ORIGINAL ARTICLE

Correlation between Serum Uric Acid Level and Left Ventricular Ejection Fraction in Patients with Heart Failure

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Abstract

Background: Heart failure (HF) is a major public health concern with increasing prevalence worldwide. Serum uric acid (SUA) has been proposed as a potential biomarker in HF, with its levels potentially correlating with the severity of systolic dysfunction. However, the relationship between SUA and left ventricular ejection fraction (LVEF) remains unclear.

Methodology: A cross-sectional study was conducted at DY Patil University School of Medicine, Navi Mumbai, involving 60 patients diagnosed with HF. Patients were categorized based on LVEF into HF with preserved ejection fraction (HFpEF), mid-range ejection fraction (HFmrEF), and reduced ejection fraction (HFrEF). SUA levels were measured, and patients were classified into hyperuricemia or normal uric acid level groups. Demographics, comorbidities, and clinical symptoms were also recorded. Statistical analysis was performed to determine the correlation between SUA and LVEF.

Results: Of the 60 patients enrolled, 65% were female, with a mean age of 61–70 years. The majority had HFrEF (70%), followed by HFmrEF (26.67%) and HFpEF (3.3%). Hyperuricemia was observed in 38.3% of patients. A weak negative correlation was found between LVEF and SUA ($r = -0.070$), which was not statistically significant ($p = 0.599$). Although hyperuricemia was more prevalent in HFrEF, no significant relationship was established between SUA levels and severity of systolic dysfunction.

Conclusion: The study found a weak and statistically insignificant correlation between SUA levels and LVEF in HF patients. This suggests that SUA may not be a reliable biomarker for assessing the severity of systolic dysfunction. Further studies involving larger, more diverse populations are needed to clarify the prognostic role of SUA in HF.

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Introduction

Heart failure (HF) is a complex clinical syndrome characterized by the inability of the heart to pump blood efficiently, leading to inadequate tissue perfusion and fluid congestion. Several mechanisms have been proposed to explain the link between elevated serum uric acid (SUA) levels and cardiovascular dysfunction, such as endothelial dysfunction, oxidative stress, inflammation, and activation of the renin-angiotensin system. Exploring the potential relationship between SUA levels and left ventricular ejection fraction (LVEF) may provide valuable insights into the pathophysiology of HF and contribute to the identification of novel therapeutic targets or risk stratification strategies. This study aims to contribute to the current understanding of this association and potentially suggest clinical decision-making and therapeutic strategies for the management of HF patients.

Aim and Objective

Correlation between SUA level and LVEF in patients with HF.

Methodology

Study Design

An observational study.

Study Population

Patients diagnosed with HF at Dr DY Patil Medical College Hospital, Navi Mumbai.

Study Time

Research study was conducted for 18 months.

Inclusion Criteria

Patients diagnosed with HF age >18 years.

Exclusion Criteria

- Oncological conditions.
- Chronic renal failure.
- Gout.
- Autoimmune disease.
- Congenital heart disease.

To collect the required information from the study subjects, the "direct interview method" of primary source of information technique was used. The patients were interviewed for collection of necessary information using the pretested, semi-structured questionnaire method. The questionnaire was prepared by a thorough review of literature.

All patients with HF >18 years of age were included in the study. Patients were subjected to blood examination for determination of uric acid level and two-dimensional (2D) echocardiography for determination of LVEF. Correlation between uric acid levels and LVEF was studied.

Primary Outcomes

- To validate the increase in SUA levels in congestive HF.
- To correlate SUA levels with ejection fraction.

Secondary Outcome

- To establish the relationship of increased uric acid levels to functional class (NYHA, New York Heart Association) in congestive HF in predicting the severity.

Statistical Analysis

International Business Machines Statistical Package for the Social Sciences (IBM SPSS) (version 25.0) was utilized for statistical analysis, and Microsoft Excel 2016 was employed for data processing. Categorical data were presented as number and percentages, whereas continuous data were presented as mean and standard deviation. Within-group comparison of mean was done by paired *t*-test. For comparison of means between two groups, unpaired *t*-test was used. ANOVA was used for evaluation of difference at different time points. When a *p*-value was <0.05, the parameters were deemed to have significant connections or differences.

