This article can be cited as: Haghaninejad H, Banifatemeh SA, Mohammadi H, et al. The role of serum uric acid in predicting left ventricular function and outcomes in patients with STEMI following PCI. Cardiovasc Biomed J. 2024; 4(1): 40-44.

Original Article

The role of serum uric acid in predicting left ventricular function and outcomes in patients with STEMI following PCI

Hasan Haghaninejad [1], Seyed Ali Banifatemeh [2], Hamidreza Mohammadi [1], Seyed Kazem Razavi Ratki [3], Reza Nafisi Moghadam [3], Nasim Namiranian [4], Ghazal Taheri Asl [1,*]

[1] Yazd Cardiovascular Research Center, Non-communicable Diseases Research Institute, Shahid Sadoughi University of Medical Sciences, Yazd, Iran
[2] Department of Internal Medicine, Shahid Sadoughi University of Medical Sciences, Yazd, Iran
[3] Department of Radiology, Faculty of Medicine, Shahid Sadoughi University of Medical Sciences, Yazd, Iran
[4] Diabetes Research Center, Shahid Sadoughi University of Medical Sciences, Yazd, Iran

*** Corresponding Author:**
Address: Afshar Hospital, Jomhouri Blvd, Yazd, Iran. **Postal code:** 8917945556; **Tel:** +98 9358355762; **Email:** taheriasl.gh@gmail.com

Article Information:
Received: 08 Jun 2024; Revised: 06 Aug 2024; Accepted: 07 Aug 2024 DOI: 10.18502/cbj.v4i1.16226

Abstract

Objectives: Percutaneous coronary intervention (PCI) is the preferred treatment for ST-elevation myocardial infarction (STEMI); however, reduced left ventricular ejection fraction (LVEF) remains a critical issue post-PCI. Serum uric acid (SUA) has emerged as a biomarker linked to cardiovascular events and may be associated with LVEF. This study aims to investigate the relationship between SUA levels and LVEF in STEMI patients treated with primary PCI and identify potential biomarkers for predicting patient outcomes.

Methods: This prospective study was conducted at Afshar Hospital, Shahid Sadoughi University of Medical Sciences of Yazd, from September 2019 to March 2021. Participants included 254 consecutive ST-elevation myocardial infarction (STEMI) patients undergoing primary PCI. Exclusion criteria were contraindications for angiography, history of myocardial infarction, PCI, CABG, NSTEMI, thrombolytic treatment, kidney diseases, gout, or alcoholism. Demographic data, clinical history, SUA levels, and echocardiographic parameters were collected. Statistical analysis was performed using SPSS version 20.0, with significance at $p<0.05$.

Results: 81.49% of patients were male, with a mean age of 57.7±11.47 years. Hypertension was the most prevalent condition (42.9%). SUA levels did not significantly differ between patients with LVEF below and above 40% ($p=0.39$). However, smoking was significantly associated with reduced LVEF ($p<0.001$). A weak negative correlation between SUA and LVEF was observed in males ($p=0.012$) but not in females ($p=0.097$). Overall, a weak negative correlation between SUA and LVEF was statistically significant ($p=0.05$).

Conclusions: Our findings revealed a weak but statistically significant overall negative correlation between SUA and EF. Further research with larger, more diverse populations is necessary to elucidate the relationship between SUA and cardiac function in STEMI patients.

Keywords: ST-elevation myocardial infarction, Serum uric acid, percutaneous coronary intervention

Introduction

Acute ST-elevation myocardial infarction (STEMI) occurs when blood flow to a part of the heart muscle is severely reduced or completely blocked. It can result in serious health problems and even death, particularly in developing countries. (1). PCI is the preferred perfusion strategy for STEMI patients (2). However, regardless of improvement in STEMI management, some patients may have poor outcomes, like reduced LVEF. Decreasing ejection fraction of the left ventricle causes dysfunctions of other organs, eventually giving rise to a poor prognosis as complications on the in-hospital prognosis of STEMI patients after PCI (3). One of the biomarkers that has appeared positively associated with cardiovascular events is SUA due to its relationship to total serum antioxidant capacity (4-7). However, the role of SUA in predicting LV function and outcomes in patients with STMI undergoing PCI remains unclear. Therefore, this study aims to investigate the relationship between SUA level and LVEF in patients with acute STEMI treated with PCI and also identify new biomarkers for predicting outcomes in these patients.

