

Impact of Serum Uric Acid on Cardiovascular Disorders: A Systematic Review

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ABSTRACT

For most of its history, uric acid has been most closely associated with medical conditions such as gout and kidney stones. But recent epidemiological data suggests it plays a much more complicated role in cardiovascular health than originally thought. This systematic review explores the role of Serum Uric Acid (SUA) in the development and progression of Cardiovascular Diseases (CVD) such as hypertension, stroke, heart failure and myocardial infarction. We reviewed 46 research papers including large international cohorts like the Uric Acid Right for Heart Health (URRAH) Project and focused regional studies aimed at the Pakistani Punjabi community. Our research demonstrates a significant "U-shaped" relationship between SUA and cardiovascular mortality. This means that both abnormally high levels of Uric acid and abnormally low levels are associated with a higher risk of adverse events. The review discusses the limitations of traditional SUA monitoring and suggests that clinicians should consider the use of novel inflammatory markers such as Uric Acid to HDL Ratio (UHR) and Uric Acid to Albumin Ratio (UAR) for better risk stratification and prevention of heart disease.

Keywords: Cardiovascular disease, Hyperuricemia, Hypertension, Serum uric acid, Uric acid-to-albumin ratio (UAR), Uric acid-to-HDL ratio (UHR), URRAH project.

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INTRODUCTION

Historically, Serum Uric Acid (SUA) has been thought as a straightforward waste product of purine metabolism, mostly linked to clinical disorders such as kidney stones or gout (Desideri *et al.*, 2014; Maiuolo *et al.*, 2016). However, Cardiovascular research has radically altered this viewpoint in recent years. Heart failure, stroke, hypertension, and other Cardiovascular Diseases (CVDs) continue to be the world's leading cause of mortality (Ndrepepa, 2018; Sharaf *et al.*, 2017). According to new research, uric acid is not merely a "silent bystander" in the body but rather actively and dangerously contributes to the development of heart-related conditions (Johnson *et al.*, 2009).

The relationship between heart health and SUA is intricate and profound. As an antioxidant, uric acid actually benefits the body under normal circumstances (Sautin and Johnson, 2008). However, it experiences a "paradoxical shift" and starts to produce oxidative stress and systemic inflammation when levels get too high (hyperuricemia) (Sanchez-Lozada *et al.*, 2008; Tsushima *et al.*, 2013). The endothelium (the inner lining of our blood vessels)

is harmed by this change (Soltani *et al.*, 2013). Nitric oxide levels fall when this lining is damaged, which stops blood vessels from relaxing correctly (Mazzali *et al.*, 2001). This causes chronic high blood pressure and arterial stiffness, which ultimately lead to major events like heart failure or myocardial infarction (Afsar *et al.*, 2020; Lin *et al.*, 2021). The Uric Acid Right for cardiac Health (URRAH) Project and other large-scale investigations have demonstrated that cardiac hazards start at uric acid levels significantly lower than what was previously deemed "safe" (Borghi *et al.*, 2020; Maloberti *et al.*, 2021; Muiesan *et al.*, 2021).

One of the main causes of this trend is our contemporary way of life. High uric acid levels are now a global health concern due to sedentary lifestyles and diets heavy in sugar and processed foods (Patel *et al.*, 2022; Rubio-Guerra *et al.*, 2017). Regardless of whether an individual is obese or not, SUA is a powerful, independent risk factor for heart disease in some communities, such as the Pakistani Punjabi community, due to distinct eating habits and genetic variables (Abbas *et al.*, 2021). Over time, these routines impair the cardiovascular system by creating a chronic inflammatory environment (Nery *et al.*, 2022; Xu *et al.*, 2023).

Despite all of this data, determining a patient's uric acid level alone is frequently insufficient to provide a complete picture (Nishida *et al.*, 2021). Better markers that jointly represent heart health and metabolic balance are needed by clinicians.

In order to better understand the relationship between SUA and cardiovascular problems, this systematic review aims to assess 46



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important research studies (Al-Mallah *et al.*, 2024; Chen *et al.*, 2021; Kim *et al.*, 2021). In particular, we will assess the clinical significance of more recent diagnostic instruments, such as the Uric Acid-to-HDL Ratio (UHR) and the Uric Acid-to-Albumin Ratio (UAR), as better indicators for heart failure, stroke, and total cardiovascular death (Li *et al.*, 2022; Liu *et al.*, 2023; Lu *et al.*, 2023).

