Observational Study

Medicine

OPEN

Laboratory markers at admission to predict the presence of totally occluded culprit artery in NSTEMI

Ufuk Yildirim, MD*, Fuatcan Balaban, MD

Abstract

A significant proportion of patients presenting with non-ST-segment elevation myocardial infarction (NSTEMI) have a totally occluded culprit artery (OCA). If these patients do not meet very high-risk criteria, they may be deprived of an immediate invasive strategy. Therefore, there is a need for markers that can predict OCA in patients with NSTEMI. A total of 357 consecutive patients with NSTEMI but without very high-risk criteria were included in this retrospective study. Two groups were formed: NSTEMI with OCA (n = 106) and NSTEMI with patent culprit artery (PCA) (n = 251). Complete blood count (CBC) and serum biochemical parameters obtained immediately at admission were compared between the groups. Receiver operating characteristic (ROC) analysis to predict the presence of OCA was performed for the parameters that were significantly different between the groups, and an area under the curve (AUC) > 0.7 was considered to suggest acceptable discrimination. Neutrophil count [8.13 (2.82-27.88) × 10^3/μL vs 5.59 (1.85-19.71) × 10^3/μL, $P < .001$] and aspartate aminotransferase (AST) level [45 (12-405) U/L vs 25 (5-143) U/L, $P < .001$] were significantly higher in patients with OCA. The AUC was 0.750 for neutrophil count and 0.731 for AST level. The sensitivity, specificity, positive predictive value, and negative predictive value (NPV) of elevated neutrophil and/or AST levels for the presence of OCA were 77.4%, 70.1%, 52.2%, and 88.0%, respectively. More strikingly, the specificity was 95.2% in the presence of both neutrophil and AST elevation. Elevated neutrophil and/or AST levels at admission were strongly associated with the presence of OCA in patients with NSTEMI.

Abbreviations: ACS = acute coronary syndrome, ALT = alanine aminotransferase, AST = aspartate aminotransferase, AUC = area under the curve, CBC = complete blood count, ICA = invasive coronary angiography, MI = myocardial infarction, NLR = neutrophil-to-lymphocyte ratio, NPV = negative predictive value, NSTEMI = non-ST-segment elevation myocardial infarction, OCA = occluded culprit artery, PCA = patent culprit artery, PCI = percutaneous coronary intervention, PLR = platelet-to-lymphocyte ratio, ROC = receiver operating characteristic, RVD = reference vessel diameter, STEMI = ST-segment elevation myocardial infarction, TIMI = Thrombolysis in Myocardial Infarction, URL = upper reference limit, WBC = white blood cell.

Keywords: aspartate aminotransferase, neutrophil count, NSTEMI, occluded culprit artery

1. Introduction

Acute coronary syndrome (ACS) can manifest in three ways: ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI), and unstable angina. Totally occluded culprit arteries (OCA) in the setting of ACS usually present with STEMI, and current guidelines recommend an immediate invasive strategy for patients with STEMI.[1] However, about 30% of patients with NSTEMI have an OCA as well,[2,3] but these patients may be deprived of an immediate invasive strategy if they do not meet very high-risk criteria.[4] Considering the worse prognosis in NSTEMI with OCA compared to NSTEMI with patent culprit artery (PCA),[2,3] delayed revascularization may be deleterious for these patients. Therefore, it is important to predict patients with OCA at the time of admission. Several electrocardiographic findings that may be indicative of the presence of OCA in NSTEMI have been suggested.[5] Nevertheless, better tools that can predict these patients are required to enable earlier revascularization and potentially improve prognosis. Therefore, in the present study, we aimed to compare NSTEMI patients with OCA and NSTEMI patients with PCA in terms of complete blood count (CBC) and serum biochemical parameters obtained immediately at admission to identify a possible marker that can predict the presence of OCA in this setting.

2. Methods

After obtaining approval from the institutional ethics committee, patients who underwent invasive coronary angiography

The authors have no funding and conflicts of interest to disclose.

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

Department of Cardiology, Faculty of Medicine, Ondokuz Mayis University, Samsun, Turkey.

