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Research Article

The Relationship of Hematologic Parameters and Lipid Profile with Acute Ischemic Stroke

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Objective: This study aims to investigate the effect of routine laboratory parameters on clinical outcomes in patients with acute ischemic stroke (AIS).

Materials and Methods: Our study was designed as retrospective and cross-sectional. 94 patients with AIS who received inpatient treatment in our clinic with a diagnosis of acute ischemic stroke were included in the study. Laboratory data, demographic, and clinical characteristics were recorded at the time of admission. Neurological disabilities of the patients 3 months after treatment were evaluated with the modified Rankin Scale (mRS). mRS 0-2 was considered a good prognosis and mRS 3-6 was considered a poor prognosis.

Results: Of the 94 patients included in the study, 47 were men and 47 were women. The mean age was 71.18±11.92 years. Laboratory parameters hemoglobin, eosinophil, total-cholesterol, ldl-cholesterol, and triglyceride values were significantly lower in the group with mRS≥3. As a result of ROC analysis, the area under the curve of hemoglobin, eosinophil, total-cholesterol, ldl-cholesterol, and triglyceride was significantly higher[(AUC: 0.710 95%CI: 0.60-0.81); Sensitivity=67.6%, Specificity=67.9%, p=0.001]. In the multivariable logistic regression model, only the NIHSS score (National Institutes Of Health Stroke Scale Scores) was independently associated with the degree of disability (p=0.001). NIHSS is an independent factor in predicting stroke outcomes.

Conclusion: It was determined that there was a significant inverse relationship between the routine laboratory parameters hemoglobin level, eosinophil level, LDL and total cholesterol levels, and infarct volume with the NIHSS scores of hemoglobin and LDL-cholesterol.

Keywords: Acute ischemic stroke, Laboratory, Infarct volume, Clinical outcome

1. INTRODUCTION

Stroke is known as an important cause of mortality and morbidity worldwide.[1,2] Patients and their relatives always worry about the negative situations that may occur after a stroke. Clinicians' prediction of post-stroke prognosis will help determine stroke management. In this respect, it is important to predict the prognosis of stroke and take the necessary precautions.[3] In previous studies, complete blood count and biochemical tests were routinely used to indicate the prognosis of stroke. Studies have reported that routine laboratory tests may be useful in taking necessary steps to prevent the negative consequences of stroke.[3,4] In general, an increase in neutrophils, total white blood cells, and a decrease in lymphocytes are common laboratory findings during inflammation.[5] There are numerous studies to evaluate the relationship between hematological parameters and cardiovascular diseases. RDW (Red Blood Cell Distribution Width) and Mean platelet volume (MPV) are reliable prognostic parameters in cardiovascular diseases. Evaluation of white blood cells, NLR (neutrophil/lymphocyte ratio), monocyte/HDL-cholesterol, and PLR (platelet/lymphocyte ratio) can predict the

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prognosis of cardiovascular diseases.[6] Complete blood count and some biochemical parameters are routinely checked in stroke patients. These tests are easily accessible and cost-effective. In this respect, predicting prognosis after stroke will be easy and low-cost. In our study, we aimed to investigate the effect of routine laboratory parameters at admission on functional outcomes after stroke.

2. METHODS

2.1.Study population

This study was designed as a retrospective and cross-sectional study. 94 patients who were admitted to our clinic from the emergency department in the first 24 hours with the diagnosis of acute ischemic stroke and then came for outpatient clinic controls for at least 3 months were included. The patients' data were retrospectively examined from the hospital database following the ethics committee and institutional permissions. Strokes other than acute ischemic stroke, patients with missing brain CT, hemogram, and biochemical examinations during hospitalization, patients without outpatient clinic follow-up after discharge, patients with systemic diseases that would affect complete blood count parameters on admission, and patients with severe sequelae motor deficits upon admission were not included in the study. In this study; A total of 94 patients, who were 18 years of age and above, came for regular check-ups at the stroke outpatient clinic during the 3-month follow-up period after discharge, and had complete clinical data, were included in the study. Neurological disabilities of the patients 3 months after treatment were evaluated with Modified Rankin Scale (mRS) scores. Patients were divided into two groups according to Modified Rankin Scale (mRS) scores. mRS 0-2 was considered a good prognosis and mRS 3-6 was considered a poor prognosis. Ethics committee approval was received for the study and the study was conducted by the Ethical Standards of the Declaration of Helsinki. Since it was a retrospective file scan, patient consent could not be obtained.

