International Journal of Biology and Life Sciences
ISSN: 2957-9511 | Vol. 5, No. 3, 2024

Correlation Study between Body Mass Index and Serum Uric Acid Level in Patients with ST-segment Elevation Myocardial Infarction

Yinyan He *, Weiwei Qin, Ruiting Yang, Xianjing Shang, Ruihong Yang, Yufei Xu

Affiliated Hospital of Jining Medical University, Jining, Shandong, China

*** Corresponding author:** Yinyan He (Email: 1024668353@qq.com)

Abstract: Background Although significant progress has been made in the treatment of acute ST-segment elevation myocardial infarction, acute myocardial infarction remains a common cause of heart failure, and the correlation between body mass index and serum uric acid levels in patients with acute ST-segment elevation myocardial infarction is unclear. Objective To analyze the relationship between body mass index and serum uric acid levels in hospitalized patients with acute ST-segment elevation myocardial infarction and to preliminarily investigate whether body mass index is independently associated with serum uric acid levels in patients with myocardial infarction. Methods This was a retrospective study. Patients with acute ST-segment elevation myocardial infarction hospitalized in the Department of Cardiology from January 2022 to December 2022 were selected. General information gender, age, height, weight, blood pressure, body mass index, history and related metabolic indexes were collected and recorded, and Spearman correlation analysis was used to analyze the correlation between body mass index and serum uric acid, and to analyze the independent risk factors of patients with acute ST-segment elevation myocardial infarction according to linear regression. Results A total of 875 patients with acute ST-segment elevation myocardial infarction were collected in this study, with a mean age of (62.22±12.06) years, of which 73.6% were men and 26.4% were women. The mean body mass index was (25.00±3.93) kg/m2 and the mean serum uric acid was (297.72±95.37) μmol/l. Spearman's correlation analysis showed a negative correlation between age, left ventricular ejection fraction, and serum uric acid, and a significant positive correlation between cardiac function, triglycerides, body mass index, and serum uric acid in patients with acute ST-segment elevation myocardial infarction. significant positive correlation. Linear regression models showed that gender, hypertension, cardiac function, triglycerides, left ventricular ejection fraction and body mass index were independent risk factors for serum uric acid in patients with acute ST-segment elevation myocardial infarction. Conclusion Body mass index is associated with the development and progression of hyperuricemia in patients with acute ST-segment elevation myocardial infarction, and the higher the body mass index, the higher the serum uric acid level in patients with acute ST-segment elevation myocardial infarction. Clinical weight management is needed for patients with acute ST-segment elevation myocardial infarction, and patients with acute ST-segment elevation myocardial infarction who have a normal body mass index are encouraged to maintain their current weight.

Keywords: Myocardial Infarction; Body Mass Index; Serum Uric Acid.

1. Introduction

Acute ST-segment elevation myocardial infarction (STEMI), a manifestation of acute coronary syndrome (ACS), leads to sudden death due to hemodynamic disturbances, and the optimal therapeutic strategy to minimize the risk of sudden death in patients who develop ventricular tachycardia (VT) or ventricular fibrillation (VF) during or early thereafter in STEMI is not yet fully understood [1]. Today, myocardial infarction (MI) remains the most common cause of heart failure (HF), despite significant advances in the treatment of coronary artery disease and acute myocardial infarction (AMI) [2]. Studies have shown that most STEMIs are caused by underlying coronary artery disease [3], and metabolic factors such as body mass index (BMI) and serum uric acid (SUA) are risk factors for the occurrence or exacerbation of STEMIs, in addition to risk factors such as hypertension and hyperlipidemia [4-6].The Framinghamy study [7] found that, after 32 years of followup, the greater variability in BMI population had a much higher incidence of cardiovascular events than those with relatively stable BMI variability; a study by Cai Zefeng et al [8] showed that BMI variability was an independent risk factor for the risk of cardiovascular events, and that the risk of cardiovascular events increased with the increase in BMI variability. In addition, SUA, which is a purine metabolite, has also been shown to be an independent risk factor for cardiovascular disease and is closely related to the prognosis of STEMI patients [9], and can be used as an important indicator of the short-term prognosis of STEMI patients [10]. However, the relationship between BMI and SUA levels in STEMI patients is yet to be verified. Based on this, this study investigates whether BMI is an independent risk factor for SUA levels in STEMI patients, aiming to provide clinical data support.

2. Information and Methods

2.1. Study Design

This is a retrospective study to investigate the relationship between BMI and SUA levels in STEMI patients. In this study, BMI of STEMI patients was used as the independent variable and SUA level of STEMI patients was the dependent variable.

