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Long-term Prognosis after Treatment of Total Occluded Coronary Artery is well Predicted by Neutrophil to High-Density Lipoprotein Ratio: a Comparison Study

Aim	Mortality prediction is very important for more effective treatment of patients with acute coronary syndrome. Hematological and lipid parameters have been used for this purpose, as this approach is non-invasive and cost effective. In this study, our aim was to evaluate which parameter predicts mortality most accurately.
Material and Methods	Data of 554 patients with at least one total coronary artery occlusion were collected retrospectively. Receiver operating characteristic curves were used to determine the optimal cut-off points of Neu/HDL, Neu/Lym, Mono/HDL, Trig/HDL, HDL/LDL, Plt/Lym and Lym/HDL according to long-term cardiovascular survival. Median follow-up time was 520 days, and 30 patients died.
Results	The mean age was 60.96±0.50 yrs. The area under the curve (AUC) for Neu/HDL was 0.830 (p<0.001, 95% confidence interval [CI]: 0.753 to 0.908). The cut-off point was 0.269, with a sensitivity of 74.2% and a specificity of 74.2%. The AUC for Neu/Lym was 0.688 (p<0.001, 95% CI: 0.586 to 0.790). The cut-off point was 5.322, with a sensitivity of 67.7% and a specificity of 67.1%. The Neu/HDL (hazard ratio, HR [confidence interval, CI]: 0.202 [0.075–0.545], p=0.002) and Neu/Lym (0.306 [0.120–0.777], p=0.013) were associated with increased risk of death according to multivariate Cox regression analysis.
Conclusions	Neu/HDL offers a better long-term mortality prediction than Neu/Lym, Mono/HDL, Trig/HDL, HDL/LDL, Plt/Lym, or Lym/HDL after treatment of total coronary artery occlusion.

Keywords	Acute coronary syndrome; cost-benefit analysis; coronary occlusion; mortality prediction
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Introduction

Cardiovascular diseases are the leading cause of morbidity and mortality, affecting about 200 million adults worldwide [1]. Although great improvements have been made in diagnosis and treatment, acute coronary syndrome (ACS) is still often fatal. Many studies have identified optimal prognostic parameters [2, 3]. However, there is still debate on this issue, and an effective outcome predictor is urgently needed.

Cardiovascular diseases are caused by the processes of atherosclerosis in which inflammation and lipid accumulation play an important role [4–7]. Low density lipoprotein (LDL) causes an increase in the atherosclerotic burden, while high density lipoprotein (HDL) plays a protective role [8].

The accumulation of anti-inflammatory agents, including neutrophils, lymphocytes, and monocytes, in atherosclerotic plaques cause plaque rupture, and this situation starts the ACS [9]. In addition, HDL, LDL, and anti-inflammatory agents each affect the function of the other [10–12]. Therefore, the concentrations of these agents in the circulating blood are very important for determining the cardiovascular disease burden.

Mortality prediction is very important for effective treatment of ACS patients. Hematological and lipid parameters have been used for this purpose, as this method is non-invasive, easily accessible, and cost effective. The ratios of neutrophil to HDL (Neu/HDL), neutrophil to lymphocyte (Neu/Lym), monocyte to HDL (Mono/HDL), triglyceride to HDL (Trig/HDL), HDL to LDL (HDL/LDL), platelet to lymphocyte (Plt/Lym), and lymphocyte to HDL (Lym/HDL) are the most studied parameters in clinical practice for prediction of ACS mortality [13–18]. In this study, we aimed to evaluate which mortality predictor parameter among those mentioned above is the most accurate.

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Material and Methods

Patient Data Collection

In this study, the data of 554 patients who were treated at our center between October 2018 and January 2020 were collected retrospectively. All patients were diagnosed with ST segment elevation myocardial infarction (STEMI) or Non-ST segment elevation myocardial infarction (Non-STEMI) with at least one totally occluded coronary artery. All patients gave informed consent to the coronary angiography procedure and data collection. This retrospective study was carried out in accordance with the October 2008 Declaration of Helsinki, and the study was approved by the Institutional Review Board of Osmangazi University (2020-328).

