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ORIGINAL ARTICLE / ÖZGÜN ARAŞTIRMA MAKALESİ

Platelet Mass Index as a Predictor of Prognosis in Hemorrhagic Stroke

Hemorajik İnmede Platelet Kitle İndeksinin Prognoz ile İlişkisi

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ABSTRACT

Aim: Platelet mass index (PMI) is an inexpensive parameter which can be easily calculated from complete blood count. It has been recently claimed as a good inflammation parameter that is closely related to platelet function and is also believed to indicate the aggregation capacity of platelets. The present study aimed to investigate the prognostic value of PMI in patients with acute hemorrhagic stroke.

Material and Methods: All patients aged 18 years or over who were diagnosed with subarachnoid or intracerebral hemorrhage between 2014 and 2018 were included in the study. Platelet mass index was calculated with the formula "PMI = platelet count x MPV". The type and localization of hemorrhagic stroke, presence of secondary brain injury, laboratory parameters, need for surgical intervention, length of hospital stay, and 30-day mortality were compared between survivor and non-survivor patients. Receiver Operating Characteristic (ROC) analysis was performed in order to discriminate surviving patients on the 30th day.

Results: A total of 103 patients were included. PMI was significantly higher in the surviving patients (1912 [IQR 25–75%: 1544.2-2468.2]) compared to the non-surviving ones (1722.1 [IQR 25–75%: 1332.2-2114]) (p=0.039). There was no significant difference in the parameters showing platelet function and inflammation, including PMI levels in patients with and without secondary injuries or surgical intervention (p> 0.05 for all). AUC value was found to be 0.628 (95% CI: 0.517-0.738) for PMI in discriminating surviving patients on the 30th day.

Conclusion: This study revealed that the patients who died by 30th day after hemorrhagic stroke had lower PMI levels. However, the predictive accuracy of PMI for 30-day survival was poor. Therefore, we believe that PMI cannot be used alone in predicting prognosis of hemorrhagic stroke, but in combination with other markers of platelet function and inflammation, it may contribute to clinicians in patient risk assessment.

Keywords: Hemorrhagic stroke, subarachnoid hemorrhage, intracerebral hemorrhage, platelet mass index

ÖZ

Amaç: Platelet kitle indeksi (PKİ), tam kan sayımından kolayca hesaplanabilen ucuz bir parametredir. Son zamanlarda trombosit fonksiyonu ile yakından ilişkili olan iyi bir enflamasyon parametresi olduğu ve aynı zamanda trombositlerin agregasyon kapasitesini gösterdiği iddia edilmiştir. Bu çalışmada PKİ parametresinin akut hemorajik inmeli hastalarda prognostik değerinin araştırılması amaçlanmıştır.

Gereç ve Yöntemler: Çalışmamıza 2014-2018 yılları arasında subaraknoid ya da intraserebral hemoraji tanısı alan 18 yaş ve üzeri tüm hastalar dâhil edildi. PKİ, platelet sayısı ve ortalama platelet hacminin çarpımı formülü ile elde edildi. Yaşayan ve ölen hastalar arasında hemorajik inmenin tipi ve lokalizasyonu, sekonder beyin hasarı varlığı, laboratuar değerleri, cerrahi girişim gerekliliği, hastane kalış süreleri ve 30 günlük mortalite oranları karşılaştırıldı. 30. Günde yaşayan hastaların ayrımında PKİ'nin etkinliği için Receiver Operating Characteristic (ROC) analizi yapıldı.

Bulgular: Çalışmaya toplam 103 hasta dâhil edildi. Yaşayan hastalarda PMI (1912 [IQR 25–75%: 1544.20-2468.28]) ölen hastalara göre (1722.10 [IQR 25–75%: 1332.26-2114]) istatiksel olarak anlamlı düzeyde yüksekti (p=0.039). Sekonder beyin hasarı veya cerrahi girişim olan ve olmayan hastalar karşılaştırıldığında, PKİ dâhil trombosit fonksiyonu ve inflamasyonu gösteren parametreler açısından anlamlı bir fark bulunmadı (p <0.05). ROC analizinde 30. günde yaşayan hastaları ayırt etmede PMI için eğri altı alan (AUC) değeri 0.628 (% 95 CI: 0.517-0.738) saptandı.

Sonuç: Çalışmamızda hemorajik inme sonrası ilk 30 günde mortal seyreden hastalarda PKİ düzeylerinin daha düşük olduğunu belirledik. Ancak, PKİ'nin 30 günlük yaşam tahmininde performası zayıftı. Bu nedenle, PKİ'nin hemorajik inmede prognoz öngörüsünde tek başına kullanılamayacağını ancak diğer platelet fonksiyon ve inflamatuvar belirteçlerle birlikte klinisyenlerin hasta risk değerlendirmesine katkı sunabileceğini düşünmekteyiz.