2.2. Study Subjects

1.2According to the electronic medical record system of our hospital, 875 patients with STEMI admitted to the cardiology department of our hospital from January 2022 to December 2022 were selected, and the screening of such patients was consecutive. Inclusion criteria:(1) STEMI met the diagnostic criteria in the Diagnostic and Therapeutic Guidelines for Acute ST-Segment Elevation Myocardial Infarction (2019); (2) onset time <24h;(3) no medication affecting the uric acid level was used. Exclusion Criteria: (1) other cardiovascular diseases such as pulmonary heart disease and congenital heart disease; (2) anemia and hyperthyroidism; (3) coagulation disorders or bleeding tendency; (4) severe hepatic and renal insufficiency; (5) recent stroke and cerebral hemorrhage; and (6) allergy to antiplatelet drugs or contrast media.

2.3. Ethics

All the procedures of this study were in accordance with ethical standards. In addition, the methodology of this study complied with the guidelines.

2.4. Main Variables Included

The consultation information of the patients was reviewed from the electronic medical record system of the hospital, including the patients' age, gender, height, weight, BMI, smoking history, drinking history, hypertension, diabetes mellitus, Systolic Blood Pressure (SBP), Diastolic Blood Pressure (DBP), Left Ventricular Ejection Fraction (LVEF), triglycerides, total cholesterol, lipoproteins, SUA.

2.5. Data Collection

Nurses collected clinical information from patients, and standardized measurement criteria were used for SBP and DBP. For blood pressure measurement, patients were placed in the supine position with the blood pressure cuff at the same level as the heart, rested for 10 minutes, and took three measurements, with the average of the three measurements taken as the final blood pressure value. For height and weight measurements, patients removed their coats and shoes and wore only light clothing. Body mass index (BMI) was defined as weight in kilograms divided by the square of height in meters (kg/m^2). On the morning of the second day of admission, the nurse collected fasting blood from the patient (after 8 to 12 hours of fasting) to collect laboratory indices. A cardiac ultrasound was completed by a qualified sonographer and the patient's LVEF was collected.

2.6. Statistical Methods

Data were statistically analyzed using BIM SPSS 26.0 software. In this study, all normal-distributed measurements were expressed as mean ± standard deviation with t-test; all non-normal-distributed measurements were expressed as median (lower quartile ~ upper quartile) [M (P_{25}, P_{75})] with non-parametric test; counts were expressed as percentage with χ^2 test or Fisher's exact test. Spearman's analysis used to compare the correlation of BMI and other metrics with SUA, and bias correlation was used to control for linear correlation after other confounders. Independent influences on SUA were also analyzed using multivariate linear analysis. Differences were considered statistically significant when $p < 0.05$.

3. Results

3.1. Baseline Characteristics of Participants

Table 1. Baseline Characteristics of Participants(n=875)

Parameter		$\bar{x} \pm s$ */n (%)*
Gender	Female	231(26.4)
	Male	644(73.6)
Infarct site	Lower wall	357(40.8)
	Lateral wall	20(2.29)
	Front wall	382(43.66)
	Anterior intermediate wall	47(5.37)
	Wide anterior wall	69(7.89)
Cardiac function	Grade I	703(80.34)
	Grade II	110(12.57)
	Grade III	31(3.54)
	Grade IV	31(3.54)
History of alcohol consumption	No	473(54.06)
	Yes	402(45.94)
Smoking history	No	370(42.29)
	Yes	505(57.71)
High blood pressure	No	502(57.37)
	Yes	373(42.63)
Diabetes mellitus	No	680(77.71)
	Yes	195(22.29)
Age		62.221±12.056
LVEF		0.493±0.086
Height		166.607±8.042
Weight		69.710±13.109
BMI		24.997±3.925
SBP		125.336±22.480
DBP		79.129±14.742
Lipoprotein		257.114±237.571
Triglycerides		1.523±1.314
Total Cholesterol		4.302±1.043
SUA		297.722±95.368

After screening, a total of 875 STEMI patients were included, including 644 (73.6%) males and 231 (26.4%) females; the mean age was (62.221±12.056) years; the mean BMI was (24.997±3.925) kg/m2; the mean SUA was (297.722±95.368) ummol/L; according to the different infarct sites of STEMI patients, they were categorized into lower wall, lateral wall, anterior wall, anterior interstitial wall and extensive anterior wall, of which the number of lower wall and anterior wall was higher, accounting for 40.8% and 43.66% of the total number of patients, respectively; according to the cardiac function Killip grading criteria, the participants were categorized into I, II, III and IV in total, and 80.34% of the participants were mainly in the cardiac function class I. The details are shown in Table 1.