STEMI has been defined as a typical symptom of myocardial injury with ST-segment elevation > 1 mm in ≥2 contiguous leads and/or a new onset of left bundle branch block [19]. Non-STEMI has been defined as a rise of myocardial injury markers in combination with typical symptoms of myocardial ischemia but without ST-segment elevation [20].

The exclusion criteria were 1) patients younger than 18 years old, 2) Non-STEMI patients without any totally occluded coronary arteries, 3) patients with coincident trauma and sepsis, 4) patients with lack of clinical data, 5) patients that died from causes unrelated to ACS, 6) patients with malignancies, hematological disease, oncological disease, and usage of drugs impacting cholesterol or lymphocyte count.

Clinical Data Collection

Demographic data were collected from hospital records regarding the gender, age, smoking status, and history of hypertension (HT), diabetes mellitus (DM), coronary artery disease (CAD), congestive heart failure (CHF), and atrial fibrillation (AF). Laboratory data gathered were hemoglobin (Hb), hematocrit (Htc), white blood cell (Wbc), Neu, Lym, Mono, platelet (Plt), plateletcrit (Pct), HDL, LDL, and Trig concentrations. All blood samples were collected in the first 24 hrs of the ACS.

Coronary angiograms were evaluated by two cardiology specialists. Diseased vessels were defined as having 70%, or greater, stenosis. Patients were grouped as 1, 2, or 3 according to number of diseased vessels. All patients were treated according to the most critically diseased vessel. If the patient had two or more critically diseased vessels, a stage approach was performed. All patients were revascularized percutaneously.

The locations of the coronary vessel lesions were grouped according to the BARI protocol [21]. The proximal group was defined by BARI 1, 11, 12, or 18. The mid group was defined by 2, 13, 15, 19, 20, or 28. The distal group was defined by BARI 3, 4, 5, 6, 7, 8, 9, 14, 19a, or 23.

Clinical Follow-up and End Points

The end point of the present study was cardiovascular mortality that occurred during the follow-up period. Cardiovascular mortality was defined as death resulting from any reason related to ACS, including cardiac mechanical complications or ventricular arrhythmias. The follow-up of all patients began from the day of the angiography procedure until November 2020 (median 520 days). The status of all patients concerning mortality was determined from our national medical care system.

