The Egyptian Journal of Hospital Medicine (January 2023) Vol. 90, Page 1069-1074

Association of Monocyte to High-Density Lipoprotein Ratio with Contrast-Induced Nephropathy in ST-Segment Elevation Myocardial Infarction Patients Treated with Primary Percutaneous Coronary Intervention

Ahmed A. Emara, Ahmed A. Elhelbawy*, Neveen I. Samy
Cardiology Department, Faculty of Medicine, Menoufia University, Shibin El-Kom, Menoufia, Egypt
***Corresponding Author:** Ahmed Abd Elsamie Elhelbawy, **Mobile**: (+20) 01017853369,
E-mail: ahmedhelbawy.com@gmail.com

ABSTRACT

Background: Following primary percutaneous coronary intervention (PCI), contrast-induced nephropathy (CIN) is linked to higher mortality and morbidity. The monocyte to high density lipoprotein cholesterol ratio (MHR), which has been used as a novel predictive marker in patients with cardiovascular and renal disorders, has been linked in several studies to a number of negative cardiovascular outcomes.

Objective: The current study was conducted to explore correlation between monocyte to HDL ratio and CIN after primary PCI in patients presented with ST-segment Elevation Myocardial Infarction (STEMI).

Patients and methods: A total of 80 patients with acute STEMI were included. Patients were differentiated into two groups according to development of contrast induced nephropathy; *Group (A)* Patients who developed contrast induced nephropathy, and *Group (B)* Patients did not develop contrast induced nephropathy. CIN was defined as either a 25% increase in serum creatinine from baseline or 0.5 mg/dL increase in absolute value, within 48-72 h of intravenous administration of contrast medium.

Results: A total of 16 (20%) patients developed CIN. MHR was significantly higher in CIN positive group [mean 0.0202 (range 0.009-0.0633) 10^8/mg] than non-CIN group [mean 0.0129 (range 0.0031-0.0307) 10^8/mg] with P-value = 0.003. MHR was also significantly correlated with creatinine levels 48 hours after PCI (r_s: 0.322, P: 0.004). ROC statistical analysis showed that MHR >0.0131 was the best cutoff values for predicting contrast induced nephropathy with sensitivity 76% and specificity 71.2%.

Conclusion: Higher MHR levels may predict CIN development after primary PCI in STEMI patients.

Keywords: Percutaneous coronary intervention, Contrast-induced nephropathy, High density lipoprotein cholesterol ratio, Myocardial Infarction.

INTRODUCTION

One of the significant side effects of primary PCI that is linked to higher mortality and morbidity is contrast-induced nephropathy (CIN) [1,2,3]. To enable preventative actions and enhance therapeutic outcomes, it is crucial to identify and intervene early in acute coronary syndrome (ACS) patients who are at a high risk of CIN.

In patients with ST-segment Elevation Myocardial Infarction (STEMI) within 12 hours of the start of symptoms, primary PCI is the preferable reperfusion technique, if it can be completed quickly (i.e., within 120 minutes of the STEMI diagnosis) by a skilled team. When a patient presents late (12-48 hours following the beginning of symptoms), a regular primary PCI approach should also be taken into account [4,5,6]. As pathophysiological causes of CIN, it has been suggested that vasoconstriction, oxidative stress, free radical damage, endothelial dysfunction, and inflammation [7,8].Age over 70, nephrotoxic drugs, impaired left ventricular systolic function, chronic kidney disease (CKD), and diabetes mellitus (DM) have all been identified as risk factors for the development of CIN [9,10].

There are several risk score models that have been created for CIN prediction. However, the majority of these models rely on characteristics that are unknown at admission [11,12,13]. To increase the early detection of people at risk for CIN, more objective measures are required.

A new laboratory measure called the monocyte to HDL-cholesterol ratio (MHR) has a predictive significance in renal and cardiovascular patients' disorders since it is associated with inflammation [14].

The current study was conducted to explore correlation between monocyte to HDL ratio and CIN after primary PCI in patients presented with STEMI.

PATIENTS AND METHODS

This is a case control study that included 80 patients with acute STEMI admitted to Menoufia University Hospital and Nasser Institute Hospital who underwent primary PCI from May 2019 to September 2021.

According to the definition CIN [15] as a 25% increase in serum creatinine from baseline or a 0.5 mg/dl increase in absolute value within 48-72 hours of intravenous injection of contrast media. Patients were divided into two groups and placed in the following treatment groups: Patients in *Group A* experienced contrast-induced nephropathy, but patients in *Group B* did not have such a condition.