3.2. Spearman Correlation Coefficients between SUA and Each Parameter

Spearman correlation analysis was used to study the correlations between age, cardiac function, LVEF, SBP, DBP, lipoproteins, triglycerides, total cholesterol, and BMI with SUA, respectively, and the Spearman correlation coefficients were used to indicate the strength of the correlations. Specific analyses showed that: age, LVEF and SUA showed significant ($p < 0.05$) and negative correlations; cardiac function, triglycerides, BMI and SUA showed significant ($p < 0.05$) and positive correlations; and SBP, DBP, lipoproteins, total cholesterol and SUA did not show significant ($p > 0.05$) and no correlations. there is no correlation. The details are shown in Table 2.

Table 2. Spearman Correlation Coefficients Between SUA and all Parameters(n=875)

		1	2	3	4	5	6	7	8	9	10	11	12
SUA	r	1											
	p	-											
Age	r	-0.078*	1										
	p	0.022	-										
Cardiac function	r	0.159**	0.218**	1									
	p	0.000	0.000	-									
LVEF	r	-0.093**	-0.220**	-0.273**	1								
	p	0.006	0.000	0.000	-								
Height	r	0.220**	-0.349**	-0.087*	0.056	1							
	p	0.000	0.000	0.010	0.100	-							
Weight	r	0.238**	-0.431**	-0.069*	0.054	0.591**	1						
	p	0.000	0.000	0.040	0.111	0.000	-						
DBP	r	-0.010	-0.197**	-0.069*	0.003	0.077*	0.177**	1					
	p	0.761	0.000	0.041	0.936	0.023	0.000	-					
SDP	r	-0.035	-0.049	-0.079*	0.081*	-0.029	0.085*	0.795**	1				
	p	0.305	0.145	0.019	0.016	0.399	0.012	0.000	-				
Lipoprotein	r	-0.058	0.132**	0.080*	-0.059	-0.101**	-0.114**	0.011	0.031	1			
	p	0.085	0.000	0.019	0.080	0.003	0.001	0.744	0.353	-			
Triglycerides	r	0.201**	-0.242**	-0.079*	0.047	0.080*	0.305**	0.111**	0.086*	-0.131**	1		
	p	0.000	0.000	0.019	0.168	0.018	0.000	0.001	0.011	0.000	-		
Total Cholesterol	r	0.054	-0.131**	-0.094**	-0.030	-0.076*	0.022	0.138**	0.089**	0.087*	0.341**	1	
	p	0.112	0.000	0.005	0.379	0.024	0.511	0.000	0.009	0.010	0.000	-	
BMI	r	0.162**	-0.308**	-0.022	0.033	0.127**	0.843**	0.147**	0.118**	-0.080*	0.330**	0.079*	1
	p	0.000	0.000	0.521	0.329	0.000	0.000	0.000	0.000	0.018	0.000	0.019	-

* p<0.05, ** p<0.01

SUA is serum uric acid; LVEF is left ventricular ejection fraction; DBP is diastolic blood pressure; SBP is systolic blood pressure; BMI is body mass index; 1 is uric acid; 2 is age; 3 is cardiac function; 4 is left ventricular ejection fraction; 5 is height; 6 is body weight; 7 is diastolic blood pressure; 8 is systolic blood pressure; 9 is lipoprotein; 10 is triglyceride; 11 is total cholesterol; and 12 is body mass index.

3.3. Non-parametric Tests of SUA and Parameters

Summarizing the analysis, using the MannWhitney test statistic, the analysis showed that: gender, history of alcohol consumption, history of smoking, and history of hypertension all showed significant differences for SUA ($p<0.05$), and history of diabetes was not statistically different for SUA ($p>0.05$); using the Kruskal-Wallis test statistic for analysis, the analysis showed that none of the different infarct sites would exhibit significant differences for SUA ($p > 0.05$). The details are shown in Table 3.