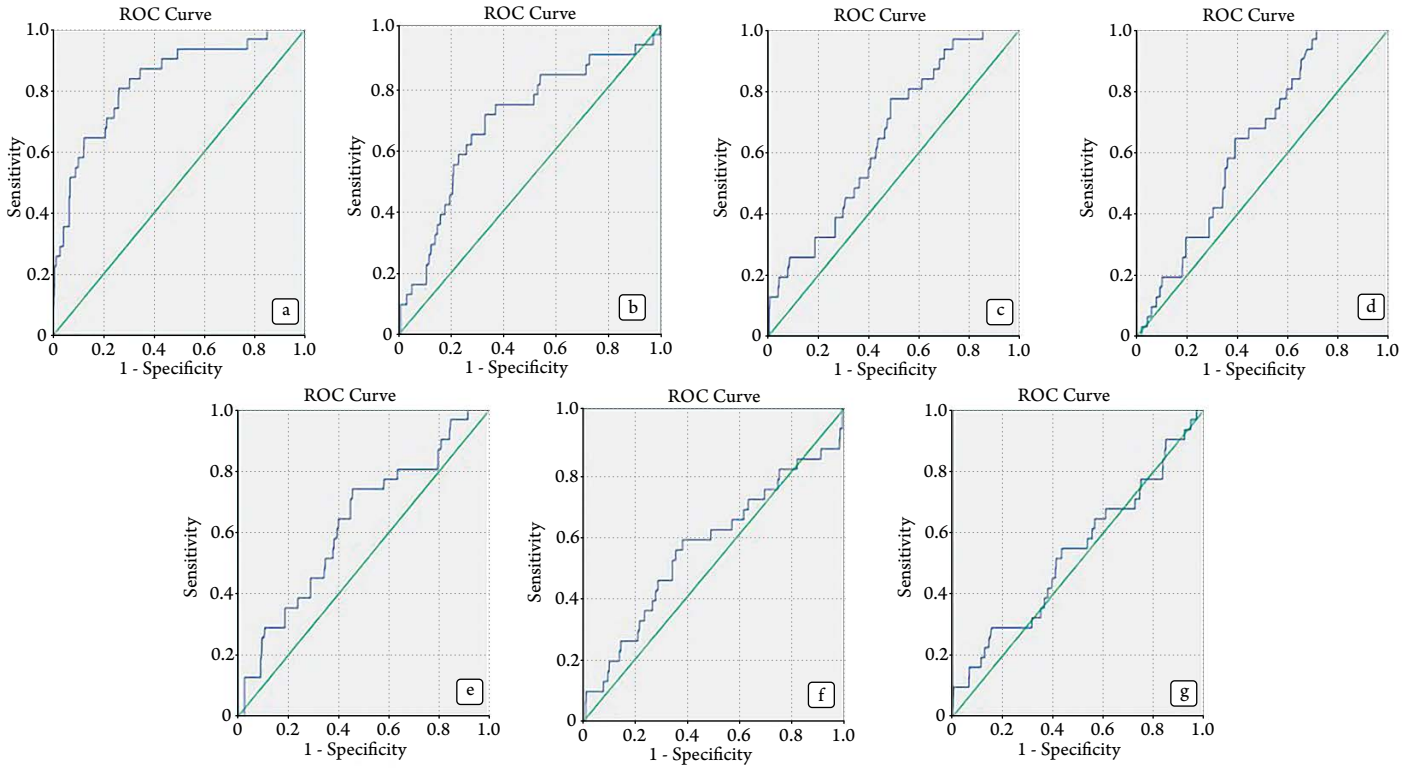
Statistical Analysis

Categorical and continuous data were expressed as ratios (%) and medians (range), and they were then compared with the chi-square and one-way ANOVA tests, respectively. The Kolmogorov-Smirnov test was performed to test if the numerical variables were normally distributed. Receiver operating characteristic (ROC) curves were used to determine the optimal cut-off points of Neu/HDL, Neu/Lym, Mono/HDL, Trig/HDL, HDL/LDL, Plt/Lym, and Lym/HDL according to cardiovascular survival. Optimal cut-off values were decided according to the Youden index [22]. The regions of culprit lesions were grouped as proximal, mid, and distal. These groups were analyzed according to the ROC curve cut-offs with a one-way ANOVA. The analyses of subgroups were performed with the Tukey and Tamhane tests according to homogeneity and non-homogeneity, respectively. Survival analyses were computed by the Kaplan–Meier method. Overall survival (OS) was calculated from the day of the procedure to the date of mortality resulting from cardiovascular causes. Patients who had not died by the last follow-up were assumed as survivors. The Kaplan–Meier curve for survival analysis was plotted to assess the prognosis between subgroups, divided according to the ROC curve cut-off points with the log-rank test. Univariate analyses were performed to determine the significance of prognostic variables with the Kaplan–Meier method. Parameters related to survival were compared with the multivariate Cox regression analyses. IBM SPSS Statistics for Windows v. 23 was used for statistical analyses. p values <0.05 were considered statistically significant.

Results

This study included 554 patients that were admitted to our clinic with STEMI or Non-STEMI with at least one totally occluded coronary artery. The mean age was 60.96±0.50 yrs, and 128 (23.1%) patients were female. According to clinical presentation, 246 (44.4%), 203 (36.6%), 15 (2.7%), 9 (1.6%) and 81 (14.6%) of patients were diagnosed with inferior, anterior, lateral, posterior, and Non-STEMI, respectively. Also, 295 (53.2%), 175 (31.6%), and 84 (15.2%) patients had one, two, or three diseased vessels, respectively.