Because they had all been diagnosed with acute STEMI, all research participants were eligible for primary PCI. In the absence of LV hypertrophy or LBBB, the following ECG abnormalities are indicative

Received: 28/07/2022
Accepted: 30/09/2022

of ischemia: at least two continuous leads with ST-segment elevation of 2.5 mm in 40-year-old males, 2 mm in 40-year-old men, 1.5 mm in 40-year-old women, and/or 1 mm in the other leads. In the same way as ST-segment depression in leads V1-V3 indicates myocardial ischemia, concomitant ST-segment elevation of 0.5 mm in leads V7-V9 should be considered to detect posterior MI. This is especially true when the terminal T-wave is positive (ST-segment elevation equivalent) [16,17,18].

Patients who had recently undergone a cardiac arrest, had contrast material supplied within the previous 10 days, required dialysis due to end-stage renal failure, had cancer, or were on lipid-lowering medicines were excluded from the research. A complete medical history was collected, as well as past medical records and medicines, to rule out any chronic or acute inflammatory illnesses that may affect the laboratory results.

Age, gender, and coronary artery disease risk factors such as diabetes, hypertension, and smoking-defined as a current or former smoker-were all taken into consideration when taking the patient's history (defined as quit smoking in the past 6 months), comprehensive examination of chest pain, focusing on duration (pain to first medical contact time, first medical contact to intervention), History points to a recent illness that might impact the number of monocytes (excluded from the study). A family history of coronary artery disease (CAD) was defined as sudden cardiac death or premature CAD in a first-degree relative before the ages of 55 for males and 65 for women. Prior PCI, MI, or coronary artery-bypass graft procedures (CABG).

A focus on the arterial blood pressure (ABP), heart rate, neck veins, cardiac auscultation to identify mechanical complications, and chest auscultation to identify tiny basal crepitation were all parts of the clinical examination. Additionally, a 12-lead surface ECG was performed to look for acute STEMI (as described before).

Within the first 24 hours of admission, fasting blood samples were taken to determine the lipid profile and HDL level. On admission, 24 hours later, and 48 hours later, the creatinine level was assessed (reference range: 0.4 - 1.4 mg/dl). Complete blood count (CBC) testing was also carried out. By dividing the monocyte count (10^9/L) by the HDL-C level (mg/dl), the monocyte to HDL-C ratio was computed and reported as 108/mg. All patients who had transthoracic echocardiography following PPCI (during their hospital stay) had their left ventricular ejection fraction (LVEF) assessed using 2D (eyeball) and M-mood. The risk score for Mehran was determined [9].

All patients had primary percutaneous coronary intervention (PPCI) in the manner described below: All patients received 10,000 units of heparin, 300 mg of aspirin, 180 mg of ticagrelor, or 600 mg of clopidogrel. The femoral artery was punctured using the Seldinger method, and each coronary artery was examined from at least two angles. Diagnostic catheter was utilised to perform coronary angiography and identify the responsible artery (low osmolar nonionic contrast medium either iohexol or lopromide was used). The offending vessel was mended after additional coronaries were examined. Also estimated was the quantity of contrast used.

Monocyte to high-density lipoprotein ratio (MHR) and the onset of contrast-induced nephropathy are important outcomes.

Ethical approval:

This study was ethically approved by the Institutional Review Board of the Faculty of Medicine, Menoufia University. Written informed consent was obtained from all participants. This study was executed according to the code of ethics of the World Medical Association (Declaration of Helsinki) for studies on humans.

Statistical analysis

The collected data were introduced and statistically analyzed by utilizing the Statistical Package for Social Sciences (SPSS Inc., Chicago, Illinois, USA) version 20 for windows. Qualitative data were defined as numbers and percentages. Chi-Square test and Fisher's exact test were used for comparison between categorical variables as appropriate.

Quantitative data were tested for normality by Kolmogorov-Smirnov test. Normal distribution of variables was described as means and SD. For skewed data, the median (Med) and range were used to express quantitative variables. Independent sample t-test/Mann-Whitney U-test was used for comparison between groups. With non-parametric quantitative data, Spearman's correlation was utilized to examine the relationship between two variables. P value ≤ 0.05 was considered to be statistically significant.

RESULTS

The study was carried out on 80 patients; 16 (20%) patients developed CIN while 64 (80%) patients did not develop CIN. Demographic data, clinical history and risk factors of the studied groups were demonstrated in **Table 1** and showed that age was significantly higher in *Group A* (mean 56.81 ± 9.85) years than in *Group B* (mean 49.78 ± 10.66) years (P value <0.05).