2.2. Demographic, radiological examinations and laboratory

Anamnesis information, demographic characteristics, vascular risk factors [arterial hypertension (HT), diabetes mellitus (DM), coronary artery disease (CAD), Atrial Fibrillation (AF), hyperlipidemia (HL)], antiplatelet, statin, included in the prepared case report form. The presence of smoking was obtained from file data. Patients whose fasting blood sugar was 120 mg/dL and above in at least three measurements or who were previously diagnosed and received anti-diabetic treatment were diagnosed with DM. Patients who smoked at least half a pack/day and smoked for more than 1 year before the stroke were considered to be smokers. For the diagnosis of arterial hypertension, conditions were required: having been previously diagnosed and still using anti-hypertensive medication, or having an average blood pressure above 140/90 mmHg during hospitalization. HL was diagnosed in patients whose fasting blood total cholesterol and/or triglyceride levels were above 200 mg/dl or who were previously diagnosed with HL and were currently receiving anti-hyperlipidemia treatment.

Diffusion-weighted MRI (Magnetic Resonance Imaging) examinations of the patients taken at the time of initial admission were reviewed. In diffusion MRI (1.5 tesla, geo signa explorer, 2019), images were obtained using a section thickness of 4 mm. The volume of the ischemic area was calculated with the 'Region of Interest (ROI)' method in the diffusion-weighted imaging seen at the time of admission 7 and the infarct volume (cm3) was recorded.

After an overnight fast of at least 12 hours, complete blood count, total cholesterol (TC), high-density lipoprotein cholesterol (Hdl-cholesterol), low-density lipoprotein cholesterol (Ldl-cholesterol) and triglyceride (TG) concentrations, and liver and kidney function tests were measured by routine laboratory methods.

2.3. Statistical analysis

The suitability of continuous variables to normal distribution was examined with the Kolmogorov-Smirnov test. According to the results of the normality test, variables that comply with normal distribution are given with their mean and standard deviation, and variables that do not comply with normal distribution are given with their median, minimum, and maximum values. According to the normality test results, Mann Whitney U test and Independent paired sample t-test were used for intergroup comparisons of continuous variables. Categorical variables were compared between groups using the chi-square test and Fisher's exact chi-square test. The relationships between continuous variables were examined by correlation analysis and the Spearman correlation coefficient was calculated. ROC analysis was performed to determine the cut-off point for hemoglobin, eosinophil, LDL-cholesterol, and triglyceride levels to predict mRS ≥3. Factors that are effective in observing poor prognosis were examined by logistic regression analysis. SPSS (IBM SPSS Statistics for Windows, Version 25.0. Armonk, NY: IBM Corp.) program was used for statistical analysis, and $p<0.05$ was considered statistically significant.

3. RESULTS

Of the 94 patients included in the study, 47 were male (50%) and 47 were female (50%). The mean age was 71.18± 11.92 years. It was determined that the incidence of hypertension, heart failure, and AF was higher in the poor prognosis group (84.2% & 46.4%; $p<0.001$, 21.1% & 5.4%; $p=0.04$, 50% & 26%, respectively). 8; $p=0.03$). Median infarct volume was also higher in the group with mRS ≥3 (11.22 & 1.37; $p<0.001$). NIHSS score was also determined to be higher in the group with mRS ≥3 ($p<0.001$). The mean age was also higher in the group with mRS ≥3 (75.23±10.50; $p=0.006$). Other demographic and vascular risk factors did not differ significantly between the groups ($p>0.05$), (Table 1). As a result of the comparison of laboratory parameters between the groups, hemoglobin, eosinophil, Ldl-cholesterol, total-cholesterol, and triglyceride values were significantly lower in the poor prognosis group (respectively; $p\leq0.001$, $p=0.01$, $p=0.01$, $p=0.02$, $p=0.01$). No differences were detected between the groups in terms of other laboratory parameters (Table 2).