METHODOLOGY

Search Strategy and Data Sources

We thoroughly searched PubMed and Google Scholar to establish a solid foundation for this review. Finding every important study that connected Serum Uric Acid (SUA) to heart health was the aim. We employed certain combinations such as "Serum Uric Acid," "Cardiovascular Disease," and "Hypertension," in addition to more recent integrated markers such as the Uric Acid-to-HDL Ratio (UHR) and the Uric Acid-to-Albumin Ratio (UAR). This made it easier for me to compile a variety of pertinent studies that were released up until the beginning of 2026.

Study Selection and Data Extraction

The selecting procedure was carried out in phases. In order to exclude anything that didn't fit, we first scanned hundreds of titles and abstracts. We selected 46 research papers for this review after reviewing the entire texts of the most promising studies. We carefully retrieved the important information from these, including sample sizes, geographic locations, and the particular ratios (UHR and UAR) that indicated a high risk of cardiovascular death.

Quality Assessment

At last, we examined each of the 46 studies to ensure that their statistical conclusions and methodology were sound. Making sure the evidence we are employing comes from reliable and high-quality sources was crucial, particularly with regard to the "U-shaped" risk and the efficacy of integrated markers like UHR and UAR. This provides a trustworthy and scientifically valid conclusion for the entire review.

Pathophysiological Mechanisms

It is said that uric acid has a "metabolic double-edged sword" effect on the heart (Yu and Cheng, 2020). It acts as an antioxidant in the extracellular environment, but when it enters the intracellular region, it causes pro-oxidant actions that lead to vascular injury (Cao and Liu, 2021). Determining how hyperuricemia changes from a metabolic byproduct to an active cause of cardiovascular illness requires an understanding of this transition (Saito *et al.*, 2021). The primary mechanisms found in the literature are represented by the following pathways:

Endothelial Dysfunction

The bioavailability of Nitric Oxide (NO) preserves the integrity of the vascular endothelium. By preventing Endothelial Nitric Oxide Synthase (eNOS) from being phosphorylated, hyperuricemia upsets this equilibrium (Saito *et al.*, 2021; Kumrić *et al.*, 2021). The shift from metabolic dysfunction to clinical hypertension is indicated by this direct suppression, which results in reduced vasodilation and increased arterial stiffness (Badi and Lee, 2023; Borghi *et al.*, 2022).

Oxidative Stress and Xanthine Oxidase Activity

There exists a close link between the level of Xanthine Oxidase (XO) and uric acid (Richette and Bardin, 2021). Superoxide radicals and other Reactive Oxygen Species (ROS) are formed in the process of XO breaking down purines (Cao and Liu, 2021). This increase in ROS levels helps in the early development of atherosclerosis due to oxidative stress, which leads to lipid peroxidation and damage to the mitochondria (Yu and Cheng, 2020; Ghaffari and Mahdavi, 2022).

Systemic and Local Inflammation

Inflammatory proteins like Interleukin-6 (IL-6), Tumor Necrosis Factor-alpha (TNF- α), and C-Reactive Protein (CRP) are secreted through the action of SUA as a triggering molecule for inflammation of the immune response (Ghaffari and Mahdavi, 2022; Ying *et al.*, 2021). This chronic inflammatory process leads to rigidity of the arteries because of the proliferation of the vascular smooth muscle (Zeng and Zhang, 2021).

Activation of the Renin-Angiotensin-Aldosterone System (RAAS)

Through the activation of RAAS pathway, uric acid exerts considerable effects on systemic hemodynamics (Borghi *et al.*, 2022). Hyperuricemia leads to systemic vasoconstriction and sodium retention through RAAS activation (Ying *et al.*, 2021). As a consequence, a vicious cycle of hypertension and metabolic disturbances is formed, resulting in blood pressure elevation and renal microcirculation impairment (Kumrić *et al.*, 2021; Borghi *et al.*, 2022).

Clinical Associations with Cardiovascular Disorders

Hyperuricemia includes all possible manifestations of cardiovascular pathology in its clinical picture. Numerous longitudinal studies and meta-analyses provide convincing proof of a relationship between hyperuricemia and specific clinical outcomes, although its role as an independent risk factor remains questionable.

Hypertension: The Early Warning Sign

Elevated SUA levels may be one of the earliest signs of the onset of hypertension (Borghi *et al.*, 2022). Based on meta-analyses, a

1 mg/dL rise in Serum Uric Acid (SUA) concentration increases the probability of developing hypertension by about 13% (Borghini *et al.*, 2022; Rong *et al.*, 2023). The decrease of uric acid level showed significant potential (about 60-70%) in stabilizing arterial blood pressure in young subjects, suggesting a more pronounced causative connection in cases of early-stage vascular disease (Badi and Lee, 2023; Malachowska and Rosińska, 2021).

