Ann Med Res

Current issue list available at AnnMedRes

Annals of Medical Research

journal page: www.annalsmedres.org

Investigation of inflammation marker ratios that can be used as predictors of ın-hospital mortality in traumatic acute subdural hematoma

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ARTICLE INFO

Keywords:
C-reactive protein/albumin
C-reactive protein/lymphocyte
Mortality
Mean platelet volume/platelet count
Traumatic acute subdural hematoma

Received: May 10, 2023
Accepted: Jun 19, 2023
Available Online: 23.06.2023

DOI:
10.5455/annalsmedres.2023.05.110

Abstract

Aim: The aim of the present study is to determine inflammatory mediators that may be used in predicting in-hospital mortality in traumatic acute subdural hematoma (TASDH) patients and to evaluate their significance as prognostic markers.

Materials and Methods: The medical records of adult patients with traumatic acute subdural hematoma admitted to the emergency department were reviewed retrospectively. The primary clinical outcome was in-hospital mortality. Univariate and multivariate Cox regression analyses were performed for the identification of independent predictors of in-hospital mortality. Besides, receiver operating characteristic (ROC) analysis was applied to determine the power of inflammatory mediators in predicting mortality.

Results: According to the Cox hazard models, the mean platelet volume (MPV) /platelet count (PLT) ratio (MPR) (Hazard Ratio (HR) = 1.129; 95% CI = 1.059 – 1.204; p<0.001), C-reactive protein (CRP)/lymphocyte ratio (CLR) (HR = 1.011; 95% CI = 1.002 – 1.020; p = 0.022) and C-reactive protein(CRP)/albumin ratio (CAR) (HR =1.004; 95% CI = 1.001 – 1.007; p = 0.004 were risk factors for in-hospital mortality. The results of ROC analysis indicated that the MPR of 0.06 or above predicted in-hospital mortality with 67% sensitivity and 88% specificity (p = 0.001). In addition, the CLR of 39.12 and above predicted in-hospital mortality with 63% sensitivity and 93% specificity and the CAR of 8.79 (p = 0.001) and above predicted it with 66% sensitivity and 87% specificity (p = 0.002).

Conclusion: Increased MPR, CLR and CAR values are strong predictors of in-hospital mortality in TASDH patients.

Introduction

Subdural hematoma is a type of hemorrhage that occurs due to bleeding of the bridging veins, which is usually located under the dura [1]. Traumatic Acute Subdural Hematoma (TASDH), which is an emergency neurological condition, is an important public health problem due to its high mortality and morbidity [2]. In the literature, its mortality rate has been stated to range in a wide interval; there are studies reporting mortality rates of 27% [3] and 75% [4]. Although the exact prevalence of TASDH is not known, ASDH has been observed on computed brain tomography of 12-29% of patients with traumatic brain injury [5]. The mortality rate in these patients is influenced by patient age and various factors such as duration of postoperative intensive care unit (ICU) stay [4]. The main aim of the treatment applied to patients with TASDH is to lower mortality and morbidity. Clinopathological studies toward reducing mortality and morbidity are currently in progress. In the reduction of mortality and morbidity in TASDH patients, each step from the time of admission to the hospital to the time of discharge and subsequent rehabilitation of patients is individually important.

One of the major etiologies of mortality is inflammation. Platelet size has been indicated to reflect platelet activity and appears to be a helpful predictor and prognostic biomarker of cardiovascular events [6]. It is associated with various prothrombotic and pro-inflammatory diseases [6]. Inflammation is a series of vital responses that the body gives to tissue damage through cellular, hormonal and vascular pathways [7]. Although inflammation is a pathological condition under normal circumstances, an inflammatory reaction is a response the body exhibits physiologically [8]. In the literature, there are studies regarding

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many inflammatory mediator associated with the mortality of neurosurgical diseases such as stroke, however, the information about the relationship between mortality and morbidity of TASDH patients and their hematological parameters at the time of admission to the hospital is very limited [9].

