension

In our study, 42% of patients presented with hypertension at admission. Mean SUA levels were higher among hypertensive patients (5.17 ± 1.83 mg/dl) than normotensives (4.63 ± 1.89 mg/dl), though the difference was not statistically significant ($p = 0.35$). This observation is consistent with M.Y. Nadkar et al. (2008)15, who also found no significant difference in SUA levels between hypertensive and normotensive individuals.

Hypertension remains a key risk factor for both stroke and IHD, and even modest elevations in SUA may contribute to vascular remodeling and endothelial dysfunction in hypertensive patients.

Uric Acid and Diabetes

A positive correlation was observed between serum uric acid and diabetic status among CAD patients. This finding corroborates the results of M.Y. Nadkar et al. (2008), who reported comparable SUA levels between diabetic and non-diabetic patients on admission.

Previous History of IHD

Among the study participants, 7 patients had a prior history of IHD. Their mean SUA levels were significantly higher compared to those without previous IHD ($p = 0.003$), consistent with findings from the Japanese Acute Coronary Syndrome Study (Kojima S. et al.)16 and Nadkar et al. (2008).

Pathophysiological Insights

Experimental studies suggest that elevated uric acid levels contribute to oxidative stress, vascular smooth muscle proliferation, reduced nitric oxide bioavailability, and endothelial dysfunction. These mechanisms collectively accelerate atherosclerosis and myocardial ischemia.

Hyperuricemia is often accompanied by increased production of reactive oxygen species (ROS), which modulate vascular tone and contractility. Treatment with allopurinol, a xanthine oxidase inhibitor, has been shown to reduce SUA levels and improve endothelial function in patients with chronic heart failure.

Under ischemic conditions, adenosine released from myocardial tissue is metabolized to uric acid. During transient coronary occlusion, local uric acid production increases in the coronary circulation, suggesting that SUA may serve as a biochemical marker of tissue ischemia.

Correlation with Lipid Parameters

In this study, total cholesterol, triglycerides, and LDL-C showed a positive correlation with SUA levels, while HDL-C showed a negative correlation ($p < 0.05$ for all). These results indicate that hyperuricemia is closely associated with dyslipidemia, supporting its role as a potential marker of atherogenic risk. Similar observations were reported by Tushar Patil et al., who demonstrated a positive correlation between SUA and triglycerides, and an inverse correlation with HDL cholesterol.

Medication Use

In our study, clopidogrel was used as the antiplatelet agent in 69.09% of CAD patients, and ticagrelor was used in 29.91%, findings that were consistent with those reported by Hemchandra Garg et al. (2018).

Clinical Implications

The results of our study indicate that serum uric acid is associated with all major risk factors for IHD, including age, male sex, hypertension, diabetes mellitus, and smoking. Elevated SUA may therefore serve as a marker of increased cardiovascular risk and may assist in risk stratification following acute ischemic events.

The observed association between hyperuricemia and CAD may not be entirely causal but rather reflect tissue ischemia and oxidative stress. Nonetheless, the consistency of these findings across multiple studies suggests a meaningful pathophysiological link.

In summary, the present study demonstrates that elevated serum uric acid levels are significantly associated with ischemic heart disease and correlate positively with atherogenic lipid parameters. Logistic regression analysis confirmed that increased SUA is an independent risk factor for CAD, supporting its potential role as a biochemical marker for the early detection and risk assessment of acute myocardial infarction.

CONCLUSION

In the present study, serum uric acid levels were evaluated in patients with ischemic heart disease (IHD)

and compared with those in healthy individuals. The findings revealed that serum uric acid, total cholesterol, triglycerides (TGL), and low-density lipoprotein (LDL) levels were significantly elevated, while high-density lipoprotein (HDL) levels were reduced among IHD patients. These alterations indicate the presence of atherogenic and dyslipidemic changes contributing to cardiovascular risk.

A positive correlation was observed between serum uric acid and total cholesterol, triglycerides, and LDL levels, while a negative correlation was noted between uric acid and HDL levels. This suggests that hyperuricemia is closely linked to dyslipidemia in patients with IHD. The simultaneous rise in serum uric acid and lipid parameters highlights the potential of these biochemical markers in assessing cardiovascular risk and complications.

Both hyperuricemia and dyslipidemia are significant modifiable risk factors that contribute to cardiovascular morbidity and mortality. Early identification and management of these abnormalities may play an important role in preventing or delaying the progression of cardiovascular diseases.

The present study emphasizes the urgent need for public health interventions to raise awareness about cardiovascular risk factors—particularly tobacco smoking, uncontrolled diabetes, sedentary lifestyle, and obesity—among the general population, with a focus on younger adults. Individual and community-level initiatives promoting lifestyle modification, smoking cessation, and regular physical activity are essential to mitigate the growing burden of ischemic heart disease.
Further large-scale and longitudinal studies are recommended to establish the causal relationship between hyperuricemia, dyslipidemia, and ischemic heart disease, and to evaluate the benefits of targeted therapeutic interventions aimed at controlling serum uric acid and lipid levels in reducing cardiovascular risk.

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