Matherials and Methods

Patient selection

This retrospective study was conducted at Afshar Hospital, affiliated with Shahid Sadoughi University Medical Sciences, from September 2019 to March 2021. Participants included 254 consecutive patients diagnosed with acute STEMI who underwent primary PCI. The diagnosis of STEMI was based on the criteria set by the American College of Cardiology (ACC) and the European Society of Cardiology. Exclusion criteria included contraindications for angiography, history of at least one MI, PCI, CABG, NSTEMI, or thrombolytic treatment, kidney diseases, gout, and alcoholism.

Data collection

Demographic variables included age, sex, BMI, and history of other diseases such as hyperlipidemia, hypertension, diabetes mellitus, and smoking. Serum urea levels of patients and echocardiographic parameters including ejection fraction, were collected.

Laboratory measurements

Blood samples were collected intravenously from each patient before the PCI procedure. SUA was assessed using a 7600 automatic biochemistry analyzer (Hitachi Limited Corporation, Japan).

Coronary angiography

Participants with STEMI underwent coronary angiography and PCI via femoral artery access. Coronary angiography was examined with Philips V 5000 (Netherlands) and by the Judkin protocol. After the primary PCI, the patients received medical treatment based on the routine instructions and were monitored for 24 hours in the CCU.

Echocardiography

Echocardiographic analysis was performed 48 hours after hospitalization using a VIVID 4 ultrasound systems device (GE Medical Systems, USA). The procedure adhered to the guidelines of the American Society of Echocardiography, and measurements were obtained.

Ethical consideration

This study was approved by the ethical committee of Shahid Sadoughi University of Medical Sciences (IR.SSU.MEDICINE.REC.1397.082), and written informed consent was obtained from all patients before the study.

Statistical analysis

The data were analyzed using SPSS version 20.0 software. Quantitative data were expressed as mean±standard deviation and analyzed by independent *t*-test. The Chi-square test was used to analyze the categorical data. The relationship between SUA and EF was analyzed using Pearson's correlation coefficient analysis. $P<0.05$ was considered statistically significant.

Results

The basic and clinical characteristics of all 254 patients with STEMI who had undergone PCI are summarized in Table 1. The most prevalent medical condition among these patients was hypertension, while diabetes was the least common Table 1.

Table1. Baseline characteristics of patients

Age; mean±sd	57.7165 ± 11.47
Sex, Male n (%)	207(81.49)
DM n (%)	70(26%)
HTN n (%)	109(42.9%)
HLP n (%)	73(28.7%)
smoking n (%)	96(37.8%)
BMI; mean±sd	26.79 ±4.35
Hospitalization; mean±sd	4.44 ±3.12

Data are presented with mean±SD, number (%); BMI: Body Mass Index; DM: diabetes mellitus

Table 2 presents demographic and clinical features of patients with EF below and above 40 that indicate age, sex, BMI, hypertension, hyperlipidemia, and SUA did not demonstrate any significant association with ejection fraction (EF) in patients following PCI. However, there is a notable and significant association between smoking and EF in these patients.(p-values >0.05). The average length of hospital stay was similar for both groups. (Average hospital stay was 4.44±1.68 days)

Table2. Comparison of demographic and basic clinical data in patients with EF<40 and EF>40

		Ef<40 N=72	**Ef>40 N=182**	**p-value**
Sex, n(%)	Male	63(30.4%)	144(69.6%)	
	Female	9(19.14%)	38(80.85%)	0.15
UricAcid, mean±sd		5.72 ± 1.54	5.32 ± 1.32	0.39
BMI, mean±sd		26.87 ± 1.7 3	26.77 ± 4	0.88
Age, mean±sd		59.5 ± 14.1	57 ±11.64	0.56
Hospitalization, mean±sd		4.7 ± 2.67	4.35 ± 1.63	0.23
DM, n (%)		20(27.5%)	50(27.8%)	0.96
HTN, n (%)		27(37.5%)	82(45.1%)	0.27
Smoking, n (%)		41(56.9%)	55(30.2%)	<0.001
HLP, n (%)		21(8.2%)	52(20.4%)	0.92

Data are presented using mean± SD and frequency (percentage). Statistical analysis was conducted using the chi-square test and independent t-test.

In males, there was a weak negative correlation between EF and uric acid, but it was not statistically significant. Additionally, there was a weak positive correlation in females, which also was not statistically significant. However, there was an overall weak negative correlation between EF and SUA, which was statistically significant. Table 3 and Figures 1-2 show the results.