Table (1): Comparison of the demographic data, Clinical history and risk factor witin the study groups according to development of CIN.

Variable	Group A (CIN) (N= 16)	Group B (No CIN) (N= 64)	Test of sig.
Age (years)	56.81 ± 9.85	49.78 ± 10.66	t= 2.394 P= 0.019*
Sex			
Males	13 (81.2%)	58 (90.6%)	χ^2/FET= 1.127 P= 0.288
Females	3 (18.8%)	6 (9.4%)	
BMI (Kg/m^2)	27.25 ± 2.01	26.64 ± 1.78	t= 1.159 P= 0.236
DM	8 (50%)	14 (21.9%)	χ^2/FET= 5.078 P= 0.024*
HTN	9 (56.2%)	16 (25%)	χ^2/FET= 5.818 P= 0.016*
Smoking	9 (56.2%)	42 (65.6%)	χ^2/FET= 0.487 P= 0.485
Previous MI	0 (0%)	1 (1.6%)	χ^2/FET= 0.253 P= 0.615

χ^2/FET =Chi-square test/Fischer's exact test. BMI: body mass index, DM: diabetes mellitus, HTN: hypertension, MI: myocardial infarction.

Vital signs, contrast amount, MERHAN risk score and echocardiographic data within the study were demonstrated in **Table 2**.

Table (2): Comparison of the Vital signs, contrast amount, MERHAN risk score and echocardiographic data within the study groups according to development of CIN.

Variable	Group A (CIN) (N= 16)	Group B (No CIN) (N= 64)	Test of sig.
SBP (mmHg)	111.25 ± 14.08	123.28 ± 12.92	t= -3.274 P= 0.002*
DBP (mmHg)	70 ± 9.49	75.23 ± 7.89	t= -2.278 P= 0.025*
Contrast amount (ml)	225.88 ± 21.52	206.09 ± 34.90	t= 2.161 P= 0.034*
MERHAN risk score	7 (2-14)	2 (1-10)	U= -3.076 P= 0.002*
EF (%)	40.75 ± 7.71%	44.05 ± 5.83%	t= -2.293 P= 0.025*

Median and range: non-parametric test, U: Mann-Whitney U-test, t: Independent samples t-test,*: Statistically significant (P<0.05), SBP: systolic blood pressure, DBP: diastolic blood pressure, EF: ejection fraction.

Laboratory data in the studied groups are demonstrated in **Table 3**.

Table (3): Comparison of the laboratory data within the study groups according to development of CIN.

Variable	Group A (CIN) (N= 16)	Group B (No CIN) (N= 64)	Test of sig.
Serum creatinine (admission day) (mg/dl)	0.65 (0.4-1.6)	0.7 (0.5-1.6)	z = -0.509, P = 0.611
Serum creatinine (after 24 hours) (mg/dl)	1 (0.6 -2)	0.7 (0.5-1.8)	z = -2.740, P = 0.006*
Serum creatinine (after 48 hours) (mg/dl)	1.7 (0.7-2.8)	0.7 (0.5-1.9)	z = -4.330, P < 0.001*
Haemoglobin (mg/dl)	13.42 ± 1.98	14.45 ± 1.84	t= -1.959, P = 0.048*
Platelets (10^3/fl)	245 (120-483)	231 (109-364)	z = -1.029, P = 0.304
WBCs (10^3/fl)	11.8 (4.8-22)	11.15 (5.1-23)	z = -0.818, P = 0.413
Monocytes (10^3/fl)	071 (0.38-1.78)	0.605 (0.17-1.05)	z = -1.953, P = 0.038*
Total cholesterol (mg/dL)	196 ± 36	194 ± 43	t= 0.195, P = 0.846
LDL (mg/dL)	130 ± 39	128 ± 11	t= 0.170, P = 0.866
HDL (mg/dL)	37.45 ± 5.66	45.80 ± 5.12	t= -3.360, P = 0.002*
Monocytes/HDL(10^8 /mg)	0.0202 (0.009-0.0633)	0.0129 (0.0031-0.0307)	z = -3.043, P = 0.003*

Median and range: non-parametric test, U: Mann-Whitney U-test, t: Independent samples t-test, *: Statistically significant (P<0.05), WBCs: White blood cells, LDL: Low-density lipoprotein, HDL: High-density lipoprotein.