TASDH causes traumatic stress reaction in the body and an increase in various inflammation markers related to it [8]. In recent studies, it has been shown that various inflammatory mediators, including MPV, CLR, neutrophil count/ lymphocyte count (NLR), platelet/ lymphocyte ratio (PLR) and MPR, calculated from the results of complete blood count and biochemical blood parameters determined at the first admission in the emergency department, are effective in mortality associated with various diseases [10]. Lymphocytes have a role in the body's defense system [11]. A decreased lymphocytes ratio has been associated with malnutrition [11]. MPV is a value obtained by calculating platelet size in the complete blood count test, which also includes platelet count. Studies regarding the role of MPV in inflammation are available in the literature [10]. Large platelets containing more dense granules are more active in terms of metabolic and enzymatic properties than small platelets and carry a higher thrombotic potential [12]. Albumin level is a direct indicator of malnutrition [11]. CRP is a parameter that increases in acute inflammation [10].

Systemic inflammation, which is considered as an integral part of disease progression in critical diseases, is often associated with sepsis and causes an increased risk of mortality [13]. Recently, CLR, NLR, PLR and MPR have been investigated as promising predictors of mortality in sepsis patients in critical condition [13]. The aim in our study was to determine inflammatory markers that may be used as mortality predictors in TASDH patients and to investigate whether these values can be utilized to reduce mortality and morbidity.

Materials and Methods

The research was planned as a retrospective cohort study after receiving local ethical committee approval (Tokat Gaziosmanpasa University Clinical Research Ethics Committee, 22-KAEK-113). In the study, the medical files of 148 patients who were admitted to the emergency department between 2011 and 2020 with the diagnosis of TASDH in a single center (Tokat Gaziosmanpasa University Faculty of Medicine) were examined. The patients were separated into two groups as alive and deceased with respect to in-hospital mortality. Inclusion criteria were patients with traumatic acute subdural hematoma, older than 18 years, no history of malignancy, no additional injuries (e.g., pelvis fracture), no hematological disease, and those who did not get medical treatment that impairs platelet function. Exclusion criteria, on the other hand, were identified as patients with the presence of intracranial space-occupying lesion, those with multiple fractures, cancer patients undergoing chemotherapy treatment, as well as those with incomplete clinical data and those with severe cognitive impairment. The data of the study were collected from the patients' anesthesia and medical records using the electronic patient record system (ENLIL hospital information management system, version v2.19.46 20191118) of the hospital. Blood tests were routinely performed for each patient during the admission to the emergency department. The primary outcome was the MPR calculated based on the in-hospital measurement performed at the time of the first admission to the emergency department. In this study, it was aimed to reveal the relationship between this ratio and mortality as well as to determine differences in specificity and sensitivity values in predicting mortality between MPR and other inflammatory mediators.

The relationship of mortality with factors including gender, age groups, Charlson comorbidity index (CCI) and preoperative American Society of Anesthesiologists scores, comorbidities, body mass index, length of stay in intensive care unit (ICU), postoperative length of hospital stay, blood gas, international normalized ratio (INR), blood urea nitrogen (BUN), creatinine, CRP, albumin, biochemical parameters (alanine aminotransferase, activated partial thromboplastin time, aspartate aminotransferase, glucose, calcium, potassium, chloride, sodium, hematocrit (HCT), red blood cell distribution width (RDW), white blood cell (WBC) and artery blood pH, potassium, sodium, calcium, chloride lactate and creatinine) and undergoing surgery was evaluated using the blood picture at the time of admission to the emergency department (Table 1). In addition, the data of platelet count ($\times 10^9$/L, reference interval: 173–390), lymphocyte count ($\times 10^3$/L, reference interval: 1.26–3.31), hemoglobin (HGB, g/L, reference interval: 11.90–14.6), neutrophil count ($\times 10^3$/L, reference interval: 2.1–8.86) and monocyte count ($\times 10^3$/L, reference interval: 0.25–0.84) were collected.