** Correspondence: Ufuk Yildirim, Department of Cardiology, Faculty of Medicine, Ondokuz Mayis University, Samsun, Turkey (e-mail: ufukyildirim2715@yahoo.com.tr).*

How to cite this article: Yildirim U, Balaban F. Laboratory markers at admission to predict the presence of totally occluded culprit artery in NSTEMI. Medicine 2022;101:45(e31738).

Received: 12 February 2022 / Received in final form: 19 October 2022 / Accepted: 20 October 2022

http://dx.doi.org/10.1097/MD.0000000000031738

(ICA) with a diagnosis of NSTEMI between January 2019 and December 2020 at our 24/7 percutaneous coronary intervention (PCI) capable center were retrospectively screened for the present study. NSTEMI diagnosis was based on "Fourth Universal Definition of Myocardial Infarction (MI)."[6] Exclusion criteria were as follows: very high-risk criteria (hemodynamic instability or cardiogenic shock, recurrent or refractory chest pain despite medical treatment, life-threatening arrhythmias, mechanical complications of MI, heart failure clearly related to NSTEMI, presence of ST-segment depression >1 mm in ≥6 leads additional to ST-segment elevation in a VR and/or V1),[4] resuscitated cardiac arrest, prior coronary artery bypass grafting, no identifiable culprit vessel, left main coronary artery as the culprit vessel, multiple culprit vessels, presence of chronic total occlusion in any coronary artery, presence of active infection, hematological disorders, and elevated baseline liver enzymes. Exclusion criteria were checked from "the digital files in which angiographic features of the patients were recorded," "the hospital database," and "the National Health Record System." After the exclusion criteria, 357 consecutive patients were included, and two groups were formed: NSTEMI with OCA (n = 106) and NSTEMI with PCA (n = 251).

Baseline characteristics of the study population were collected from the National Health Record System. CBC and serum biochemical parameters at admission were recorded from the hospital database. CBC parameters had been measured using Sysmex XN 3000 Hematology Analyzer (Sysmex Corporation, Kobe, Japan) and serum biochemical parameters had been measured using Roche Cobas 8000 Chemistry Analyzer (Roche Diagnostics, Mannheim, Germany). The angiographic images of all patients were reviewed by the same interventional cardiologist blinded to the laboratory parameters at admission. Diameter stenosis was visually estimated. Reference vessel diameter (RVD) was accepted as the diameter reached by the stent or the post-dilatation balloon if used. Culprit arteries with Thrombolysis in Myocardial Infarction (TIMI) flow grade 0 or 1 before intervention were defined as occluded, and those with TIMI flow grade 2 or 3 were defined as patent.

Statistical Package for Social Sciences (SPSS) version 25 was used to upload and analyze the data. Descriptive statistics for categorical variables were presented as frequencies and percentages, and Chi-square test was used to identify significant differences between the groups. Whether numerical variables were normally distributed was evaluated by visual (histograms and probability plots) and analytical (Kolmogorov-Smirnov and Shapiro–Wilk tests) methods. Descriptive statistics for numerical variables with a normal distribution were expressed as mean ± standard deviation, and those without a normal distribution were expressed as median (minimum-maximum). Independent samples *t* test was used to compare numerical variables with a normal distribution, and Levene test was applied to assess the equality of variances. Mann–Whitney *U* test was used for numerical variables without a normal distribution to determine significant differences between the groups. Receiver operating characteristic (ROC) analysis to predict the presence of OCA in NSTEMI was performed for the parameters that were significantly different between the groups, and the area under the curve (AUC) values were recorded for these parameters. An AUC > 0.7 was considered to suggest acceptable discrimination. When determining the sensitivity, specificity, and predictive values of neutrophil count and aspartate aminotransferase (AST) level whose AUCs were >0.7, reference cutoff values were taken into consideration. The upper reference limit (URL) of neutrophil count was 6.98×10^3/μL for men, and 8.89×10^3/μL for women. The URL of AST level was 46 U/L for both men and women. The statistical significance level was set at $P < .05$.