Table3. Correlation of SUA and EF

Gender	**Pearson Correlation**	**p-value**
male	-.174*	.012
Female	.245	.097
Total	-0.123	0.05

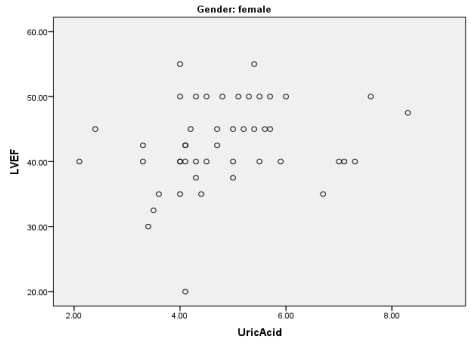

Figure1. Correlation between the LVEF SUA in females

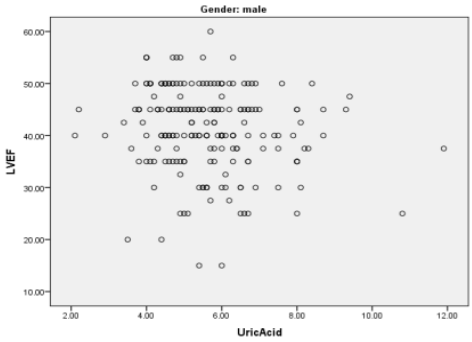

Figure2. Correlation between the LVEF and SUA in males

Discussion

This study aims to investigate the relationship between SUA level and LVEF in patients with acute STEMI treated with primary PCI and guide clinical decision-making in this high-risk population. Our study revealed that demographic factors such as age, sex, BMI, and SUA did not show significant associations with EF after PCI. The absence of significant associations in our study necessitates further discussion. First, in the context of our study population age is a well-known factor influencing cardiovascular outcomes (8), including EF (9).

However, within the context of our study, age may not independently impact EF after PCI. This may be due to the relatively narrow age range of participants or to the successful treatment of age-related comorbidities. Similarly, sex differences in cardiovascular outcomes have been extensively studied, with women generally showing better outcomes than men (10). However, this study did not find a significant association between sex and EF after PCI. This may reflect advances in the treatment of female STEMI patients. Additionally, SUA levels have been implicated in cardiovascular disease progression, including adverse left ventricular remodeling and impaired cardiac function (4, 11). However, our study did not find a significant association between SUA levels and EF post-PCI. This lack of association may be attributed to the complex interplay between SUA levels and other pathophysiological mechanisms in STEMI. The present study examined the correlation between SUA levels and EF in patients with acute STEMI undergoing PCI. However, previous research has suggested a potential link between elevated SUA levels and adverse cardiovascular disease outcomes (12). The relationship between SUA levels and EF post-PCI in STEMI patients is still not well understood. Our analysis has revealed a weak negative correlation between EF and SUA levels in male patients, but this association did not reach statistical significance. Conversely, in female patients, there was a weak positive correlation between EF and SUA levels, but this correlation also did not reach statistical significanceBased on these findings, it can be suggested that SUA levels and EF post-PCI may be attributed to several underlying mechanisms. Elevated SUA levels are linked to oxidative stress, inflammation, and endothelial dysfunction, all contributing to myocardial injury and adverse left ventricular remodeling. Additionally, SUA has been shown to promote the formation of reactive oxygen species and impair nitric oxide bioavailability, further exacerbating myocardial damage and compromising cardiac function (13, 14).Previous studies have shown a significant association between elevated SUA levels and reduced EF in STEMI patients undergoing PCI (15, 16).Our study partially supports this observation revealing a statistically significant weak negative correlation between SUA levels and EF overall. In contrast, some studies did not find a significant association between SUA levels and EF in patients with acute myocardial infarction (17). This variability in findings across studies may also be attributed to patient demographics and comorbid conditions. Despite valuable insights provided by our study, several limitations should be acknowledged. Firstly, the small sample size and a single-center study may have resulted in limited statistical power and generalizability. Secondly variations in the timing of

SUA and EF measurements post-PCI could introduce data variability. Additionally, our study was a cross-sectional study with a limited sample size that cannot investigate causality.