ROC (Receiver Operator Characteristic Curve) analysis was performed to determine the cut-off point of hemoglobin, eosinophil, Ldl-cholesterol, total-cholesterol, and triglyceride to predict poor prognosis. As a result of ROC analysis, the area under the curve of hemoglobin, eosinophil, Ldl-cholesterol, total-cholesterol, and triglyceride was significantly higher (Figure 1). Hemoglobin (mg/dl) had the highest area under the curve with a cut-off value of 13.35. [(AUC: 0.710 95% CI:0.60-0.81); Sensitivity = 67.6%, Specificity = 67.9%, $p=0.001$]. The cut-off value for eosinophils (103/µl) was 0.12 (103/µl) [(AUC: 0.646 95% CI:0.53-0.76); Sensitivity = 64.9%, Specificity = 57.1%, $p=0.01$]. The cut-off value for Ldl-Cholesterol was 124.5 mg/dl [(AUC: 0.64195% CI: (0.52-0.76); Sensitivity = 64.9%, Specificity = 64.3%, p = 0, 02], the cut-off value for total-cholesterol was 179.5 mg/dl [(AUC: 0.64495% CI: (0.52-0.76); Sensitivity = 62.2.9%, Specificity = 69.6%. , $p=0.01$], the cut-off value for triglyceride was 121.5 mg/dl [(AUC: 0.64495% CI: (0.53-0.75); Sensitivity = 56.8%, Specificity = 58%, 9, $p=0.01$], (Table 3).

Table 1.

Comparison of demographic, clinical characteristics and vascular risk factors between groups

	Total (n=94)	**mRS <3 (n=56,%59,4)**	**mRS ≥3 (n=38,%40,4)**	**Test statistics**	***P***
Age (year)	71,18± 11,92	68,42±12,12	75,23±10,50	**-**2,81	**0,006**[a]
Gender, n (%)					
• Male • Female	47 (50) 47 (50)	30 (53,6) 26 (46,4)	17 (44,7) 21 (55,3	0,39	0,52[b]
Smoking, n (%)	22 (23,4)	15 (26,8)	7 (18,4)	0,47	0,48[b]
Antiplatelets, n (%)	39 (41,50)	19 (33,9)	20 (52,6)	2,5	0,11[b]
Statin, n (%)	10 (10,60)	5 (8,9)	5 (13,2)	-	0,51[c]
HT, n (%)	58 (61,70)	26 (46,4)	32 (84,2)	12,12	**<0,001**[b]
DM, n (%)	38 (40,40)	20 (35,7)	18 (47,4)	0,83	0,36[b]
HL, n(%)	13 (13,80)	7 (12,5)	6 (15,8)	0,02	0,88[b]
HF, n (%)	11 (11,70)	3 (5,4)	8 (21,1)	**-**	**0,04**[c]
CAD, n (%)	28 (29,80)	13 (23,20)	15 (39,5)	2,13	0,14[b]
AF, n (%)	34 (36,20)	15 (26,8)	19 (50)	4,32	**0,03**[b]
Infarct Volume (cm^3)	27,94 (0,04:356,48)	1,37 (0,04:66,47)/38,29	11,22 (0,18:356,48)/61,07	Z= -3,97	**<0,001**[d]
NIHSS_Initial	4 (1:25)	2 (1:20)/32,47	11 (2:25)/69,64	Z= -6,52	<0,001[d]