Monocytes count was significantly higher in Group A [0.71 (0.38-1.78) (10^3/fl)] than in Group B [0.605 (0.17-1.05) (10^3/fl)] with P-value=0.038, while HDL level was significant lower in Group A [37.45 ± 8.66mg/dl] than in Group B [45.80 ± 8.12mg/dl] with P-value=0.002. Monocytes/HDL ratio was significant higher in Group A [0.0202 (0.009-0.0633) (10^8/mg)] than in Group B [0.0129 (0.0031-0.0307) (10^8/mg)] with P-value=0.003. There was a significant positive correlation between monocytes/HDL ratio and (creatinine after 48 hours) with P-value=0.004 and a significant negative correlation between monocytes/HDL ratio and EF with P-value=0.008, SBP with P-value=0.004 and DBP with P-value=0.04 (**Table 4**).

Table (4): Correlation between Monocytes/HDL and other variables in the study.

Variable		Monocyte /HDL
Age (years)	r_s	0.076
	P	0.501
BMI (kg)	r_s	0.186
	P	0.099
Creatinine admission day (mg/dl)	r_s	-0.047
	P	0.978
Creatinine after 48 hours (mg/dl)	r_s	0.185
	P	0.101
Creatinine after 48 hours (mg/dl)	r_s	0.322
	P	0.004*
Hemoglobin (mg/dl)	r_s	- 0.113
	P	0.320
Platelet (10^3/fl)	r_s	0.042
	P	0.709
WBC (10^3/fl)	r_s	0.190
	P	0.091
Total cholesterol (mg/dL)	r_s	0.095
	P	0.401
LDL (mg/dL)	r_s	-0.121
	P	0.287
Contrast amount (ml)	r_s	0.143
	P	0.204
EF (%)	r_s	-0.294
	P	0.008*
SBP (mmHg)	r_s	- 0.315
	P	0.004*
DBP (mmHg)	r_s	- 0.231
	P	0.040*

r_s: (Spearman correlation), *: Statistically significant (P<0.05), BMI: body mass index, WBCs: White blood cells, LDL: Low density lipoprotein, EF: Ejection fraction, SBP: Systolic blood pressure, DBP: Diastolic blood pressure.

ROC statistical analysis showed that MHR >**0.0131** was the best cutoff values for predicting contrast induced nephropathy with sensitivity **76%** and specificity **71.2%** [**Table 5 and Figure 1**].

Table (5): Predictive value of Monocytes/HDL in differentiating cases who developed CIN (N=16).

Diagnostic criteria	Monocytes/HDL
AUC	0.787
Cut off point	0.0131
P	0.003*
Sensitivity	76 %
Specificity	71.2 %
PPV	68.4 %
NPV	74.8 %
Accuracy	72.6 %

AUC: area under curve, PPV: Positive predictive value, NPV: Negative predictive value.

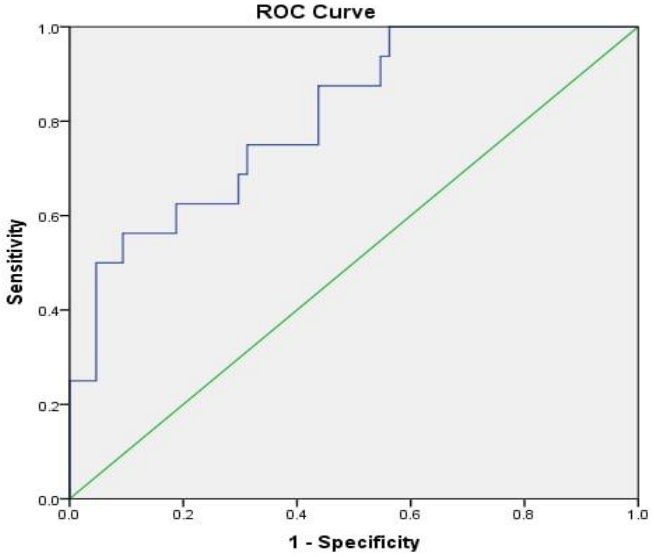

Figure (1): ROC curve of Monocytes/HDL in differentiating cases who developed CIN.

DISCUSSION

STEMI is a time-sensitive, life-threatening emergency. Effective care depends on an early, precise diagnosis and fast, percutaneous coronary intervention (PCI)-based treatment to restore coronary perfusion [4]. Following primary PCI, CIN is linked to higher mortality and morbidity [1].

There are several risk score models that have been created for CIN prediction. These models, however, often make use of characteristics that are unknown at enrollment [11,12]. To increase the early detection of people at risk for CIN, more objective measures are required.