Conclusion

Our study provides significant insights into the relationship between SUA levels and EF in patients with acute STEMI treated with PCI. Despite research suggesting a potential adverse impact of elevated SUA levels on cardiac function, our findings revealed a weak but statistically significant overall negative correlation between SUA and EF. However, there were no significant associations in the gender-specific analyses. The comparison with previous studies indicates consistency and discrepancies, emphasizing the necessity for cohort studies to aim to incorporate larger and more diverse populations.

Acknowledgements

We are grateful to the management of Afshar Hospital for their cooperation in conducting this study.

Conflicts of Interest

The authors declare no conflict of interest.

Funding/Support

None

Ethical statements

This study was approved by the ethical committee of Shahid Sadoughi University of Medical Sciences (IR.SSU.MEDICINE.REC. 1397.082), and written informed consent was obtained from all patients before the study.

References

1.Thygesen K, Alpert JS, White HD, et al. Universal definition of myocardial infarction.*Circulation*. 2007; 116(22):2634-53.
2.Tang N, Chen X, Li K, et al. Myocardial Perfusion in ST-Segment Elevation Myocardial Infarction Patients After Percutaneous Coronary Intervention: Influencing Factors and Intervention Strategies.*Cureus*. 2023;15(8): e42841.
3.Wang R, Mei B, Liao X, et al. Determination of risk factors affecting the in-hospital prognosis of patients with acute ST segment elevation myocardial infarction after percutaneous coronary intervention.*BMC Cardiovasc Disord*.2017;17(1):243.
4.Wu AH, Gladden JD, Ahmed M, et al. Relation of serum uric acid to cardiovascular disease. *Int J Cardiol*. 2016;213:4-7.
5.Nieto FJ, Iribarren C, Gross MD, et al. Uric acid and serum antioxidant capacity: a reaction to atherosclerosis?*Atherosclerosis*. 2000;148(1):131-9.
6.Cox P, Gupta S, Zhao S, et al. The incidence and prevalence of cardiovascular diseases in gout: a systematic review and meta-analysis. *Rheumatol Int*. 2021;41(7):1209-1219.
7.Strasak A, Ruttmann E, Brant L, et al. Serum Uric Acid and Risk of Cardiovascular Mortality: A Prospective Long-Term Study of 83 683 Austrian Men .*Clin Chem*.2008;54(2):273-84.
8.Yan F, Zhang Y, Pan Y, et al. Prevalence and associated factors of mortality after percutaneous coronary intervention for adult patients with ST-elevation myocardial infarction:A systematic review and meta-analysis.*J Res Med Sci*. 2023;28:17.
9.Tanik VO, Cinar T, Arugaslan E, et al. The Predictive Value of PRECISE-DAPT Score for In-Hospital Mortality in Patients With ST-Elevation Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention. *Angiology*. 2019;70(5):440-447.
10.Regitz-Zagrosek V, Gebhard C.Gender medicine: effects of sex and gender on cardiovascular disease manifestation and outcomes. *Nat Rev Cardiol*. 2023;20(4):236-247.
11.Feig DI, Kang DH, Johnson RJ. Uric acid and cardiovascular risk. *N Engl J Med*. 2008;359(17):1811-21.
12.Yamamoto H, Nagatomo Y, Mahara K, et al. In-Hospital Serum Uric Acid Change Predicts Adverse Outcome in Patients With Heart Failure. *J Card Fail*. 2020;26(11):968-976.
13.Hsu PF, Chuang SY, Yu WC, et al. The Impacts of Serum Uric Acid on arterial hemodynamics and Cardiovascular Risks. *Acta Cardiol Sin*. 2013;29(2): 142-50.
14.Münzel T, Camici GG, Maack C, et al. Impact of Oxidative Stress on the Heart and Vasculature: Part 2 of a 3-Part Series. *J Am Coll Cardiol*.2017;70(2):212-229.
15.Ndrepepa G, Braun S, Haase HU, et al. Prognostic value of uric acid in patients with acute coronary syndromes. *Am J Cardiol*. 2012;109(9):1260-5.
16.Kojima S, Sakamoto T, Ishihara M, et al. Prognostic usefulness of serum uric acid after acute myocardial infarction (the Japanese Acute Coronary Syndrome Study). *Am J Cardiol*.2005; 96(4):489-95.
17.Kanbay M, Segal M, Afsar B, et al.The role of uric acid in the pathogenesis of human cardiovascular disease.*Heart*.2013;99(11):759-766.