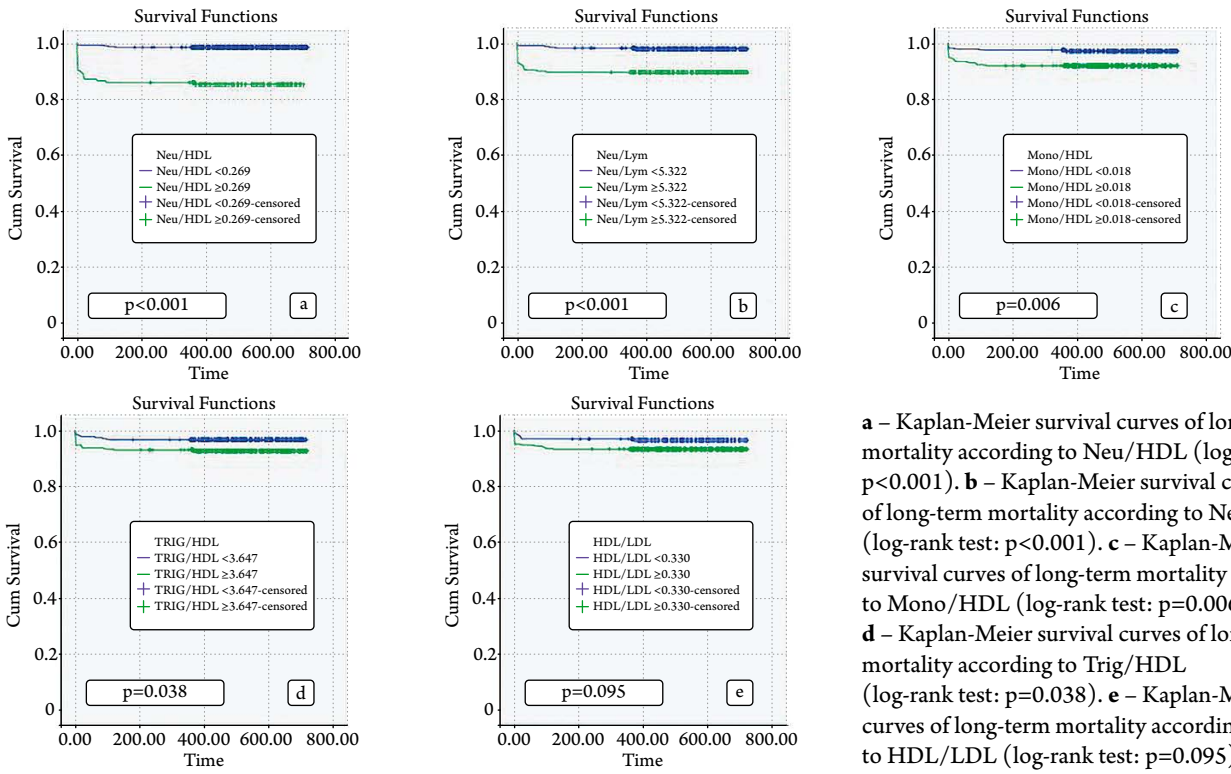
Figure 1. ROC curve analysis for Neu/HDL, Neu/Lym, Mono/HDL, Trig/HDL, HDL/LDL, Plt/Lym, and Lym/HDL (a, b, c, d, e, f, g , respectively)



ROC curve analysis

ROC curve analysis was performed to detect the optimal cut-off values of Neu/HDL, Neu/Lym, Mono/HDL, Trig/HDL, HDL/LDL, Plt/Lym, and Lym/HDL for the evaluation of long-term clinical outcomes in our study population. The results are presented in Figure 1. The area under the curve (AUC) for Neu/HDL was 0.830 (p<0.001, 95% confidence interval [CI]: 0.753 to 0.908). The cut-off

Figure 2. Kaplan–Meier survival curves for long-term mortality after ACS according to Neu/HDL, Neu/Lym, Mono/HDL, Trig/HDL, and HDL/LDL (a, b, c, d, e)



a – Kaplan-Meier survival curves of long-term mortality according to Neu/HDL (log-rank test: p<0.001). **b** – Kaplan-Meier survival curves of long-term mortality according to Neu/Lym (log-rank test: p<0.001). **c** – Kaplan-Meier survival curves of long-term mortality according to Mono/HDL (log-rank test: p=0.006). **d** – Kaplan-Meier survival curves of long-term mortality according to Trig/HDL (log-rank test: p=0.038). **e** – Kaplan-Meier survival curves of long-term mortality according to HDL/LDL (log-rank test: p=0.095).