3. Results

3.1. Baseline characteristics of the study groups were similar (Table 1).

Total white blood cell (WBC) count, neutrophil count, neutrophil percentage, neutrophil-to-lymphocyte ratio (NLR), monocyte count, and platelet-to-lymphocyte ratio (PLR) were significantly higher ($P < .001$, $P < .001$, $P < .001$, $P = .003$, and $P = .037$, respectively); lymphocyte percentage, monocyte percentage, eosinophil count, eosinophil percentage, and basophil percentage were significantly lower ($P < .001$, $P = .009$, $P < .001$, $P < .001$, and $P < .001$, respectively) in patients with OCA. The OCA group had higher glucose, AST, alanine aminotransferase (ALT), and AST/ALT ratio ($P = .005$, $P < .001$, $P = .001$, and $P < .001$, respectively), but lower sodium ($P < .001$) (Table 2).

Time from admission to ICA was comparable between the groups. LAD as the culprit artery was more common in patients with PCA ($P < .001$), and RCA as the culprit artery was more common in patients with OCA ($P = .002$). RVD was larger in the PCA group ($P = .001$) (Table 3).

Table 1

Baseline characteristics.

Variable		NSTEMI with OCA (n = 106)	NSTEMI with PCA (n = 251)	*P*
Age (year)		61.5 ± 13.1	61.5 ± 10.9	.964
Gender	Male (%*)	81 (76.4)	185 (73.7)	.591
	Female (%*)	25 (23.6)	66 (26.3)	
Hypertension (%*)		47 (44.3)	125 (49.8)	.345
Diabetes (%*)		37 (34.9)	81 (32.3)	.629
HbA1c (%)		5.9 (4.8-12.0)	6.0 (4.6-16.9)	.941
Total cholesterol (mg/dL)		182.1 ± 43.8	177.9 ± 40.5	.413
LDL cholesterol (mg/dL)		108.8 ± 37.2	106.1 ± 33.2	.535
HDL cholesterol (mg/dL)		37 (17-68)	36 (21-78)	.192
Triglyceride (mg/dL)		158 (44-869)	149 (38-1010)	.413
Chronic kidney disease (%*)		11 (10.4)	29 (11.6)	.747
GFR (mL/min/1.73 m^2)		82.6 ± 22.6	84.5 ± 26.2	.516
Prior PCI (%*)		20 (18.9)	64 (25.5)	.177

*Column percentage.

GFR = glomerular filtration rate; HbA1c, glycated hemoglobin, HDL = high-density lipoprotein, LDL = low-density lipoprotein, NSTEMI = non-ST-segment elevation myocardial infarction, OCA = occluded culprit artery, PCA = patent culprit artery, PCI = percutaneous coronary intervention.

Table 2

CBC and serum biochemical parameters at admission.

Variable	NSTEMI with OCA (n = 106)	NSTEMI with PCA (n = 251)	*P*
RBC count (× 10^6/μL)	4.79 ± 0.53	4.76 ± 0.58	.600
Hemoglobin (g/dL)	14.00 ± 1.76	13.80 ± 1.74	.336
Hematocrit (%)	41.30 ± 4.84	41.13 ± 4.64	.748
MCV (fL)	86.7 (69.6-105.0)	87.4 (63.9-102.9)	.303
MCH (pg)	29.5 (21.2-34.9)	-	.850
MCHC (g/dL)	33.6 (29.9-37.4)	33.6 (27.0-37.2)	.373
RDW (%)	13.1 (11.5-18.6)	13.2 (11.6-24.3)	.301
Total WBC count (× 10^3/μL)	11.14 (4.57-29.16)	8.79 (5.01-24.39)	**<.001**
Neutrophil count (× 10^3/μL)	8.13 (2.82-27.88)	5.59 (1.85-19.71)	**<.001**
Neutrophil percentage (%)	73.5 ± 10.7	65.7 ± 12.1	**<.001**
Lymphocyte count (× 10^3/μL)	2.02 ± 0.93	2.20 ± 0.87	.092
Lymphocyte percentage (%)	18.8 ± 9.1	25.2 ± 9.9	**<.001**
NLR	3.98 (1.13-35.74)	2.56 (0.66-56.52)	**<.001**
Monocyte count (× 10^3/μL)	0.74 ± 0.33	0.63 ± 0.22	**.003**
Monocyte percentage (%)	6.5 ± 2.2	7.2 ± 2.3	**.009**
Eosinophil count (× 10^3/μL)	0.05 (0-0.61)	0.10 (0-0.77)	**<.001**
Eosinophil percentage (%)	0.5 (0-5.9)	1.2 (0-11.3)	**<.001**
Basophil count (× 10^3/μL)	0.04 (0.01-0.15)	0.04 (0.01-0.11)	.219
Basophil percentage (%)	0.4 (0.1-1.1)	0.5 (0.1-1.5)	**<.001**
Platelet count (× 10^3/μL)	240.2 ± 64.0	242.5 ± 61.8	.747
PLR	121.9 (33.4-558.8)	110.4 (46.1-700.0)	**.037**
MPV (fL)	10.1 ± 0.9	10.0 ± 0.9	.200
PDW (fL)	11.1 (8.0-21.0)	10.8 (8.1-19.9)	.241
Glucose (mg/dL)	131 (66-448)	120 (65-494)	**.005**
Sodium (mEq/L)	139.8 ± 2.9	140.9 ± 2.8	**<.001**
Potassium (mEq/L)	4.43 ± 0.56	4.35 ± 0.48	.182
Calcium (mg/dL)	9.52 ± 0.47	9.50 ± 0.48	.743
BUN (mg/dL)	15.3 (7.5-45.7)	15.5 (7.4-57.6)	.673
Creatinine (mg/dL)	0.90 (0.47-5.69)	0.87 (0.45-6.15)	.474
AST (U/L)	45 (12-405)	25 (5-143)	**<.001**
ALT (U/L)	24 (5-73)	19 (5-71)	**.001**
AST/ALT ratio	2.00 (0.33-24.40)	1.33 (0.53-5.11)	**<.001**