AF: Atrial Fibrillation, DM: Diabetes Mellitus, HL: Hyperlipidemia, HT: Hypertension, CAD: Coronary Artery Disease, HF: Heart Failure, NIHSS: The National Institutes of Health Stroke Scale, mRS: Modified Rankin Scale

mean ± standard deviation, median (minimum: maximum)/mean Rank and number (%)

a: t-Test for independent paired samples, b: Continuity correction, c: Fisher's Exact Chi-Square Test, d: Mann Whitney U Test

Table 2.

Comparison of laboratory parameters between groups

	Total (n=94)	mRS <3 (n=56)	mRS ≥3 (n=38)	Test statistics	*p*
Hemoglobin (g/dL)	13,32±2,07	13,97±1,75	12,35±2,13	t=4,01	**<0,001**[a]
WBC (*10³/µl)	8,45±2,27	8,38±2,12	8,53±2,49	Z=-0,29	0,76[a]
Eosinophil, (*10³/µl)	0,12 (0,01:0,55)	0,16 (0,01:0,55)/52,96	0,10 (0,01:0,47)/39,46	Z= -2,35	**0,01**[b]
Lymphocyte, (*10³/µl)	2,02 (0,21:5,70)	2,21 (0,64:5,70)/52,07	1,84 (0,21:5,46)/40,76	Z= -1,97	0,05[b]
ELR	0,06 (0:0,37)	0,07 (0:0,37)/51,47	0,04 (0:0,29)/41,64	Z=-1,71	0,08[b]
Neutrophil, (*10³/µl)	5,52 (1,85:13,08)	5,31 (1,85:11,18)/45,89	5,45 (1,96:13,08)/49,87	Z=-0,69	0,48[b]
Platelet, (*10³/µl)	251,34±76,33	247,10±68,60	257,57±87,07	t=-0,65	0,51[a]
PLR	107,71 (36,33:966,67)	101,24 (36,33:323,44)/43,79	110,40 (59,52:966,67)/52,96	Z=-1,59	0,11[b]
MPV, (*10³/µl)	9,57±1,10	9,71±1,09	9,37±1,07	1,48	0,14[a]
Hdl-Cholesterol, mg/dl	40,69±9	42,03±8,87	38,64±9,16	1,78	0,07[a]
Ldl-Cholesterol, mg/dl	125,33±39,39	133,10±37,83	113,56±39,28	2,40	**0,01**[a]
Total Cholesterol, mg/dl	190,60±51,01	200,07±51,75	176,27±46,98	2,35	**0,02**[a]
Triglyceride, mg/dl,	124(13:431)	133,00 (43:431)/52,32	115,00 (13:234)/38,95	Z=-2,33	**0,01**[b]
TC/HDL	4,77±1,20	4,85±1,18	4,66±1,24	0,70	0,48[a]
TG/HDL	3,35 (0,28:11,65)	3,47(0,84:11,65)/49,38	2,97(0,28:5,68)/43,41	1,60	0,08[a]

ELR: Eosinophil/Lymphocyte Ratio, MPV: Mean Platelet Volume, Hdl: High-density lipoprotein, Ldl: Low-density lipoprotein, PLR: Platelet/Lymphocyte Ratio, TC: Total Cholesterol, TG: Triglyceride

median (minimum: maximum)/mean Rank and mean ± standard deviation

a: t-Test for independent paired samples, b: Mann Whitney U Test

Table 3.