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Results

- The majority of patients (33.3%) were in the 61–70 age-group, with a female predominance (65%) (Figs 1 and 2).
- Common comorbidities included diabetes mellitus (56.7%), hypertension (50%), and myocardial infarction (55%) (Fig. 3).
- The most frequent symptom was dyspnea (93.3%), followed by edema (66.7%).
- About 70% of patients had HF with reduced ejection fraction (HFrEF), while 26.67% had HF with mid-range ejection fraction (HFmrEF) (Fig. 4).
- Hyperuricemia was present in 38.3% of patients (Fig. 5).
- There was a distribution of hyperuricemia across all LVEF categories, with the highest prevalence in the HFrEF group.

Discussion

Our study examined 60 patients with HF, analyzing their demographic characteristics, clinical presentation, comorbidities, LVEF, and SUA levels. The findings provide valuable insights into the complex interplay between these factors in HF patients.

Age and Gender Distribution

The study population predominantly consisted of older adults, with the highest proportion (33.3%) in the 61–70 age-group. This age distribution aligns with the known epidemiology of HF, which is more prevalent in older individuals.[1] The gender distribution showed a male predominance (65%), which is consistent with findings from other studies. For instance, a large-scale study by Maggioni et al.[2] reported a similar male predominance in HF patients across Europe.

Comorbidities

Our study revealed a high prevalence of comorbidities, with diabetes mellitus (56.7%), hypertension (50%), and previous myocardial infarction (55%) being the most common. These findings are in line with those reported by van Deursen et al.,[3] who emphasized the significant burden of comorbidities in HF patients and their impact on outcomes.

Clinical Presentation

Dyspnea was the most common presenting symptom (93.3%), followed by edema (66.7%) and chest pain (50%). These findings are consistent with the typical clinical presentation of HF described in major guidelines.[4]

Left Ventricular Ejection Fraction

The majority of patients in our study (70%) had HFrEF, defined as LVEF <40%. This is higher than the proportion reported in some population-based studies. For example, the EPICA study[5] found that approximately 50% of HF patients had reduced ejection fraction. The higher proportion in our study might be due to referral bias or differences in patient selection criteria.

Hyperuricemia and Heart Failure

Our study found that 38.3% of HF patients had hyperuricemia. This prevalence is similar to that reported by Huang et al.[6] who found hyperuricemia in 55.8% of acute HF patients. When analyzing the distribution of hyperuricemia across different LVEF categories, we found that 16 out of 42 patients (38.1%) with HFrEF had hyperuricemia, compared to 6 out of 16 (37.5%) with HFmrEF, and one out of 2 (50%) with HF with preserved ejection fraction (HFpEF).

While our study does not show a clear trend of increasing hyperuricemia prevalence with decreasing LVEF, it is important to note the small sample size, particularly in the HFpEF group. Other studies have reported a more pronounced association. For instance, Cicero et al.[7] found a significant inverse correlation between SUA levels and LVEF in HF patients.