To evaluate the thickness of hemorrhage, in the axial section of brain computed tomography (CT), the diameter at which the hemorrhage was widest was measured by using the PACS system (Sectra Workstation IDS7, Version 21.2.11.6289, ©2019 Sectra AB). In our hospital, blood samples are collected into test tubes containing ethylene diamine tetra acetic acid (EDTA). Bloods taken in the emergency service are transported to the Blood Center of the hospital by a pneumatic system for complete blood count and analyzed within 30 min. CLR was calculated by dividing CRP by lymphocyte count, MPR was obtained by dividing MPV by platelet count, CAR was determined by dividing CRP by albumin, PLR was found by dividing platelet by lymphocyte and NLR was obtained by dividing neutrophil count by lymphocyte count.

The patients were categorized according to the ASA score ranging from 1 to 5, which is a subjective measure of preoperative physical condition. There were no patients with ASA scores of 1 or 5 in our study cohort, and the patients were separated into three groups according to their ASA scores (2, 3, 4). We evaluated whether MPR can be utilized as a prognostic marker to predict early mortality. All patients continued to take the medicines they regularly use due to their comorbid diseases and used antiembolism stockings as prophylaxis against deep vein thrombosis.

Statistical analysis

Quantitative variables were expressed as mean ± standard deviation. Shapiro Wilk test was used to test for

normality of continuous variables. The independent sample t test and one-way analysis of variance were used to compare the normally distributed continuous data between/among groups. Pairwise post-hoc comparisons between the groups were performed by the Tukey HSD test. Qualitative variables were presented as frequency and percentage. The chi-square test was employed to compare the categorical data between/among groups. Receiver operating characteristic (ROC) analysis was applied to determine the power of inflammatory mediators calculated in predicting mortality. Cox regression analysis was used for the variables Heart failure, DM, MPR, CLR, CAR, NLR, PLR associated with in-hospital mortality. A p-value of <0.05 was regarded significant for all analyses. All statistical calculations were performed using the SPSS 22 software (IBM SPSS Statistics 22, SPSS inc., an IBM Co., Somers, NY).

Results

A total of 45 females and 103 males with the mean age of 62.18 ± 21.01 were included in the study after applying the exclusion criteria. The mean BMI of the patients was 25.71 $\pm$ 2.49 kg/m^2. Thirty-six patients deceased in the hospital and surgical intervention was required in 43 patients. Surgical history was present in 11.1% of the deceased patients and 34.8% of the alive patients ($p<0.006$). Heart failure and diabetes mellitus (DM) were more common in the patients who deceased. The univariate analysis conducted revealed that heart failure increased the mortality risk by 3.3 times (HR = 3.322; 95.0% CI = 1.428-7.731; p = 0.005) and DM by 3.01 times (HR = 3.149; 95.0% CI = 1.478-6.711; p = 0.003). Postoperative mortality is higher in patients with high CCI and ASA scores [14]. The CCI and ASA scores of the deceased patients were significantly higher ($p<0.001$). The comorbid diseases are shown in Table 1.

Among the in-hospital mortality patients, the values of MPR, CLR, CAR, NLR and PLR were detected to be higher ($p<0.001$). Figure 1 demonstrates the comparison between the deceased and alive patients in terms of MPR.

Although the thickness of hemorrhage was higher in-hospital mortality patients, its effect on mortality was detected to be statistically insignificant (p = 0.954). According to the results obtained in the univariate analysis, one unit increase in MPR increased the mortality risk by 1.1 times (HR = 1.104; 95.0% CI = 1.055-1.156; $p<0.001$) and one unit increase in CAR increased the mortality risk by 1.02 times (HR = 1.022; 95.0% CI = 1.003-1.041; p = 0.001). The blood parameters belonging to both groups are given in Table 2.