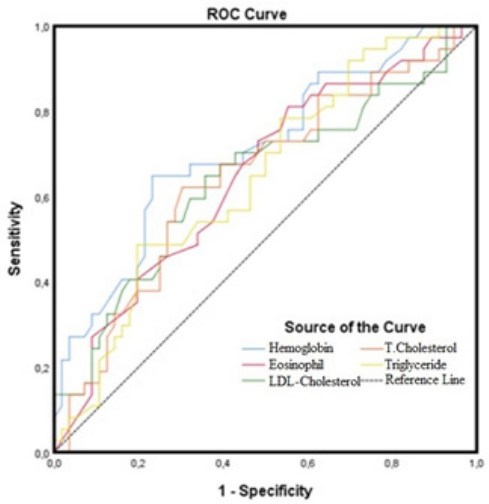
Area under curve (AUC), Sensitivity and Specificity

	AUC (%95 CI)	Cut off	Sensitivity	Specificity	P
Hemoglobin, (g/dl)	0,710 (0,60-0,81)	≤13,35	0,676	0,679	0,001
Eosinophil, (*103/µl)	0,646 (0,53-0,76)	≤0,12	0,649	0,571	0,01
Ldl-Cholesterol, mg/dl	0,641 (0,52-0,76)	≤124,5	0,649	0,643	0,02
Total Cholesterol, mg/dl	0,644 (0,52-0,76)	≤179,5	0,622	0,696	0,01
Triglyceride, mg/dl	0,644 (0,53-0,75)	≤121,5	0,568	0,589	0,01

Ldl: Low-density lipoprotein, AUC: Area under curve, CI: Confidence Interval

Figure 1.

ROC curve showing the predictive value of routine laboratory parameters for poor prognosis



Variables were included in the univariate and multivariate logistic regression model to identify risk factors for predicting poor prognosis. The results of univariate analyses and multivariate analyses are presented in Table 4, and the risk factors that are thought to affect poor prognosis are stated in the table. As a result of the analysis, it was seen that the multivariate logistic regression model was compatible with the data (Hosmer and Lemeshow test p=0.66) and the resulting logistic regression model was significant (p<0.001). As a result of the analysis, it was determined that the NIHSS score was 1.34 times higher than the risk of mRS ≥3 in the good prognosis group. In the multivariate logistic regression model, only the NIHSS score was independently associated with the degree of disability (p=0.001), (Table 4).

In the correlation analysis, it was determined that there was a significant negative relationship between laboratory values such as hemoglobin level (rs= -0.26; p=0.011), eosinophil level (rs= -0.24; p=0.021), Ldl-Cholesterol (rs= -0.28; p=0.006), and total-cholesterol levels (rs = -0.27; p = 0.008) and infarct volume. It was determined that there was a significant negative relationship between the laboratory parameters hemoglobin (rs= -0.23; p=0.02) and Ldl-cholesterol (rs= -0.21; p=0.04) and the NIHSS score (Table 5). No significant relationship was detected between the other parameters listed in Table 5 and infarct volume and NIHSS (p>0.05).

Table 4.

Analysis of parameters affecting clinical outcome using logistic regression model

	Univariate Logistic Regression Model		Multivariate Logistic Regression Model (Enter)	
	OR (%95CI)	***p***	**OR (%95CI)**	***p***
Age (year)	1,05 (1,01:1,09)	**0,008**	1,02 (0,95:1,09)	0,47
HT	6,15 (2,22:17,03)	**<0,001**	3,58 (0,77:16,45)	0,10
HF	4,71 (1,16:19,11)	**0,03**	1,10 (0,65:18,70)	0,94
AF	2,73 (1,14:6,51)	**0,02**	0,80 (0,18-3,41)	0,76
Infarct Volume (cm3)	1,05 (1,01:1,08)	**0,006**	1,02 (0,99:1,05)	0,19
NIHSS_Initial	1,41 (1,21:1,64)	**<0,001**	1,34 (1,12:1,60)	**0,001**
Hemoglobin (g/dl)	0,64 (0,49:0,82)	**0,001**	0,70 (0,44:1,11)	0,13
Eosinophil , (*103/µl)	0,01 (0,001:0,55)	**0,02**	0,03 (0,01-15,05)	0,27
Ldl-Cholesterol , mg/dl	0,98 (0,97:0,9)	**0,02**	1.00 (0,95:1,04)	0,85
Total Cholesterol , mg/dl	0,99 (0,98:0,99)	**0,03**	0,99 (0,96:1,04)	0,80
Triglyceride , mg/dl,	1 (0,98:0,99)	**0,02**	0,99 (0,97:1,01)	0,54
Constant			1,27	0,96