Table 3. Nonparametric Teats Between SUA and all Parameters(n=875)

Parameter		M (P_{25}, P_{75})	U	Z	H	p
Gender	Female	250.000(197.0,325.0)	54397.500	-6.064	-	0.000**
	Male	297.000(244.3,352.0)				
History of alcohol consumption	No	281.000(218.0,341.5)	80427.000	-3.931	-	0.000**
	Yes	299.000(247.0,353.5)				
History of smoking	No	275.000(217.0,340.0)	78994.500	-3.907	-	0.000**
	Yes	298.000(241.5,352.8)				
History of high blood pressure	No	285.000(231.0,343.0)	85285.000	-2.255	-	0.024*
	Yes	299.000(235.0,356.0)				
History of diabetes	No	291.600(237.3,349.0)	62780.000	-1.131	-	0.258
	Yes	280.000(219.0,353.0)				
Site of infarction	Front wall	288.500(232.0,350.3)	-	-	9.464	0.050
	Extensive anterior wall	320.000(271.5,384.5)				
	Anterior interstitial wall	291.000(220.0,316.0)				
	Lateral wall	283.000(245.8,317.3)				
	Lower wall	286.000(230.0,345.0)				
* *p*<0.05, ** *p*<0.01						

3.4. Multivariate Linear Results of SUA in STEMI Patients

Table 4. Multiple linear analysis of SUA in patients with STEMI(n=875)

	Unstandardized coefficient		Standardized coefficient	t	p	covariance diagnosis	
	B	standard error	Beta			VIF	tolerance level
Constant	246.438	39.551	-	6.231	0.000**	-	-
Gender	32.726	9.120	0.151	3.589	0.000**	1.766	0.566
Age	-0.241	0.290	-0.030	-0.830	0.407	1.333	0.750
LVEF	-134.473	37.673	-0.121	-3.569	0.000**	1.142	0.876
History of alcohol consumption	2.899	7.534	0.015	0.385	0.700	1.540	0.649
History of smoking	3.173	8.124	0.016	0.391	0.696	1.760	0.568
Hypertension	12.602	6.272	0.065	2.009	0.045*	1.051	0.951
Cardiac Function	27.807	4.584	0.205	6.066	0.000**	1.135	0.881
Triglycerides	9.894	2.398	0.136	4.126	0.000**	1.084	0.923
BMI	1.944	0.828	0.080	2.349	0.019*	1.152	0.868
R^2	0.129						
Adjusted R^2	0.119						
F	F (9,865) =14.176, p=0.000						
Dependent variable: uric acid							
*p<0.05, **p<0.01 LVEF is left ventricular ejection fraction; BMI is body mass index							

According to the summarized analyses in Tables 2 and 3, the analyses showed that gender, age, LVEF, history of alcohol consumption, history of smoking, history of hypertension, cardiac function, triglycerides, and BMI were the suspected influencing factors of SUA. For further analysis, gender, age, LVEF, history of drinking, history of smoking, history of hypertension, cardiac function, triglycerides, and BMI were used as independent variables, while SUA was used as the dependent variable in a linear regression analysis, with the model equation of SUA=246.438+32.726*Gender-0.241* Age-134.473*LVEF+2.899*History of drinking+ 3.173* History of smoking+ 12.602*High blood pressure+ 27.807*Cardiac function+ 9.894*Triglycerides+ 1.944*BMI, the R2 value of the model was 0.129 implying that gender, age, LVEF, history of drinking, history of smoking, history of high blood pressure, cardiac function, triglycerides, and BMI explained 12.9% of the variation in SUA. The F-test of the model found that the model passed the F-test (F=14.176, p=0.000<0.05), which means that at least one of the gender, age, LVEF, history of alcohol consumption, history of smoking, hypertension, cardiac function, triglycerides, and BMI could have an effect on the SUA, and in addition, the test of the model for multicollinearity found that all the values of the VIF in the model were less than 5 which implies that there is no covariance problem; finally, the specific analysis shows that: gender (B=32.726,t=3.589,p=0.000<0.01), hypertension (B=12.602,t=2.009, p=0.045<0.05), cardiac function (B=27.807,t=6.066,p=0.000<0.01), triglycerides (B=9.894, t=4.126,p=0.000<0.01), and BMI (B=1.944, t=2.349, p=0.019<0.05) all had a significant positive effect on SUA; LVEF (B=-134.473,t=-3.569,p=0.000<0.01) had a significant negative influence relationship; there was no statistically significant relationship between age, drinking history, smoking history and SUA (p>0.05). In summary, gender, hypertension, cardiac function, triglyceride, BMI, and LVEF were independent influences on SUA in the STEMI population, but age, drinking history, and smoking history were not independent influences on SUA. The details are shown in Table 4.