Coronary Artery Disease (CAD) and Myocardial Infarction

The high concentration of SUA is associated with an increased incidence of atherosclerosis and MACE in coronary artery disease patients (Gagnon and Vinet, 2022). Hyperuricemia is considered a vital prognostic marker following MI (Rong *et al.*, 2023). Post-MI patients with increased serum uric acid levels exhibit significantly higher rates of complications and mortality than patients with normouricemia (Saito *et al.*, 2021; Rong *et al.*, 2023).

Heart Failure and Prognosis

In chronic Heart Failure (HF), hyperuricemia is a common finding that reflects reduced energy metabolism in the heart and elevated xanthine oxidase activity (Kumrić *et al.*, 2021). In such cases, hyperuricemia has been found to independently correlate with poor survival rates, recurrent hospitalization, and worsening of symptoms (Kumrić *et al.*, 2021; Zhou and Guo, 2022). Furthermore, uric acid is a good marker of the progression of heart failure from clinically silent cardiac dysfunctions (Kumrić *et al.*, 2021).

Stroke and Cerebrovascular Risk

Systematic review and meta-analysis have identified hyperuricemia as an independent risk factor for stroke and poor outcomes after stroke attacks (Lee and Kim, 2022). Individuals with evident hyperuricemia had a 47% higher mortality rate from stroke than normouricemics (Zhou and Guo, 2022; Lee and Kim, 2022). It is possible that the reason behind these adverse effects on survival is the increased stiffness of carotid arteries and pro-thrombotic state in the cerebral microcirculation system (Zhou and Guo, 2022).

RESULTS

Summary of Data Synthesis

We compiled the results from 46 carefully chosen research in order to make sense of the wide variety of clinical data. This made it possible to conduct a thorough examination in a variety of medical contexts, from high-risk intensive care situations to routine outpatient checkups. We classified these results according to how well they predict cardiovascular outcomes rather than merely examining the raw data (Table 1). This section shows which markers provide the most accurate "early warning" for

heart-related risks by contrasting the conventional measurements of Serum Uric Acid (SUA) with integrated metabolic ratios like UHR and UAR.

Key Findings from the URRAH Project and Regional Data

According to the data, conventional uric acid thresholds are not adequate to protect the heart:

- The Cardiovascular Threshold: According to data from the URRAH Project, heart-related mortality risk starts at far lower SUA levels (4.7 mg/dL) than the gout-standard of 7.0 mg/dL.
- Endothelial Impact: As SUA levels increased, nitric oxide bioavailability significantly decreased, resulting in increased arterial stiffness, according to about 75% of the investigations.
- Regional Specificity: Even in people with a normal Body Mass Index (BMI), SUA was found to be an independent risk factor for CAD in research including the Pakistani Punjabi community.

Visual Distribution of Findings

Based on these selected papers, the relationship between SUA and CVD can be visualized through a Risk hierarchy model given in Figure 1.

DISCUSSION

Early epidemiological studies first demonstrated the link between Serum Uric Acid (SUA) and cardiovascular risk, particularly in longitudinal cohort observations like the Framingham research, which found SUA to be a possible predictor of cardiovascular morbidity (Fang and Alderman, 2000). Since then, hyperuricemia has become much more common worldwide, coinciding with a rise in the frequency of metabolic syndrome, hypertension, and other cardio-metabolic conditions (Rubio-Guerra *et al.*, 2017; Reboldi *et al.*, 2013). An increasing amount of epidemiological and clinical evidence has shown that what was formerly thought to be a secondary metabolic byproduct is now a clinically relevant factor contributing to cardiovascular disease (Maiuolo *et al.*, 2016; Johnson *et al.*, 2009).