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Table 1. Baseline and angiographic characteristics of the groups according to cutoff points

Variable	Neu/HDL <0,269	Neu/HDL ≥0,269	p	Mono/HDL <0,018	Mono/HDL ≥0,018	p	Neu/Lym <5,322	Neu/Lym ≥5,322	p	Trig/HDL <3,647	Trig/HDL ≥3,647	p	HDL/LDL <0,330	HDL/LDL ≥0,330	p
Age	61.26±11.77	60.24±12.02	0.35	62.14±11.16	59.78±12.41	0.02	59.12±11.59	63.75±11.70	0.01	62.90±11.36	59.14±12.02	0.01	57.69±11.43	63.23±11.63	0.01
Smoking*	204 (52.3%)	84 (51.2%)	0.81	145 (51.4%)	143 (52.7%)	0.75	177 (52.9%)	111 (50.4%)	0.55	143 (53.3%)	145 (50.6%)	0.53	134 (59%)	154 (47.2%)	0.01
Gender (Male)	297 (76.1%)	129 (78.6%)	0.52	203 (71.9%)	222 (81.9)	0.01	258 (77.2%)	168 (76.3%)	0.81	210 (78.3%)	216 (75.5%)	0.43	189 (83.2%)	237 (72.6%)	0.01
HT	271 (69.4%)	114 (69.5%)	0.99	192 (68%)	192 (70.8%)	0.48	239 (71.5%)	146 (66.3%)	0.19	176 (65.5%)	209 (73%)	0.06	160 (70.4%)	224 (68.7%)	0.65
DM	173 (44.3%)	88 (53.6%)	0.04	133 (47.1%)	128 (47.2%)	0.98	162 (48.5%)	99 (45%)	0.41	105 (39.1%)	156 (54.5%)	0.01	108 (47.5%)	152 (46.6%)	0.82
CHD	63 (16.1%)	31 (18.9%)	0.43	47 (16.6%)	47 (17.3%)	0.83	56 (16.7%)	38 (17.2%)	0.87	42 (15.6%)	52 (18.1%)	0.43	34 (14.9%)	59 (18%)	0.33
CHF	73 (18.7%)	45 (27.4%)	0.02	56 (19.8%)	62 (22.8%)	0.38	66 (19.7%)	52 (23.6%)	0.27	70 (26.1%)	48 (16.7%)	0.01	38 (16.7%)	79 (24.2%)	0.03
AF	14 (3%)	5 (3%)	0.74	9 (3%)	10 (3%)	0.74	11 (3%)	8 (%3)	0.82	11 (4%)	8 (3%)	0.39	6 (2%)	12 (3%)	0.49
Diagnosis	–	–	0.77	–	–	0.48	–	–	0.17	–	–	0.37	–		0.29
Anterior	141 (36.2%)	62 (37.8%)	–	104 (386.9%)	99 (36.5%)	–	121 (36.2%)	82 (37.3%)	–	105 (39.2%)	98 (34.3%)	–	76 (33.5%)	127 (39%)	–
İnferior	174 (44.6%)	72 (43.9%)	–	125 (44.3%)	120 (44.3%)	–	141 (42.2%)	105 (47.7%)	–	119 (44.4%)	127 (44.4%)	–	104 (45.8%)	142 (43.6%)	–
Lateral	12 (3.1%)	3 (1.8%)	–	7 (2.5%)	8 (3%)	–	11 (3.3%)	4 (1.8%)	–	7 (2.6%)	8 (2.8%)	–	4 (1.8%)	11 (3.4%)	–
Posterior	5 (1.3%)	4 (2.4%)	–	2 (0.7%)	7 (2.6%)	–	4 (1.2%)	5 (2.3%)	–	2 (0.7%)	7 (2.4%)	–	3 (1.3%)	5 (1.5%)	–
Non-STEMI	58 (14.9%)	23 (14%)	–	44 (15.6%)	37 (13.7%)	–	57 (17.1%)	24 (10.9%)	–	35 (13.1%)	46 (16.1%)	–	40 (17.6%)	41 (12.6%)	–
Number of Diseased Vessel	–	–	0.56	–	–	0.47	–	–	0.17	–	–	0.80	–	–	0.55
1	210 (53.8%)	85 (51.8%)	–	143 (50.7%)	151 (55.7%)	–	188 (56.3%)	107 (48.6%)	–	141 (52.6%)	154 (53.8%)	–	123 (54.2%)	171 (52.5%)	–
2	125 (32.1%)	50 (30.5%)	–	95 (33.7%)	80 (29.5%)	–	101 (30.2%)	74 (33.6%)	–	88 (32.8%)	87 (30.4%)	–	74 (32.6%)	101 (31%)	–
3	55 (14.1%)	29 (17.7%)	–	44 (15.6%)	40 (14.8%)	–	45 (13.5%)	39 (17.7%)	–	39 (14.6%)	45 (15.7%)	–	30 (13.2%)	54 (16.6%)	–
Region of Culprit Lesion	–	–	0.059	–	–	0.981	–	–	0.491	–	–	0.748	–	–	0.232
Proximal	137 (35.1%)	71 (43.2%)	–	106 (37.5%)	102 (37.6%)	–	119 (35.6%)	89 (40.4%)	–	100 (37.3%)	108 (37.7%)	–	82 (36.1%)	126 (38.6%)	–
Mid	192 (49.2%)	78 (47.5%)	–	137 (48.5%)	133 (49%)	–	169 (50.5%)	101 (45.9%)	–	134 (%50)	136 (47.5%)	–	107 (47.1%)	162 (49.6%)	–
Distal	61 (15.6%)	15 (9%)	–	39 (13.8%)	36 (13.2%)	–	46 (13.7%)	30 (13.6)	–	34 (12.6%)	42 (14.6%)	–	38 (16.7%)	38 (11.6%)	–