Jiang *et al.* [14] study, which demonstrated the importance of MHR in clinical practice, found that MHR value was autonomously correlated with overall cardiovascular mortality in the general population, suggesting that MHR may be a potential predictor for identifying those who are more likely to have poor clinical outcomes. This study was conducted to explore correlation between monocyte to HDL ratio and contrast induced nephropathy after primary PCI in patients presented with STEMI & included 80 patients differentiated into two groups according to development of contrast-induced nephropathy

This study demonstrated that the results of a study by **Sa *et al.* [19]** who examined the relationship between MHR and CIN in 209 STEMI patients who underwent primary PCI showed that MHR was significantly higher in the CIN group [0.0202 (0.009-0.0633) 108/mg] than the non CIN group [0.0129 (0.0031-0.0307) 108/mg], with a P-value of PCI. With a p-value of 0.01, they observed that MHR was substantially greater in the CIN (+) group.

Additionally, this is consistent with a study by **Ulus *et al.* [20]** that looked at 674 patients with acute coronary syndrome receiving primary percutaneous coronary intervention to see if preprocedural MHR had a predictive role for the development of CIN. They discovered that preprocedural MHR may be used as a straightforward marker of CIN with a p-value of 0.001.

Which explains why, monocytes have a significant part in each step of atherosclerosis, from the development of foam cells in the sub-endothelial region through the instability of the fibrous cap and plaque rupture [21]. On the other hand, because it has anti-inflammatory, antioxidant, and reverse cholesterol transporter qualities, HDL-C exerts a variety of athero-protective benefits [22]. Inflammation may be a major factor in both the beginning and extension phases of CIN [23]. In theory, a higher monocyte count and a lower HDL-C level might signify uncontrolled inflammatory responses that could hasten atherosclerosis, raise the danger of un-favorable cardiovascular events, and raise the likelihood of CIN.

In contrast to our research, **Zehir *et al.* [23]** looked at the monocyte to HDL ratio as a potential predictor of CIN in patients who underwent primary PCI and had STEMI. They came to the conclusion that MHR cannot be a possible predictor of CIN development in patients with STEMI after discovering that it did not differ noticeably from that of stable patients at the early stage of AMI (12h). This might be as a result of the fact that fewer patients (6.6%) in this experiment developed CIN than in other trials; 10.8%, 15.5%, and 20%, respectively, in **Ulus *et al.* [20]** and **Sa *et al.* [19],** and our research.

In our study, by using statistical analysis we found that the MHR's optimum cutoff value for determining the likelihood of contrast-induced nephropathy is >0.0131 with sensitivity 76% and specificity 71.2%.

In the study done by **Ulus *et al.* [20]** the MHR cutoff value was 0.0174 with sensitivity and specificities of 65.7% and 67% respectively.

while in the study done by **Sağ *et al.* [19]** the MHR cutoff value was 0.025 with sensitivity and specificities of 75.1% and 74.9% respectively.

Our study demonstrated that age, monocytes count, amount of contrast and Mehran risk score were notably higher in the CIN group. Also, Diabetic and hypertensive patients were significantly more in CIN

group. However, HDL level, hemoglobin level, EF, systolic and diastolic Blood Pressure were significantly lower in CIN group.

CONCLUSION

In patients with acute STEMI following initial PCI, a high MHR level may be utilized as a predictor of CIN. The clinical implications of our findings need to be confirmed and revealed by more prospective trials.