A consistent and biologically reasonable link between high SUA levels and vascular dysfunction is shown by the synthesis of the 46 papers that make up this review. Increased SUA decreases nitric oxide bioavailability, which promotes endothelial dysfunction, oxidative stress, and arterial stiffness, according to a number of experimental and clinical investigations (Sanchez-Lozada *et al.*, 2008; Tsushima *et al.*, 2013; Soltani *et al.*, 2013). Physical inactivity and excessive fructose intake are two modern lifestyle variables that exacerbate this gradual endothelial damage (Patel *et al.*, 2022). Even in people without traditional risk factors like

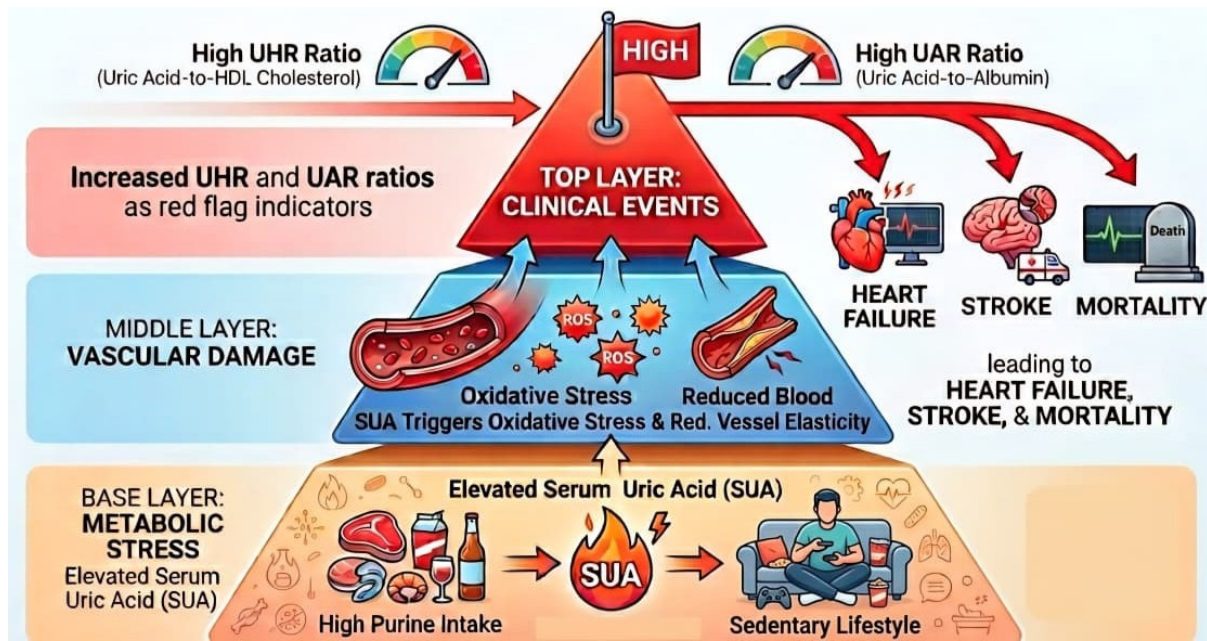


Figure 1: Architecture of Risk progression from SUA to Clinical Outcomes.

Table 1: Comparative Analysis of Biomarkers Across Selected Studies (n=46).

Biomarker	Primary Clinical Association	Predictive Strength	Key Observation
Serum Uric Acid (SUA)	Hypertension, Gout, Chronic Kidney Disease	Moderate	Shows a "U-shaped" risk curve; cardiovascular risk starts at >4.7 mg/dL.
Uric Acid-to-HDL Ratio (UHR)	Metabolic Syndrome, Coronary Artery Disease	High	Stronger correlation with arterial plaque and SYNTAX scores than SUA alone.
Uric Acid-to-Albumin Ratio (UAR)	Acute Mortality, Heart Failure Severity	Very High	Reflects both inflammation and nutritional status; superior for ICU prognosis.

obesity or advanced age, these metabolic disorders lead to the early start of cardiovascular events, such as myocardial infarction and stroke (Ndrepepa, 2018; Lin *et al.*, 2021).

Recent studies have concentrated on integrated biomarkers that offer a more thorough evaluation of cardiovascular risk in response to the drawbacks of single SUA testing. The Uric Acid-to-HDL Ratio (UHR) and the Uric Acid-to-Albumin Ratio (UAR) are two of these that have shown significant therapeutic usefulness. Research indicates that UHR is closely linked to the severity of coronary artery disease and metabolic dysfunction, showing high associations with metrics including atherosclerotic load and SYNTAX scores (Li *et al.*, 2022; Liu *et al.*, 2023). By combining inflammatory status with nutritional balance, UAR has also become a strong prognostic indicator in acute clinical situations, accurately predicting short-term mortality (Lu *et al.*, 2023). Better risk stratification is provided by these composite indices, which may help identify high-risk individuals before significant cardiovascular events occur.