Table 2. Post-hoc analyses of region of culprit lesions according to cutoff values

Dependent Variable				Mean Difference (I-J)	Std. Error	p	95% Confidence Interval	
							Lower Bound	Upper Bound
Neu/HDL	Tamhane	Proximal	Mid	0.05	0.04	0.53	-0.05	0.15
			Distal	0.14*	0.06	0.03	0.00	0.28
		Mid	Proximal	-0.05	0.04	0.53	-0.15	0.05
			Distal	0.09	0.05	0.24	-0.03	0.22
		Distal	Proximal	-0.14*	0.06	0.03	-0.28	-0.00
			Mid	-0.09	0.05	0.24	-0.22	0.03
Mono/HDL	Tukey HSD	Proximal	Mid	-0.00	0.04	0.99	-0.11	0.11
			Distal	0.01	0.07	0.98	-0.15	0.17
		Mid	Proximal	0.00	0.05	0.99	-0.11	0.11
			Distal	0.01	0.07	0.98	-0.14	0.17
		Distal	Proximal	-0.01	0.07	0.98	-0.17	0.15
			Mid	-0.01	0.07	0.98	-0.17	0.14
Neu/Lym	Tukey HSD	Proximal	Mid	0.05	0.04	0.45	-0.05	0.16
			Distal	0.03	0.07	0.86	-0.12	0.18
		Mid	Proximal	-0.05	0.05	0.45	-0.16	0.05
			Distal	-0.02	0.06	0.94	-0.17	0.12
		Distal	Proximal	-0.03	0.07	0.86	-0.18	0.12
			Mid	0.02	0.06	0.94	-0.12	0.17
Trig/HDL	Tukey HSD	Proximal	Mid	0.01	0.05	0.94	-0.09	0.12
			Distal	-0.03	0.07	0.87	-0.19	0.12
		Mid	Proximal	-0.01	0.05	0.94	-0.12	0.09
			Distal	-0.04	0.07	0.73	-0.20	0.10
		Distal	Proximal	0.03	0.07	0.87	-0.12	0.19
			Mid	0.05	0.07	0.73	-0.10	0.20
HDL/LDL	Tukey HSD	Proximal	Mid	0.00	0.05	0.99	-0.10	0.11
			Distal	0.10	0.07	0.24	-0.04	0.26
		Mid	Proximal	-0.00	0.05	0.99	-0.11	0.10
			Distal	0.10	0.06	0.24	-0.04	0.25
		Distal	Proximal	-0.10	0.07	0.24	-0.26	0.04
			Mid	-0.10	0.06	0.24	-0.25	0.04