No Funding
No Conflict of Interest

REFERENCES

1. **Akcay A, Nguyen Q, Edelstein C (2009):** Mediators of inflammation in acute kidney injury. Mediators of Inflammation, 9:137072. doi: 10.1155/2009/137072.
2. **Andò G, Morabito G, de Gregorio C *et al.* (2013):** Age, glomerular filtration rate, ejection fraction, and the AGEF score predict contrast-induced nephropathy in patients with acute myocardial infarction undergoing primary percutaneous coronary intervention. Catheterization and Cardiovascular Interventions, 82(6):878-85.
3. **Azzalini L, Spagnoli V, Ly H (2016):** Contrast-induced nephropathy: from pathophysiology to preventive strategies. Canadian Journal of Cardiology, 32(2):247-55.
4. **Barrett B, Parfrey P (2006):** Preventing nephropathy induced by contrast medium. New England Journal of Medicine, 354(4):379-86.
5. **Ghani A, Tohamy K (2009):** Risk score for contrast induced nephropathy following percutaneous coronary intervention. Saudi Journal of Kidney Diseases and Transplantation, 20(2):240-5.
6. **Ghattas A, Griffiths H, Devitt A *et al.* (2013):** Monocytes in coronary artery disease and atherosclerosis: where are we now? Journal of the American College of Cardiology, 62(17):1541-51.
7. **Heyman S, Rosen S, Khamaisi M *et al.* (2010):** Reactive oxygen species and the pathogenesis of radiocontrast-induced nephropathy. Investigative Radiology, 45(4):188-95.
8. **Ibanez B, James S, Agewall S *et al.* (2018):** 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Socie. European Heart Journal, 39(2):119-77.
9. **Jiang M, Yang J, Zou H *et al.* (2022):** Monocyte-to-high-density lipoprotein-cholesterol ratio (MHR) and the risk of all-cause and cardiovascular mortality: a nationwide cohort study in the United States. Lipids in Health and Disease, 21(1):1-10.
10. **Krychtiuk K, Kastl S, Pfaffenberger S *et al.* (2014):** Small high-density lipoprotein is associated with monocyte subsets in stable coronary artery disease. Atherosclerosis, 237(2):589-96.
11. **Lin K, Zheng W, Bei W *et al.* (2017):** A novel risk score model for prediction of contrast-induced nephropathy after emergent percutaneous coronary intervention. International Journal of Cardiology, 230:402-12.
12. **McCullough P, Wolyn R, Rocher L *et al.* (1997):** Acute renal failure after coronary intervention: incidence, risk factors, and relationship to mortality. The American Journal of Medicine, 103(5):368-75.
13. **Mehran R, Aymong E, Nikolsky E *et al.* (2004):** A simple risk score for prediction of contrast-induced nephropathy after percutaneous coronary intervention: development and initial validation. Journal of the American College of Cardiology, 44(7):1393-9.
14. **Mehran R, Nikolsky E (2006):** Contrast-induced nephropathy: definition, epidemiology, and patients at risk. Kidney International, 69:11-5.
15. **Ndrepepa G, Kastrati A, Mehilli J *et al.* (2009):** Mechanical reperfusion and long-term mortality in patients with acute myocardial infarction presenting 12 to 48 hours from onset of symptoms. JAMA., 301(5):487-8.
16. **Rich M, Crecelius C (1990):** Incidence, risk factors, and clinical course of acute renal insufficiency after cardiac catheterization in patients 70 years of age or older: a prospective study. Archives of Internal Medicine, 150(6):1237-42.
17. **Rihal C, Textor S, Grill D *et al.* (2002):** Incidence and prognostic importance of acute renal failure after percutaneous coronary intervention. Circulation, 105(19):2259-64.
18. **Rokos I, Farkouh M, Reiffel J *et al.* (2012):** Correlation between index electrocardiographic patterns and pre-intervention angiographic findings: Insights from the HORIZONS-AMI trial. Catheterization and Cardiovascular Interventions, 79(7):1092-8.
19. **Sağ S, Yıldız A, Kaderli A *et al.* (2017):** Association of monocyte to HDL cholesterol level with contrast induced nephropathy in STEMI patients treated with primary PCI. Clinical Chemistry and Laboratory Medicine, 55(1):132-8.
20. **Schömig A, Mehilli J, Antoniucci D *et al.* (2005):** Mechanical reperfusion in patients with acute myocardial infarction presenting more than 12 hours from symptom onset: a randomized controlled trial. JAMA., 293(23):2865-72.
21. **Thygesen K, Alpert J, Jaffe A *et al.* (2012):** Third universal definition of myocardial infarction. Eur Heart J., 33(20):2551-67.
22. **Ulus T, Isgandarov K, Yilmaz A *et al.* (2018):** Monocyte to high-density lipoprotein ratio predicts contrast-induced nephropathy in patients with acute coronary syndrome. Angiology, 69(10):909-16.
23. **Wang T, Zhang M, Fu Y *et al.* (2009):** Incidence, distribution, and prognostic impact of occluded culprit arteries among patients with non–ST-elevation acute coronary syndromes undergoing diagnostic angiography. American Heart Journal, 157(4):716-23.
24. **Zehir R, Tekkeşin A, Haykir N *et al.* (2017):** Predictive Value of Monocyte to High-Density Lipoprotein Cholesterol Ratio for Contrast-Induced Nephropathy in Patients with ST-Segment Elevation Myocardial Infarction Who Underwent Primary Percutaneous Coronary Intervention. Koşuyolu Heart Journal, 20(1):13-8.