The multivariate COX analysis performed indicated that one unit increase in MPR increased the mortality risk by 1.1 times, and one unit increase in NLR increased the mortality risk by 1.01 times (Table 3).

The results of ROC analysis indicated that MPR of 0.06 and above predicts mortality with 67% sensitivity and 88% specificity (AUC=0.69; p=0.001), whereas CLR of 39.12 or above predicts it with 63% sensitivity and 93% specificity (AUC=0.71; p=0.001) and CAR of 8.79 or above predicts it with 66% sensitivity and 87% specificity (AUC=0.68; p=0.002).

Discussion

To the best our knowledge, our study is the first study in the literature that shows the relationship between the prediction of in-hospital mortality and all blood parameters at the time of first admission to the emergency department and the ratios obtained from these parameters showing inflammation (including parameters such as MPV, MPR, CLR, CAR, NLR and PLR) in TASDH patients. In our study, we detected that MPR, CLR and CAR predicted the mortality alone in TASDH patients. The outcomes of the presents study emphasize not only the importance of blood parameters detected at the first admission to the emergency department in mortality prediction, but also significance of albumin and CRP, which are not routinely examined in the blood samples of these patients collected in the emergency department.

Platelets is known to have a substantial role in the pathophysiology of ischemic stroke through the development of intravascular thrombus after erosion or rupture of atherosclerotic plaques [15], whereas thrombocytopenia simultaneously occurs with cerebral hemorrhage [16]. In TASDH patients, endothelial damage develops due to hemorrhage [17]. An increase in the release of adhesion molecules depending on endothelial damage occurs [17]. The body gives a response by reducing the production of antiaggregant factors such as nitric oxide (NO) and prostacyclin [17]. As a consequence, platelets become active and clots occur, which is reflected as a decrease in platelet count in blood tests [17]. Platelets have the ability to initiate inflammation [18]. In addition, they play a very significant role in the pathogenesis of early brain damage and delayed cerebral ischemia. In patients with brain damage, platelets form thrombi that reduce cerebral blood flow [19]. In a study, platelets were shown to respond to subarachnoid hemorrhage in the acute phase (within a few hours) [18]. They suggested the development of treatments that intervene platelets' ability to initiate inflammation without the impairment of hemostasis [18]. In our study, as a result of examining the blood values determined at the first admission to the emergency department, we are of the opinion that the reason for the high mortality in the patients with low platelet counts in the acute period can be linked to the triggered inflammation and occurred microthrombi, and that the reason for decreased platelet is due to microthrombi. We detected that the platelet count decreased and MPR increased at the time of the first admission after hemorrhage in the patients with high mortality. This condition, which was independent of the clinical features of patients, supports the fact that hemorrhage somehow initiates platelet activation, and platelets in turn trigger inflammation. However, in the present study, hemorrhage volumes were not large enough to drop the platelet count. No patients in our cohort were applied a liquid support before blood tests. Since the blood parameters of the patients were detected at the first admission to the emergency department, platelets did not reduce with hemodilution. The results we obtained in the scope of this study support the hypothesis that microthrombosis and blood clots affect mortality.

MPV, the mean platelet volume, is higher in newly produced platelets, and higher MPV indicates newly pro-

Table 1. Distribution of variables by deceased and alive patients.