AF: Atrial Fibrillation, HT: Hypertension, HF: Heart Failure, NIHSS: The National Institutes of Health Stroke Scale, OR: Odds ratio, CI: Confidence Interval, Cox&Snell R2=0,50; Nagelkerke R2=0,68; Hosmer and Lemeshow ChiSquare=5,86

Table 5.

Relationship of laboratory parameters with infarct volume and NIHSS

	Infarct Volume	p	NIHSS	p
Monocyte (*103/µl)	0,14	0,18	0,06	0,57
Hemoglobin (g/dL)	-0,26	**0,01**	-0,23	**0,02**
WBC (*103/µl)	0,14	0,194	-0,10	0,33
Eosinophil, (*103/µl)	-0,24	**0,02**	-0,14	0,17
Lymphocyte , (*103/µl)	-0,07	0,50	-0,15	0,14
ELR	-0,17	0,10	-0,10	0,30
Neutrophil, (*103/µl)	0,15	0,13	-0,07	0,45
Platelet, (*103/µl)	0,01	0,89	-0,17	0,09
PLR	0,09	0,36	0,01	0,91
MPV, (*103/µl)	-0,09	0,39	-0,08	0,42
Hdl-Cholesterol, mg/dl	-0,12	0,26	-0,15	0,15
Ldl-Cholesterol, mg/dl	-0,28	**0,006**	-0,21	**0,04**
Total Cholesterol, mg/dl	-0,27	**0,008**	-0,19	0,06
Triglyceride, mg/dl	-0,15	0,162	-0,15	0,14
TC/HDL	-0,18	0,07	-0,08	0,43
TG/HDL	-0,06	0,52	-0,05	0,61

rs: Spearman Correlation Coefficient

ELR: Eosinophil/Lymphocyte Ratio, WBC: White Blood Cells, MPV: Mean Platelet Volume, Hdl: High-density lipoprotein, Ldl: Low-density lipoprotein, PLR: Platelet/Lymphocyte Ratio, TC: Total Cholesterol, TG: Triglyceride, NIHSS: The National Institutes of Health Stroke Scale

4. DISCUSSION

In our study, laboratory parameters; hemoglobin, eosinophil, Ldl-cholesterol, total-cholesterol, and triglyceride values were found to be low in the poor prognosis group. It is vital to identify risk factors affecting prognosis after stroke and to implement treatments to reduce the incidence of recurrent ischemic events. Some blood biomarkers can guide etiology, therapeutic approach, follow-up, and functional prognosis in acute ischemic stroke patients.[8] Many studies have been conducted on the effects of changes in hemogram and biochemistry parameters as a result of hypercortisolism and sympathetic hyperactivity resulting from the acute stress reaction associated with acute ischemic stroke on stroke prognosis. As a result of the studies conducted in this context, although the increase in the neutrophil/lymphocyte ratio and eosinopenia are among the poor prognostic factors, the reason for the relationship between biomarkers and prognosis is not fully known. [9-11] Similar to the literature, eosinopenia was detected in the poor prognosis group in our study. The relationship between hemoglobin level and post-stroke outcomes is contradictory. Some studies have revealed that low hemoglobin value at admission is associated with mortality and large infarct volume in stroke patients. This is thought to be caused by low hemoglobin levels that may reduce oxygen transport to ischemic penumbral regions.[12,13] However, in the study conducted by Zhang R. et al., it was reported that both low and high hemoglobin levels at the time of admission were associated with poor prognosis after AIS.[14] In another study investigating the factors affecting mortality, it was observed that hemoglobin and hematocrit levels were low and NIHSS scores were high in the poor prognosis group.[15] In our study, hemoglobin levels were found to be low in the poor prognosis group. It also showed a significant inverse correlation with both infarct volume and admission NIHSS score. It is thought that there is a decrease in oxygen-carrying capacity as a result of low hemoglobin levels.