point was 0.269, with sensitivity of 74.2% and specificity of 74.2%. The AUC for Neu/Lym was 0.688 (p<0.001, 95% CI: 0.586 to 0.790). The cut-off point was 5.322, with sensitivity of 67.7% and specificity of 67.1%. The AUC for Mono/HDL was 0.650 (p=0.005, 95% CI: 0.561 to 0.739). The cut-off point was 0.018, with sensitivity of 58.1% and specificity of 58.6%.

The AUC for Trig/HDL was 0.639 (p=0.009, 95% CI: 0.559 to 0.719). The cut-off point was 3.647, with sensitivity of 58.1% and specificity of 61%. The AUC for HDL/LDL was 0.626 (p=0.018, 95% CI: 0.526 to 0.727). The cut-off point was 0.330, with a sensitivity of 74.2% and specificity of 49.9%. The AUC for Plt/Lym was 0.559 (p=0.272, 95% CI: 0.442 to 0.675) and the AUC for Lym/HDL was 0.535 (p=0.51, 95% CI: 0.423 to 0.647). As results of Plt/Lym and Lym/HDL were not significant, the cut-off points were not calculated.

Baseline and Angiographic Characteristics

Baseline and angiographic characteristics are compared in Table 1 according to the cut-off points as described above. Significant differences for the mean age were observed in the Mono/HDL, Neu/Lym, Trig/HDL and HDL/LDL groups. Also, diabetes mellitus (DM) and chronic heart failure (CHF) prevalence were significantly different for Neu/HDL and Trig/HDL.

According to subgroups of diagnosis, the number of diseased vessels, and the regions of culprit lesions, there were no statistically significant differences for the cut-off points. Subgroup analyses were performed according to the regions of culprit lesions (Table 2). All cut-off points were compared, but for only the Neu/HDL cut-off point was there a significant difference between proximal and distal regions of culprit lesions.

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The Kaplan–Meier Curve was plotted with the event free survival data from the follow-up. The mean duration of follow-up was 503±7 days (median 520 days). 31 patients (5.5%) died during follow-up. Long-term mortality was significantly different according to Neu/HDL, Neu/Lym, Mono/HDL, and Trig/HDL cut-off points (p<0.001, p<0.001, p=0.006, p=0.038, respectively). In contrast, there was no significant difference for the HDL/LDL cut-off point (p=0.095). Kaplan-Meier survival analysis plots are shown in Figure 2.

Multivariate Cox regression analysis

Neu/HDL (hazard ratio, HR [confidence interval, CI]: 0.202 [0.075–0.545], p=0.002) and Neu/Lym (0.306 [0.120–0.777] p=0.013) were associated with increased risk of death in the multivariate Cox regression analysis. In contrast, Mono/HDL and Trig/HDL were not associated with increased risk of death. The multivariate Cox regression analysis for survival is shown in Table 3.

Table 3. Multivariate analysis for survival

Parameters	p	Exp (B)	95% Confidence Interval
Mono/HDL	0.051	0.430	0.185–1.003
Neu/HDL	0.002	0.202	0.075–0.545
Neu/Lym	0.013	0.306	0.120–0.777
Trig/HDL	0.266	0.632	0.282–1.418

Discussion

In present study, we found that a higher Neu/HDL ratio was associated with increased cardiovascular mortality. From the perspective as a mortality predictor, Neu/HDL had better performance than Neu/Lym, Mono/HDL, Trig/HDL, HDL/LDL, Plt/Lym, or Lym/HDL. In addition, Neu/HDL was associated with a significantly higher number in proximal culprit lesions.