Variables		Overall (n=148)	Status		p*
			Deceased (n=36)	Alive (n=112)	
		n(%)	n(%)	n(%)	
Gender	Female	45(30.4)	11(30.6)	34(30.4)	0.982
	Male	103(69.6)	25(69.4)	78(69.6)	
Surgery	No	105(70.9)	32(88.9)	73(65.2)	**0.006**
	Yes	43(29.1)	4(11.1)	39(34.8)	
Hypertension	No	64(43.2)	16(44.4)	47(42.3)	0.825
	Yes	84(56.8)	20(55.6)	64(57.7)	
Coronary artery disease	No	120(81.1)	28(77.8)	92(82.1)	0.561
	Yes	28(18.9)	8(22.2)	20(17.9)	
Heart failure	No	136(91.9)	29(80.6)	107(95.5)	**0.004**
	Yes	12(8.1)	7(19.4)	5(4.5)	
DM	No	124(83.8)	26(72.2)	98(87.5)	**0.031**
	Yes	24(16.2)	10(27.8)	14(12.5)	
Chronic liver disease	No	132(89.2)	29(80.6)	103(92)	0.055
	Yes	16(10.8)	7(19.4)	9(8)	
Alzheimer	No	129(87.2)	32(88.9)	97(86.6)	0.722
	Yes	19(12.8)	4(11.1)	15(13.4)	
Chronic renal failure	No	138(93.2)	32(88.9)	106(94.6)	0.231
	Yes	10(6.8)	4(11.1)	6(5.4)	
Cerebrovascular diseases	No	129(87.2)	28(77.8)	101(90.2)	0.053
	Yes	19(12.8)	8(22.2)	11(9.8)	
Arythmia	No	139(93.9)	36(100)	103(92)	0.079
	Yes	9(6.1)	0(0)	9(8)	
Thyroid diseases	No	135(91.2)	34(94.4)	101(90.2)	0.432
	Yes	13(8.8)	2(5.6)	11(9.8)	
ASA	2	29(19.6)	0(0)	29(25.9)	**<0.001**
	3	111(75)	30(83.3)	81(72.3)	
	4	8(5.4)	6(16.7)	2(1.8)	
CCI	0	70(47.3)	11(30.6)	59(52.7)	**<0.001**
	1	31(20.9)	3(8.3)	28(25)	
	2	46(31.1)	21(58.3)	25(22.3)	
	3	1(0.7)	1(2.8)	0(0)	

*Pearson chi-square test was used. DM: Diabetes mellitus, ASA: American Society of Anesthesiology, CCI: Charlson comorbidity index.

duced platelets to increase [6]. Increased MPV values are observed in myocardial infarction, ischemic and cerebral stroke, cardiovascular diseases, respiratory diseases, chronic renal failure, intestine diseases, rheumatoid diseases, diabetes, and various cancers [6]. On the other hand, decreased MPV has been reported in tuberculosis during disease exacerbation, ulcerative colitis, systemic lupus erythematous in adults, and different neoplastic diseases. Moreover, MPV has been found to reflect sympathetic overactivity [20], which explains that MPR can predict in-hospital mortality rates. In a study, which 91 patients who presented with acute stroke and underwent endovascular mechanical thrombectomy were included, it was evaluated that high MPV value was linked with a more severe clinical condition and mortality [21].

MPR has been commenced recently to be used as an important parameter of inflammation. The most significant advantages of MPR is its low cost and easy implementation. An increase in the significance of MPV also means that of MPR to increase, which suggests that like MPV, it is an appropriate ratio in predicting mortality. It has been stated that MPR can be a marker of mortality in various diseases such as myocardial infarction [22]. In a study, conducted on patients with bacterial sepsis reported that MPR was a marker of clinical severity and mortality [23]. Similarly, our results indicated that MPR was statistically significantly higher in the deceased patient group. MPR is important in relation to microthrombosis formation. In a study investigating the use of MPR for the prediction of mortality in hip fracture patients, it was reported that MPR could be used as a predictive predictor of progno-

Table 2. Distribution of blood parameters by status.