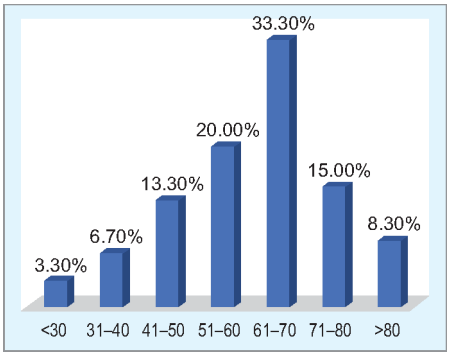

Fig. 1: Distribution of patients according to age

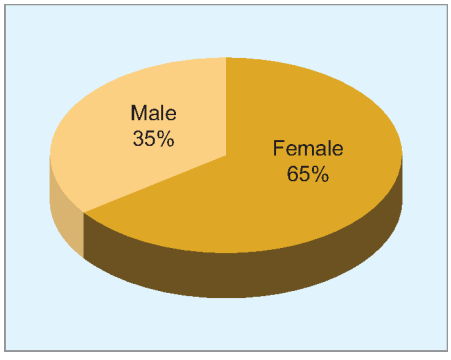

Fig. 2: Distribution of patients according to gender

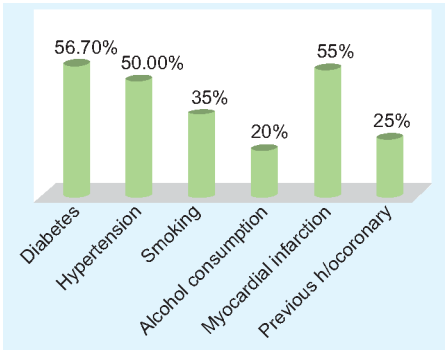

Fig. 3: Distribution of patients according to comorbidities

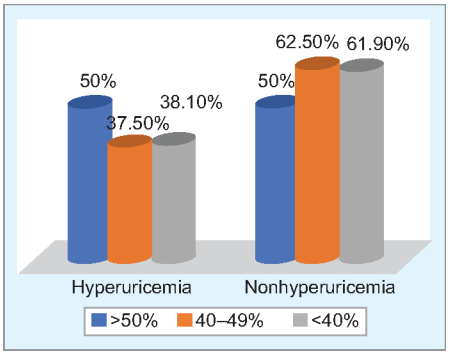

Fig. 5: Distribution of patients according to hyperuricemia and LVEF

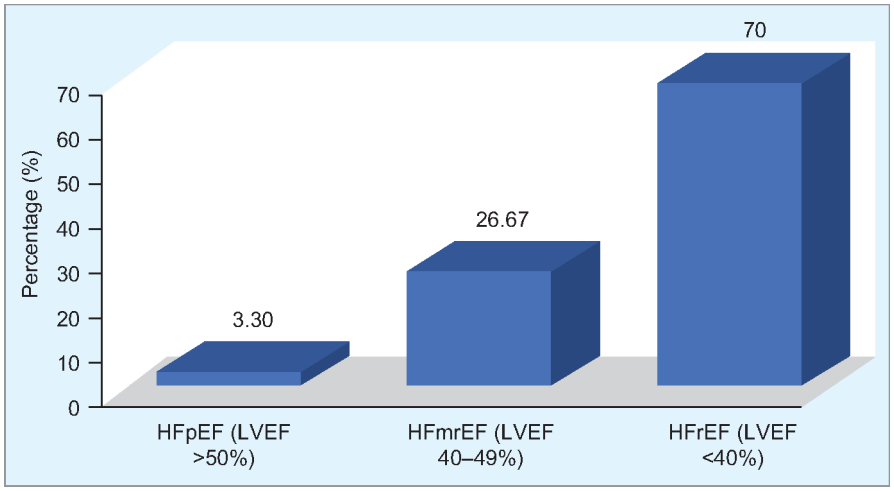

Fig. 4: Distribution of patients according to LVEF

The relationship between hyperuricemia and HF is complex and multifaceted. Elevated uric acid levels may contribute to the pathophysiology of HF through various mechanisms, including increased oxidative stress, endothelial dysfunction, and activation of the renin–angiotensin–aldosterone system.[8] Conversely, HF itself can lead to increased uric acid levels due to reduced renal perfusion and increased xanthine oxidase activity.

Our findings, while not conclusive, add to the growing body of evidence suggesting a potential role for uric acid in HF. The relatively high prevalence of hyperuricemia in our HF cohort, regardless of LVEF category, suggests that uric acid might be a valuable biomarker in HF management.

However, it is important to note the limitations of our study, including its cross-sectional nature and relatively small sample size. Larger, prospective studies are needed to further elucidate the relationship between uric acid levels and LVEF in HF patients and to determine whether uric acid-lowering therapies could have a role in HF management.

Conclusion

The distribution of patients across different LVEF categories, with a majority falling into the HFrEF group, highlights the significance of reduced ejection fraction in the study population. The presence of hyperuricemia in 38.3% of patients, with the highest proportion observed in the HFrEF group, suggests that elevated SUA levels may be associated with more severe cardiac dysfunction.

These results indicate that SUA levels could potentially serve as a biomarker for HF severity and progression. However, it is important to note that while an association has been observed, causality cannot be established based on this study alone. Further research, including longitudinal studies and investigations into the underlying mechanisms, is necessary to fully elucidate the relationship between hyperuricemia and HF.

The findings of this study may have important clinical implications. Monitoring SUA levels in HF patients could provide additional information for risk stratification and disease management. Moreover, therapies targeting uric acid metabolism might represent a potential avenue for future HF treatments, although this would require extensive further investigation.

In conclusion, this study contributes to the growing body of evidence linking hyperuricemia with HF, particularly in patients with reduced ejection fraction. While these results are promising, they also highlight the need for continued research in this area to better understand the complex interplay between uric acid metabolism and cardiac function in HF patients.

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