Recent studies have focused on easily accessible, more reliable, and non-invasive methods for predicting cardiovascular mortality. In addition, researchers want to increase diagnostic accuracy of ACS with the addition of easily accessible and cost effective tests as performed by Zuzula et al. [23]. The balance between inflammatory/oxidative and cardiovascular protective biomarkers serve well for this purpose [24, 25]. In the literature, Neu/HDL, Neu/Lym, Mono/HDL, Trig/HDL, LDL/HDL, Plt/Lym, and Lym/HDL have been most frequently discussed.

Neu/Lym has been the most studied parameter among those mentioned above. Guasti et al. found that Neu/Lym was associated with a worse outcome in ACS [26]. Cetin et al. found that Mono/HDL was a predictor of severity of coronary artery disease and future cardiovascular events for ACS patients [27]. Another study found that LDL/HDL was related with sudden cardiac death [28]. Trig/HDL, Plt/Lym, and Lym/HDL were studied and were associated with ACS and metabolic syndrome [13, 16, 29]. Huang et al. compared the prognostic values of Neu/HDL, Mono/HDL, LDL/HDL for myocardial infarction in elderly patients and found that Neu/HDL had a more superior prognostic value than the others. In our study, we compared all these values for cardiovascular long-term survival after total coronary artery occlusion. ROC curve analysis showed a greater AUC (0.830) and minimum p value (p<0.001) for Neu/HDL. The Kaplan-Meier survival curve and multivariate Cox regression analyses showed a greater significance difference for Neu/HDL (p<0.001 and p=0.002, respectively). All these statistical analyses showed that Neu/HDL indicates better survival prediction for ACS. This can be attributed to acute clinical deterioration of our study population after myocardial infarction. All parameters in our study affect the plaque formation chronically except Neu. However, unlike the others, only Neu plays an acute role in myocardial plaque deformation.

Neu/Lym was the second best survival predictor in our study (ROC AUC=688 and p<0.001, Kaplan-Meier p<0.001, multivariate survival analysis p=0.013). Neu has an acute role in plaque rupture as mentioned before, however, lymphocytes affect the plaque formation more chronically, depending on cholesterol concentrations [30]. In addition, Neu and HDL negatively affect the other's functions [31]. This situation increased the difference between effects of Neu and HDL. In conclusion, we attribute Neu/HDL's better mortality prediction when compared to Neu/Lym for the reasons mentioned here.

The regions of culprit lesions were grouped according to BARI classification. It is a fact that more proximal culprit lesions cause more myocardial damage. Also, a larger myocardial damage area increases cardiovascular mortality. In a study by Chen et al., Neu/Lym was a predictor of myocardial damage in ACS patients [32]. All parameters were compared along with the regions of the culprit lesions and only Neu/HDL showed a significant difference between proximal and distal culprit lesions. This reinforced our hypothesis that Neu/HDL is a better mortality predictor than the other parameters (Neu/Lym, HDL/LDL, etc.).

This study has a number of limitations. First, it was a retrospective study, and, as such, its level of persuasion is slightly lower than prospective research. Prospective design studies are needed for more reliability. Secondly, only patients whose blood parameters were available in hospital records were included in this study. This situation may cause bias. Thirdly, the 554 patients were from a single center. Multi-center data with more patients are needed

in subsequent investigations. Finally, our results did not show a link between mortality prediction and the number of critical diseased vessels. This was contrary to our expectations. This link should be tested in later studies with larger sample sizes, different study design, and/or outcome measurements.

Conclusion

Long-term mortality prediction after ACS syndrome is still a concern among physicians. Many studies of this issue has been conducted, and various hematological parameters were used in these studies. In this study, we compared the seven most common parameters and found that Neu/HDL offers better mortality prediction than Neu/Lym, Mono/HDL, Trig/HDL, HDL/LDL, Plt/Lym, or Lym/HDL after total occlusion of coronary arteries.

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