Variables	Overall	Status		p*
		Deceased	Alive	
	Mean ± SD	Mean ± SD	Mean ± SD	
BMI (kg/m^2)	25.71±2.49	26.01 ± 2.66	25.62 ± 2.44	0.409
PLT (10^3/L)	239.47 ± 82.74	214.88 ± 99.59	247.37 ± 75.36	**0.040**
MPV (fL)	9.26 ± 1.83	10.54 ± 1.91	8.85 ± 1.61	**<0.001**
MPR	0.05 ± 0.04	0.07 ± 0.06	0.04 ± 0.03	**<0.001**
CRP (mg/L)	19.33 ± 36.49	34.49 ± 57.67	14.45 ± 24.83	**0.004**
Albumin (gr/dL)	3.49 ± 0.51	3.08 ± 0.52	3.62 ± 0.44	**<0.001**
Lymphocyte (10^3/µL)	1.99 ± 1.62	1.68 ± 2.01	2.09 ± 1.46	0.189
CLR	22.52 ± 50.52	60.99 ± 87.52	10.16 ± 18.17	**<0.001**
CAR	5.85 ± 11.63	11.55 ± 19.5	4.01 ± 6.73	**0.001**
Neutrophil (10^3/µL)	8.55 ± 4.9	8.4 ± 4.24	8.6 ± 5.11	0.832
NLR	7.67 ± 12.55	14.1 ± 22.98	5.6 ± 4.94	**<0.001**
PLR	184.82 ± 164.08	293.48 ± 276.86	149.89 ± 80.15	**<0.001**
The thickness of hemorrhage (mm)	13.74 ± 7.84	13.81 ± 7.19	13.72 ± 8.07	0.954
Age (years)	62.18 ± 21.01	60.56 ± 22.28	62.71 ± 20.67	0.595
Hospital stay (day)	20.87 ± 25.1	23.11 ± 24.69	20.15 ± 25.3	0.540
ICU stay (day)	13.68 ± 21.19	21.14 ± 22.02	11.28 ± 20.45	**0.015**
ALT (U/L)	22.74 ± 19.3	27.31 ± 20.85	21.27 ± 18.63	0.102
aPTT (second)	38.39 ± 47.6	46.7 ± 76.47	35.72 ± 33.57	0.230
AST (U/L)	35.19 ± 29.59	43.82 ± 31.18	32.42 ± 28.65	**0.044**
Glucose (mg/dL)	157.89 ± 70.73	170.36 ± 51.56	153.88 ± 75.63	0.225
Ca (mg/dL)	8.9 ± 0.74	8.91 ± 0.65	8.89 ± 0.77	0.895
Cl (mmol/L)	104.39 ± 4.29	105.31 ± 3.64	104.1 ± 4.45	0.140
Creatinine (mg/dL)	0.98 ± 0.39	1.13 ± 0.59	0.93 ± 0.28	**0.007**
K (mmol/L)	4.31 ± 0.52	4.29 ± 0.65	4.32 ± 0.47	0.779
INR	1.51 ± 1	1.65 ± 1.08	1.46 ± 0.97	0.340
Na (mmol/L)	139.43 ± 3.8	139.94 ± 3.67	139.26 ± 3.84	0.348
HGB (gr/dL)	12.53 ± 2.05	11.82 ± 2.11	12.76 ± 1.99	**0.017**
HCT (%)	37.43 ± 5.43	36.25 ± 5.02	37.81 ± 5.52	0.134
RDW (fL)	14.26 ± 2	14.57 ± 1.63	14.16 ± 2.1	0.284
WBC (10^3/mL)	12.36 ± 5.02	13.7 ± 4.16	11.92 ± 5.21	0.065
BUN (mg/dL)	19 ± 8.53	21.49 ± 12.18	18.2 ± 6.85	**0.044**
Artery Blood pH	7.37 ± 0.06	7.32 ± 0.08	7.39 ± 0.04	**<0.001**
Artery Blood K (mmol/L)	3.79 ± 0.47	3.9 ± 0.56	3.75 ± 0.43	0.093
Artery Blood Na (mmol/L)	136.35 ± 2.79	136.75 ± 2.83	136.22 ± 2.78	0.326
Artery Blood Ca (mmol/L)	1.14 ± 0.05	1.14 ± 0.07	1.14 ± 0.05	0.705
Artery Blood Cl (mmol/L)	109.51 ± 4.14	111 ± 5.19	109.03 ± 3.64	**0.012**
Artery Blood Lactate (mmol/L)	2.09 ± 1.16	3.09 ± 1.4	1.77 ± 0.86	**<0.001**