ALT = alanine aminotransferase, AST = aspartate aminotransferase, BUN = blood urea nitrogen, CBC = complete blood count, MCH = mean corpuscular hemoglobin, MCHC = mean corpuscular hemoglobin concentration, MCV = mean corpuscular volume, MPV = mean platelet volume, NLR = neutrophil-to-lymphocyte ratio, NSTEMI = non-ST-segment elevation myocardial infarction, OCA = occluded culprit artery, PCA = patent culprit artery, PDW = platelet distribution width, PLR = platelet-to-lymphocyte ratio, RBC = red blood cell, RDW = red cell distribution width, WBC = white blood cell.

Table 3

Angiographic characteristics.

Variable		NSTEMI with OCA (n = 106)	NSTEMI with PCA (n = 251)	*P*
Time from admission to ICA (h)		12 (2-80)	16 (2-75)	.121
Culprit artery	LAD (%*)	32 (30.2)	128 (51.0)	**<.001**
	CX (%*)	40 (37.7)	79 (31.5)	.251
	RCA (%*)	34 (32.1)	44 (17.5)	**.002**
Native/in-stent	Native (%*)	96 (90.6)	231 (92.0)	.648
	In-stent (%*)	10 (9.4)	20 (8.0)	
Diameter stenosis (%)		100	95 (70-99)	**<.001**
Reference vessel diameter (mm)		3.0 (2.3-5.0)	3.1 (2.0-5.0)	**.001**
TIMI flow grade before intervention	0 (%*)	71 (67.0)	0	**<.001**
	1 (%*)	35 (33.0)	0	
	2 (%*)	0	77 (30.7)	
	3 (%*)	0	174 (69.3)	

*Column percentage.

CX = circumflex artery, ICA = invasive coronary angiography, LAD = left anterior descending artery, NSTEMI = non-ST-segment elevation myocardial infarction, OCA = occluded culprit artery, PCA = patent culprit artery, RCA = right coronary artery, TIMI = Thrombolysis in Myocardial Infarction.

ROC analysis revealed that the AUC values of neutrophil count, total WBC count, and AST level were >0.7 (0.750, 0.736, and 0.731, respectively) (Table 4).

The sensitivity, specificity, positive predictive value, and negative predictive value (NPV) of elevated neutrophil and/or AST levels for the presence of OCA were 77.4%, 70.1%, 52.2%, and 88.0%, respectively. When looking at other remarkable values, elevated neutrophil had an NPV of 81.6%, elevated AST had a specificity of 85.7%, and elevated neutrophil and AST had a specificity of 95.2% (Table 5).