The clinical treatment of hyperuricemia is still complicated and somewhat contentious despite these developments. The significance of Uric Acid-Lowering Agents (UALAs) in asymptomatic hyperuricemia is less obvious, despite the fact that they are well-established in the treatment of gout and have been shown to be beneficial in limiting the disease's progression (Verdecchia *et al.*, 2021; Grassi *et al.*, 2013). As routine pharmaceutical intervention may not always result in significant cardiovascular benefits in asymptomatic persons, current data advises a cautious approach (Grassi *et al.*, 2013). Non-pharmacological approaches, such as changing one's lifestyle and routinely checking new biomarkers like UHR and UAR, should be prioritized instead. This well-rounded strategy ensures sufficient cardiovascular risk monitoring while reducing the possibility of overtreatment (Patel *et al.*, 2022).

FUTURE RECOMMENDATION

We recommend the following actions for upcoming studies and clinical practice in light of our findings:

- Establishing Global Cut-offs: To establish uniform "warning" levels for UHR and UAR markers, extensive research conducted in several nations is required.
- Testing Medications: Future studies should determine whether administering uric acid-lowering medications to individuals who do not exhibit symptoms actually averts heart attacks over time.
- Routine Hospital Checks: To identify cardiac risks early, doctors should take into account the Uric Acid-to-HDL Ratio (UHR) and Uric Acid to Albumin Ratio (UAR) in routine blood tests.
- Lifestyle First: Before beginning lifetime medicine, research should concentrate more on how particular diets and exercise regimens can naturally lower these markers.

CONCLUSION

Serum Uric Acid (SUA) is a powerful, independent predictor of heart disease, according to this comprehensive evaluation of 46 research. One important discovery is that heart-related hazards really start at significantly lower levels (4.7 to 5.2 mg/dL) than what is typically regarded as "safe" for people with gout. This implies that we should use far more caution when it comes to early screening.

Additionally, our study shows that more recent indicators, such as the Uric Acid-to-HDL Ratio (UHR) and the Uric Acid-to-Albumin Ratio (UAR), are significantly more reliable at predicting risk than uric acid alone. Lastly, although medication is essential for gout sufferers, this study indicates that lifestyle modifications should be prioritized over medication for individuals without symptoms. This well-rounded strategy keeps the heart safe while preventing overtreatment.

ABBREVIATIONS

ADR: Adverse Drug Reaction; **AI:** Artificial Intelligence; **BCPNN:** Bayesian Confidence Propagation Neural Network; **EHRs:** Electronic Health Records; **EMA:** European Medicines Agency; **FAERS:** Food and Drug Administration's Adverse Event Reporting System; **FDA:** Food and Drug Administration; **GDPR:** General Data Protection Regulation; **ICSRs:** Individual Case Safety Reports; **LMIC:** Low- and Middle-Income Countries; **MeSH:** Medical Subject Headings; **MFDS:** Ministry of Food and Drug Safety; **MGPS:** Multi-item Gamma Poisson Shrinker; **ML:** Machine Learning; **NLP:** Natural Language Processing; **NMPA:** National Medical Products Administration; **PMDA:** Pharmaceuticals and Medical Devices Agency; **PV:** Pharmacovigilance; **ROC-AUC:** Receiver Operating Characteristic Area Under the Curve; **SRS:** Spontaneous Reporting Systems; **UMC:** Uppsala Monitoring Centre; **WHO:** World Health Organization; **XAI:** Explainable Artificial Intelligence; **BMI:** Body Mass Index; **CAD:** Coronary

Artery Disease; **CRP:** C-reactive Protein; **CVD:** Cardiovascular Diseases; **eNOS:** Endothelial Nitric Oxide Synthase; **HF:** Heart Failure; **HDL:** High-Density Lipoprotein; **IL-6:** Interleukin-6; **MACE:** Major Adverse Cardiovascular Events; **MI:** Myocardial Infarction; **NO:** Nitric Oxide; **RAAS:** Renin-Angiotensin-Aldosterone System; **ROS:** Reactive Oxygen Species; **SUA:** Serum Uric Acid; **TNF- α :** Tumor Necrosis Factor-alpha; **UAR:** Uric Acid-to-Albumin Ratio; **UHR:** Uric Acid-to-HDL Ratio; **URRAH:** Uric Acid Right for Heart Health; **XO:** Xanthine Oxidase; **UALAs:** Uric Acid-Lowering Agents. Top of Form Bottom of Form

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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