*Independent samples t-test was used. BMI: Body mass index, kg: kilogram, PLT: Platelet, L: Liter, dL: deciliter, MPV: mean platelet volume, fL: femtolitre, MPR: MPV to PLT, CRP: C-reactive protein, mg: milligram, CLR: CRP to Lymphocyte, CAR: CRP to Albumin, µL: microliter, NLR: Neutrophil to Lymphocyte, PLR : Platelet to Lymphocyte, mm: millimeter, ICU: İntensive care unit, ALT: Alanine aminotransferase, U: unıt, aPTT: Activated partial thromboplastin time, AST: Aspartate aminotransferase, Ca: Calcium, Cl: Chloride, K: Potassium, mmol: millimole, INR: International Normalized Ratio, Na: Sodium, HGB: Hemoglobin, gr: gram, HCT: hematocrit, RDW: Red blood cell distribution width, WBC: White blood cell, BUN: Blood urea nitrogen.

sis. They found the cut-off value of MPR to be 0.048 with the sensitivity of 63% and specifity of 65% [24]. In our study, on the other hand, the cut-off value was found to be 0.06, and that the sensitivity value was similar whereas the specificity value was higher.

C-reactive protein that elevates during systemic infection and indicates inflammation is an acute phase reactant [25]. CRP can cause clot formation during inflammation by activating platelets [26]. Thus, the CRP value is expected to be high in patients with additional complications and high mortality rate. Our results confirm this information by revealing that the CRP values of the deceased patient group were higher.

Lymphocytes have a role in the main defense mechanism of the body [27]. The lymphocyte count tends to rise during inflammation and its decreased level, lymphopenia, will cause the defense mechanism of the body to weaken. Lymphopenia is the indicator of malnutrition [28]. Low lymphocyte count is reported to be a risk factor for Parkinson's disease [29]. In a study in which hematological pa-

Table 3. The univariate and multivariate Cox regression analysis of factors associated with in-hospital mortality.

Variables	Univariate				Multivariate			
	p	HR	95.0% CI for HR		p	HR	95.0% CI for HR	
			Lover	Upper			Lover	Upper
Heart failure	0.005	3.322	1.428	7.731	0.302	1.836	0.580	5.819
DM	0.003	3.149	1.478	6.711	0.123	2.069	0.821	5.216
MPR	≤0.001	1.104	1.055	1.156	**<0.001**	1.129	1.059	1.204
CLR	<0.001	1.002	0.999	1.005	**0.022**	1.011	1.002	1.020
CAR	0.001	1.022	1.003	1.041	**0.004**	1.004	1.001	1.007
NLR	<0.001	1.002	0.991	1.014	**0.182**	1.011	1.000	1.021
PLR	<0.001	1.001	1.000	1.002	0.164	1.006	0.998	1.015

DM: Diabetes mellitus, MPR: mean platelet volume /platelet count, CLR: C-reactive protein /lymphocyte count, CAR: C-reactive protein / albumin, NLR: neutrophil count/ lymphocyte count, PLR: platelet count/ lymphocyte count.

rameters of patients who underwent surgery for epidural and subdural hematoma were evaluated, reached a conclusion that low lymphocyte count is related to poor prognosis [30]. In the view of such information, a decrease in lymphocyte count indicate poor prognosis during any disease.