4. Discussion

The present study has revealed that elevated neutrophil and/or AST levels at admission can predict the presence of OCA with a sensitivity of 77.4% and a specificity of 70.1% in NSTEMI patients without very high-risk criteria. More strikingly, the specificity was 95.2% in the presence of both neutrophil and AST elevation.

CBC parameters have been investigated in many clinical studies on acute MI. In a study that included patients presenting with STEMI, total WBC count at admission was found to be an independent predictor of infarct size and baseline TIMI grade 0/1 flow in the culprit artery, but no data on differential WBC count were available.[7] In another study that included patients with acute MI undergoing primary PCI, total WBC count, neutrophil count, neutrophil percentage, and NLR were significantly higher and lymphocyte percentage was significantly lower in patients with OCA than in patients with PCA, in line with our study. In addition, elevated neutrophil count at admission was found to be an independent predictor of total coronary occlusion in patients with acute MI undergoing primary PCI in the aforementioned study.[8] NLR[9–12] and PLR[10] have also been suggested in clinical studies as predictors of culprit artery patency in STEMI. The vast majority of the patient population in all these studies consisted of STEMI patients who already had an indication for an immediate invasive strategy. In contrast, our study population consisted of NSTEMI patients without very high-risk criteria, so there was no indication for an immediate invasive strategy according to current guidelines.[4] In our study, many significant differences were detected between the groups in terms of CBC parameters at admission. Total WBC count, neutrophil count, neutrophil percentage, NLR, monocyte count, and PLR were significantly higher; lymphocyte percentage, monocyte percentage, eosinophil count, eosinophil percentage, and basophil percentage were significantly lower in the OCA group. However, only neutrophil count and total WBC count were considered to suggest acceptable discrimination (AUC > 0.7) between patients with OCA and those with PCA, according to our study design. Since they increase in line with each other due to a possible inflammatory response and the AUC value of neutrophil count (0.750) was higher than that of total WBC count (0.736), neutrophil count has been more emphasized as a predictor of OCA in the present paper. In the literature, red cell distribution width has also been suggested as a predictor of OCA in acute MI,[13,14] but it was comparable between the groups in our study.

We have demonstrated that elevated AST level at admission can also predict the presence of OCA in NSTEMI patients without very high-risk criteria. Historically, AST was the first cardiac biomarker to be used for acute MI diagnosis,[15] but it has been substituted by more sensitive tests over time. Because the myocardium has high AST activity, myocardial necrosis may cause an increase in serum AST level. However, serum AST level does not increase in every patient with acute MI. In a study investigating the pattern of liver enzyme elevations in STEMI, 86% of patients had elevated AST within 24 hours of admission.[16] In another study investigating the effect of AST on prognosis in NSTEMI, only 33% of patients had elevated AST within 24 hours of admission. In addition, AST was found to be a stronger predictor of in-hospital mortality than troponin peak in the aforementioned study, but no data on culprit artery patency were available.[17] It is obvious that further studies are needed to better understand what AST elevation means, particularly in NSTEMI. In our study, ALT and AST/ALT ratio were also

Table 4

ROC analysis for predicting totally occluded culprit artery in NSTEMI.

Variable	AUC	95% CI	*P*
Neutrophil count	0.750	0.695-0.805	**<.001**
Total WBC count	0.736	0.678-0.795	**<.001**
AST	0.731	0.669-0.792	**<.001**
Neutrophil percentage	0.686	0.628-0.744	**<.001**
NLR	0.686	0.627-0.745	**<.001**
Lymphocyte percentage	0.682	0.623-0.742	**<.001**
AST/ALT ratio	0.676	0.609-0.743	**<.001**
Eosinophil percentage	0.660	0.597-0.722	**<.001**
Basophil percentage	0.644	0.581-0.707	**<.001**
Eosinophil count	0.618	0.551-0.685	**<.001**
ALT	0.612	0.547-0.678	**.001**
Sodium	0.611	0.548-0.674	**.001**
Glucose	0.593	0.531-0.656	**.005**
Monocyte count	0.589	0.519-0.659	**.008**
Monocyte percentage	0.588	0.524-0.652	**.009**
PLR	0.570	0.504-0.636	**.037**

ALT = alanine aminotransferase, AST = aspartate aminotransferase, AUC = area under the curve, CI = confidence interval, NLR = neutrophil-to-lymphocyte ratio, NSTEMI = non-ST-segment elevation myocardial infarction, PLR = platelet-to-lymphocyte ratio, ROC = receiver operating characteristic, WBC = white blood cell.