İçme et al. showed that mean platelet volume (MPV) may be an important indicator of prognosis in AIS.[16] However, similar to our results, other studies[17,18] have reported that MPV does not have any prognostic significance in ischemic stroke. Additionally, similar to our study, it has been shown that leukocyte, neutrophil, lymphocyte, and platelet counts do not have prognostic significance. [16,19]

It has been reported that cholesterol is necessary for normal membrane function in the vascular system and that adequate cholesterol levels may be important for maintaining the integrity of the vessels and their resistance to tearing. [20] Hypercholesterolemia is a well-established risk factor for cardiovascular morbidity and mortality. However, the relationship between ischemic stroke and lipid profile is complex and studies contain paradoxical results. [21-24] Hyperlipidemia causes atherosclerosis as a result of lipid accumulation in the intima layer of the vessels, and atherosclerosis also creates a basis for stroke. [25,26] It is thought that the inverse correlation between acute ischemic stroke prognosis and LDL-cholesterol and triglyceride levels observed in some studies is due to lipid disorders causing small vessel strokes.[21-23] In our study, total-cholesterol, Ldl-cholesterol, and triglyceride values were significantly lower in the poor prognosis group, and this was consistent with studies. Also, it has been found that triglyceride and total cholesterol levels in stroke patients on admission may be a predictor of in-hospital mortality. [24] Han et al.'s study revealed a nonlinear relationship and a threshold value between the TG/HDL-c ratio and 3-month adverse outcomes in patients with AIS. [27] When the TG/HDL-c ratio is below 3.51, the prognosis worsens as the TG/

HDL-c ratio decreases. When the TG/HDL-c ratio is above 3.51, the prognosis worsens as the TG/HDL-c ratio increases. [27] In our study, the TG/HDL-c ratio was evaluated below the threshold value and was consistent with these results.

Although direct evidence of the relationship between eosinophils and thrombosis cannot be obtained by thrombus analysis in patients with acute ischemic stroke, histopathological findings suggest that eosinophils may play an important role in thrombus formation.[11] Wang et al. showed that there was a significant inverse correlation between NIHSS and WBC (White blood cells), eosinophils, eosinophil percentage, and LDL-cholesterol.[11] In our study, it was observed that only hemoglobin and LDL-cholesterol had a negative significant relationship with the NIHSS score, and did not show a significant correlation with other parameters. The literature is examined; it was thought that factors such as the timing of samples taken from patients, calculation of differences by seeing control values, and the number of patients may cause the difference in results.

In multivariate regression analysis, it was determined that laboratory parameters were not significant and admission NIHSS score was the only predictor of poor prognosis.

Our study has some limitations. First, the study reflects the results of a single center after a retrospective review, and therefore generalizations cannot be made. Secondly, it was made with values obtained as a result of a measurement in a limited number of cases. To investigate the prognostic value of hematological and biochemical parameters in stroke, studies with a prospective design and larger sample size will provide more accurate results. Stronger statistical results will be obtained with more participants.

NIHSS is an independent factor in predicting stroke outcomes. In this study, a relationship was found between routine laboratory parameters hemoglobin level, eosinophil level, LDL and total cholesterol levels, and infarct volume. Additionally, it was determined that there was a significant negative correlation between hemoglobin and LDL-cholesterol and NIHSS scores.

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Disclosure of interest

The authors declare that there is no conflict of interest.

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Ethics Committee Approval

The study protocol was approved by the local ethics of Kutahya Health Sciences University (2024/06-27), and approval and local institutional approvals were obtained for the study.

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