CLR, as one of the other parameters showing inflammation, has recently been encountered in many studies regarding the prediction of mortality. Although some studies conducted lately have revealed that CLR can be used as a mortality predictor, we have not come across any publication in this topic in TASDH cases in the literature. Therefore, we were unable to one-to-one compare our results with results of other studies. The CLR value will normally increase in conditions such as increased CRP or decreased lymphocyte in patients with malnutrition and inflammation, which exhibits that CLR can be an effective parameter on mortality. In a study conducted in patients with acute appendicitis with and without perforation, it was reported that CRP and CLR could predict perforation and could be used as prognostic markers [31]. In our study, CLR was also evaluated in the TASDH patients and the results obtained revealed that it can be utilized as a parameter predicting mortality. In the literature, in addition to studies reporting that CLR can be used to predict mortality, there are also studies stating that it alone does not predict mortality [14]. Although it has been reported that CLR does not predict mortality in elderly patients with hip fracture, we found that CLR is an effective predictor of in-hospital mortality in our study [14].

Albumin, a type of protein synthesized in the liver, serves tasks such as transporting nutrients in the blood to the organs and maintaining fluid balance in the body [32]. Albumin levels that reduce in various disease are also low in malnutrition [33]. In a study examining patients admitted to the hospital for any reason, it was reported that patients with low albumin levels stayed longer in the hospital and had a higher risk of death [34]. Since the blood parameters of the patients in our study were determined from the blood samples collected in their first admission to the emergency department, the albumin levels were the actual values without replacement.

There are studies in the literature regarding that the CAR value can be utilized as a marker of prognosis and mortality in various diseases. It has been suggested that CAR affects prognosis in patients with myocardial infarction [35]. Similarly, there is a study reporting that the CAR value is independently significant in estimating 90-day mortality in acute ischemic stroke patients [36]. In our study, it was also determined that the CAR value, in consistent with the literature data, was a parameter predicting mortality in both univariate and multivariate analyses.

The cut-off value of CAR was found to be 12.15 to predict 30-day mortality in elderly patients with hip fractures [14]. However, the cut-off value of CAR in our study was determined to be 8.79. In addition, we detected CAR to be an important predictor of in-hospital mortality. The results we obtained presented that higher ASA and CCI scores were significantly associated with increased mortality. TASDH was more prevalent in males in our study, as in the study in which acute subdural hematomas due to rupture of cortical arteries were examined [37].

TASDH continues to be observed with a high mortality rate, no matter how fast and effective medical and surgical intervention is performed. In the literature, the mortality rate of TASDH patients has been reported as up to 75% [38], whereas in-hospital mortality rate was determined to be 24% in our study. It has been reported that the presence of cardiovascular disease and DM increases the mortality rate in TASDH patients [39]. The results of the present study showed as well that heart failure and DM statistically significantly increased the mortality in the deceased patient group.

Limitations

There are certain limitations in our study. The retrospective nature of the study as well as the fact that it was carried out in a single center with the inclusion of limited numbers of patients are the most important limitations. On the other hand, the completeness of the collected data can be considered the strength of our study. As platelet counts were not manually studied, the variation in blood collection time and time to reach the laboratory is one other limitation. Nevertheless, we consider that the bloods did not wait for long before analysis, given that the cases in our study were emergency department patients, and that a pneumatic system was used to transport blood in our hospital.

Conclusion

Our study reveals the importance of hematological and biochemical parameters determined at the time of admission of TASDH cases to the emergency department in terms of predicting mortality. As the results of the present study show MPR to have a superior ability to predict mortality, we recommend avoiding waiting times in the emergency department that will impair platelet function. In addition, we also recommend that CRP and albumin values, which are not routinely determined at the time of the first admission to the emergency department, should be routinely studied due to their significance in mortality prediction. In conclusion, monitoring inflammatory mediators from blood of patients with traumatic acute subdural hematoma at the first admission to the emergency department will help reduce mortality.

Ethical approval

Ethical approval was obtained for this study from the Tokat Gaziosmanpasa University Clinical Research Ethics Committee (22-KAEK-113).

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