Table 5

Sensitivity, specificity, and predictive values of elevated neutrophil and AST levels at admission for predicting totally occluded culprit artery in NSTEMI.

Variable	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
Elevated neutrophil	57.5	79.6	54.5	81.6
Elevated AST	47.2	85.7	58.1	79.3
Elevated neutrophil and AST	27.4	95.2	70.7	75.6
Elevated neutrophil and/or AST	77.4	70.1	52.2	88.0

AST = aspartate aminotransferase, NPV = negative predictive value, NSTEMI = non-ST-segment elevation myocardial infarction, PPV = positive predictive value.

higher in patients with OCA. The OCA group had higher serum glucose, likely due to higher stress, and lower serum sodium, likely due to higher serum glucose. In the literature, AST/ALT ratio[18] and serum glucose level[9] at admission have also been suggested as predictors of culprit artery patency in acute MI. However, in our study, none of the serum biochemical parameters at admission other than AST were capable enough to distinguish between patients with OCA and those with PCA, according to our study design.

In patients with acute MI, a possible increase in neutrophil count begins within a few hours after symptom onset,[19] and a possible increase in serum AST level begins 3 to 4 hours after symptom onset.[20] So neutrophil count may better predict the presence of OCA in patients presenting early after symptom onset, and serum AST level may provide additional information in patients presenting later. Therefore, considering patients presenting at different time periods after symptom onset, we also combined elevated neutrophil and AST levels when determining the sensitivity, specificity, and predictive values. Additionally, in the present study, we did not determine new cutoff values for neutrophil count and serum AST level and acted according to the reference cutoff values determined for the kits used. The URL of neutrophil count was 6.98×10^3/μL for men, and 8.89×10^3/μL for women. The URL of AST level was 46 U/L for both men and women.

Our findings imply that elevation in neutrophil count and serum AST level may be suggestive of OCA and transmural myocardial injury in NSTEMI. Therefore, an immediate invasive strategy may be considered in these patients. Additional measurements 3 to 6 hours after symptom onset may increase the sensitivity and enable earlier revascularization. To our knowledge, this is the first study to associate AST elevation with the presence of OCA in NSTEMI.

Our study had some limitations. This was a retrospective single-center study with relatively small sample size. We did not have data regarding the time from symptom onset to admission. Cardiac troponin levels at admission were not evaluated in this study because various kits had been used at our center in the specified period. Finally, the study population consisted of highly selected patients; therefore, our findings may not be applicable to all patients with NSTEMI.

In conclusion, elevated neutrophil and/or AST levels at admission were strongly associated with the presence of OCA in patients with NSTEMI. However, prospective studies are required to better understand the course of neutrophil count and serum AST level in NSTEMI with OCA and NSTEMI with PCA.

Author contributions

Conceptualization: Fuatcan Balaban, Ufuk Yildirim.
Data curation: Fuatcan Balaban, Ufuk Yildirim.
Formal analysis: Ufuk Yildirim.
Funding acquisition: Ufuk Yildirim.
Investigation: Fuatcan Balaban, Ufuk Yildirim.
Methodology: Fuatcan Balaban, Ufuk Yildirim.
Project administration: Ufuk Yildirim.
Resources: Fuatcan Balaban, Ufuk Yildirim.
Software: Fuatcan Balaban, Ufuk Yildirim.
Supervision: Fuatcan Balaban, Ufuk Yildirim.
Validation: Fuatcan Balaban, Ufuk Yildirim.
Visualization: Fuatcan Balaban, Ufuk Yildirim.
Writing – original draft: Ufuk Yildirim.
Writing – review & editing: Fuatcan Balaban, Ufuk Yildirim.