4. Discussion

BMI has long been recognized as an indicator of obesity assessment with predictive value for various chronic diseases and associated tissue and organ dysfunction [11]. The prevalence of obesity and CVD has been increasing over the past decades. The presence of obesity and CVDs together can exacerbate the progression of the disease, increasing health and financial burdens and creating new challenging situations for the chronic disease prevention and treatment. In addition, studies have confirmed that it is an independent risk factor for cardiovascular disease, and the risk of sudden death in obese patients is quite high even in the absence of organic heart disease, which may be related to the metabolic syndrome [12]. Several prospective studies [13] have shown that hyperuricemia (HUA) is an independent risk factor for obesity, that obese individuals have significant higher levels of SUA, a component of the metabolic syndrome, compared to normal weight individuals, and that obese individuals are also at higher risk for HUA, both of these, in turn, increase the risk of heart disease and metabolic syndrome [14].

A number of earlier studies had attempted to determine the effect of SUA on the development of CVD, but the results were conflicting. In a long-term observational study [15], researchers evaluated the impact of SUA levels in STEMI patients and found that high uric acid (UA) levels were a significant factor in increasing mortality in STEMI patients. Elevated concentrations of UA not only lead to the deposition of sodium urate crystals in periarticular tissues, but also within the walls of blood vessels, and the presence of these deposits in the blood vessel walls has the consequence of the presence of these deposits in the vascular wall is damage to the vascular endothelium, which promotes atherosclerotic lesions and leads to the development of ACS. In a recent study [16] from Pakistan, it was confirmed that in patients with AMI, SUA levels were higher in the test group than in the control group and the number of patients with HUA was also higher than in the control group. HUA has been linked to the development of other diseases which are known risk factors for ACS such as hypertension, dyslipidemia, obesity, metabolic syndrome and chronic kidney disease. The importance of UA in hypertension is reflected in the guidelines of the European Society of Hypertension [17], which recommend testing SUA levels as a routine laboratory test in hypertensive patients, which is consistent with the findings of the present study. Moreover, in the present study, a positive correlation was observed between high SUA levels and triglyceride levels. This result is in agreement with the findings of Hajizadeh et al, Tuomilehto et al and Nagahama et al [18-20]. Most of the studies [18,21-22] have demonstrated that high SUA levels increase mortality in AMI patients, especially in STEMI patients. A meta-analysis conducted by He et al [23] in 2019 noted a 1.5-fold increased risk of cardiovascular mortality in ACS patients with HUA. In addition, Mora-Ramírez et al [24] reported that high SUA levels at admission increased short-term mortality in STEMI in patients who have high cardiovascular risk factors.

In the present study, the findings revealed a positive correlation between BMI and SUA levels in STEMI patients, i.e., as BMI increased, SUA gradually increased in STEMI patients. This is consistent with the findings of Yao et al [25]; furthermore, Yao et al noted that this positive correlation existed in any gender and ethnicity, which is consistent with earlier epidemiologic and clinical evidence of a significant positive correlation between obesity and SUA in Chinese, Japanese, Indian, Pakistani, Iraqi, and Bangladeshi populations [26-28]. Because of the close biological association between obesity and SUA, close evaluation of the interaction between SUA and obesity is essential for preventive medicine. In addition, BMI control contributes to the management and treatment of HUA and cardiovascular disease.

This research has some limits as well. (1) The study was a single-center study that included only STEMI patients from a third-level hospital in southwestern Shandong Province, and there may be bias caused by regional or ethnic factors, and this may have certain limits on the applicability of the study outcomes externally, and whether the study conclusions can be generalized to other regions requires further research and testing, and a multicenter large-sample study can be conducted in the future to further validate the validity and Future multi-center large-sample studies can be conducted to further validate the validity and generalizability of this study and provide further suggestions for improvement; (2) The study was retrospective, that is, the results of the study could not clearly state the causal relationship, and the patients were not followed up. The cross-sectional relationship between BMI and SUA levels in STEMI patients can be further explored in the future with long-term follow-up and tracking.

5. Conclusion

BMI of STEMI patients is an independent risk factor affecting the level of SUA, and it is significantly and positively correlated with the level of SUA, i.e., the increase in BMI, the increase in the level of SUA in STEMI patients. Based on this, healthcare professionals should encourage STEMI patients with normal BMI to maintain their current body weight, while for STEMI patients with BMI outside the normal range, there is a need to consider multiple clinical perspectives, evaluate them in a timely manner, and adopt proactive strategies for the prevention and treatment of metabolic problems related to obesity, especially metabolic disorders involving SUA, and guide patients to maintain a healthy body weight.

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