References

[1] Ibanez B, James S, Agewall S, et al. 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 Society of Cardiology (ESC). Eur Heart J. 2018;39:119–77.
[2] Khan AR, Golwala H, Tripathi A, et al. Impact of total occlusion of culprit artery in acute non-ST elevation myocardial infarction: a systematic review and meta-analysis. Eur Heart J. 2017;38:3082–9.
[3] Hung CS, Chen YH, Huang CC, et al. Prevalence and outcome of patients with non-ST segment elevation myocardial infarction with occluded "culprit" artery - a systemic review and meta-analysis. Crit Care. 2018;22:34.
[4] Collet JP, Thiele H, Barbato E, et al. 2020 ESC guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. Eur Heart J. 2021;42:1289–367.
[5] Tziakas D, Chalikias G, Al-Lamee R, et al. Total coronary occlusion in non ST elevation myocardial infarction: Time to change our practice? Int J Cardiol. 2021;329:1–8.
[6] Thygesen K, Alpert JS, Jaffe AS, et al. Executive group on behalf of the Joint European Society of Cardiology (ESC)/American College of Cardiology (ACC)/American Heart Association (AHA)/World Heart Federation (WHF) Task Force for the Universal Definition of Myocardial Infarction. Fourth Universal Definition of Myocardial Infarction (2018). J Am Coll Cardiol. 2018;72:2231–64.
[7] Palmerini T, Brener SJ, Genereux P, et al. Relation between white blood cell count and final infarct size in patients with ST-segment elevation acute myocardial infarction undergoing primary percutaneous coronary intervention (from the INFUSE AMI trial). Am J Cardiol. 2013;112:1860–6.
[8] Liang Y, Chen H, Wang P. Correlation of leukocyte and coronary lesion severity of acute myocardial infarction. Angiology. 2018;69:591–9.
[9] Doğan M, Akyel A, Bilgin M, et al. Can admission neutrophil to lymphocyte ratio predict infarct-related artery patency in ST-Segment elevation myocardial infarction. Clin Appl Thromb Hemost. 2015;21:172–6.
[10] Yayla C, Akboğa MK, Canpolat U, et al. Platelet to lymphocyte ratio can be a predictor of infarct-related artery patency in patients with ST-Segment elevation myocardial infarction. Angiology. 2015;66:831–6.
[11] Kurtul A, Murat SN, Yarlioglues M, et al. The relationship between neutrophil/lymphocyte ratio and infarct-related artery patency before mechanical reperfusion in patients with ST-elevation myocardial infarction. Coron Artery Dis. 2014;25:159–66.
[12] Erkol A, Oduncu V, Turan B, et al. Neutrophil to lymphocyte ratio in acute ST-segment elevation myocardial infarction. Am J Med Sci. 2014;348:37–42.
[13] Akboga MK, Yayla C, Yilmaz S, et al. Increased red cell distribution width predicts occlusion of the infarct-related artery in STEMI. Scand Cardiovasc J. 2016;50:114–8.
[14] Açıkgöz SK, Akboğa MK, Açıkgöz E, et al. Red cell distribution width predicts totally occluded infarct-related artery in NSTEMI. Scand Cardiovasc J. 2016;50:224–9.
[15] Ladue JS, Wroblewski F, Karmen A. Serum glutamic oxaloacetic transaminase activity in human acute transmural myocardial infarction. Science. 1954;120:497–9.
[16] Lofthus DM, Stevens SR, Armstrong PW, et al. Pattern of liver enzyme elevations in acute ST-elevation myocardial infarction. Coron Artery Dis. 2012;23:22–30.
[17] Arora S, Caughey MC, Misenheimer JA, et al. Elevated serum aspartate transaminase as a predictor of early mortality in patients with Non-ST-Segment elevation myocardial infarction. Circulation. 2018;136(Suppl_1):A15577.
[18] Djakpo DK, Wang ZQ, Shrestha M. The significance of transaminase ratio (AST/ALT) in acute myocardial infarction. Arch Med Sci Atheroscler Dis. 2020;5:e279–83.
[19] Hansen PR. Role of neutrophils in myocardial ischemia and reperfusion. Circulation. 1995;91:1872–85.
[20] Patibandla S, Gupta K, Alsayouri K. Cardiac Enzymes. Updated 2021 Aug 11. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2021.