Anahtar Kelimeler: Hemorajik inme, subaraknoid kanama, intraserebral hemoraji, platelet kitle indeksi

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Introduction

Hemorrhagic stroke, which can occur in two types as intracerebral hemorrhage (ICH) and subarachnoid hemorrhage (SAH), continues to be one of the important causes of morbidity as well as mortality in adults (1, 2). It has been shown by experimental models that, immediately after the onset of bleeding, blood components entering the intracerebral spaces activate a series of complex inflammatory responses such as microglia activation, increased cytokine secretion, and infiltration of damaged areas by neutrophils and macrophages (3, 4). Therefore, determining inflammatory indices that can predict the prognosis of patients with hemorrhagic stroke has become an interesting subject of research.

Platelets, whose main task is to provide hemostasis, play an important role in inflammatory reactions and systemic immune response (5). Thus, some platelet-related parameters such as mean platelet volume (MPV) have been reported to be the prognostic markers of inflammatory response in various diseases (6). Platelet mass index (PMI) has recently been introduced into the literature as a better inflammation parameter than MPV, in addition to being closely related to platelet function (7, 8). In a recent study, PMI was found to be associated with disease severity in upper gastrointestinal bleeding (9). Nevertheless, no study in the literature has yet investigated PMI in patients with hemorrhagic stroke.

In our study, we aimed to investigate the prognostic value of PMI, an inexpensive and widely used parameter which can be easily calculated from complete blood count parameters, in the patients with acute hemorrhagic stroke.

Material and Methods

Study design and setting

This retrospective study was conducted in a tertiary emergency department (ED) after being approved by the local ethics committee (No: 2012-KAEK-15/2142). All patients aged 18 years or over who were diagnosed with SAH or ICH between 01.01.2014 and 01.01.2018 were included in the study. Patients who developed SAH or ICH due to traumatic injury, who were using antiaggregant drugs, who had bleeding disorders or a history of hematological disease, who had active infection or a history of inflammatory disease, and who had a malignancy or pregnancy were excluded from the study. Demographic data, comorbidities, Glasgow Coma Scale (GCS) scores at admission, the type and location of hemorrhagic stroke, presence of secondary brain injury (cerebral edema, midline shift, intraventricular hemorrhage), the first laboratory values at admission to ED, need for surgical intervention, length of hospital stay, and 30-day all-cause mortality were reviewed using the hospital data system in hospitalized patients. Discharged patients were followed up by reviewing the patients' online medical records and calling their phone numbers registered in the hospital information system. Patients with missing data were also excluded.

Laboratory parameters

Complete blood count analysis was performed with Mindray BC-6800 device, and the biochemical parameters were studied using Beckman Coulter AU5800 (USA) device. PMI was calculated with the formula "PMI = platelet count x MPV".

Statistical Analysis

Study data were analyzed using IBM SPSS 24.0 (Chicago, IL, USA) statistical software. Whether the distribution of discrete and continuous numerical variables was compatible with the normal distribution was analyzed with Kolmogorov-Smirnov test. As they did not meet the normality criteria, they were expressed as median values and interquartile range (IQR, 25-75%); categoric variables were expressed as number and percentage (%). Categoric variables were compared using Chi-square test and continuous variables using Mann Whitney-U test. In order to determine the PMI threshold values between the surviving and non-surviving patient groups, a Receiver Operating Characteristic (ROC) curve was drawn and area under the curve (AUC) was calculated. $p < 0.05$ was considered statistically significant for all study results.

Results

During the study period, 103 patients who were diagnosed with hemorrhagic stroke were included (Figure 1, Flowchart of patients). Males constituted 59.22% of the study population; the patients had a median age of 64 (IQR 25–75%: 53-84) years. Fifty-eight (56.31%) patients had ICH, and 45 patients had SAH. The median GCS was 15 (IQR 25–75%: 7-15). Surgical interventions were performed for 12.62% of the patients. Considering the in-hospital prognosis of patients with secondary injury, it was seen that 26 patients (40.6%) died. While 6 of 38 patients discharged from the hospital died within the first 30 days, the total 30-day all-cause mortality rate in the entire study population was 31.06%. Additionally, out of a total of 71 surviving patients, 28 were SAH and 43 were ICH, according to hemorrhagic stroke type. Table 1 shows the patients' demographic and clinical data and the laboratory parameters.

When the characteristics of surviving and non-surviving patients were compared, statistically significant differences were found for GCS, PMI and the presence of cerebral edema, midline shift and intraventricular hemorrhage.

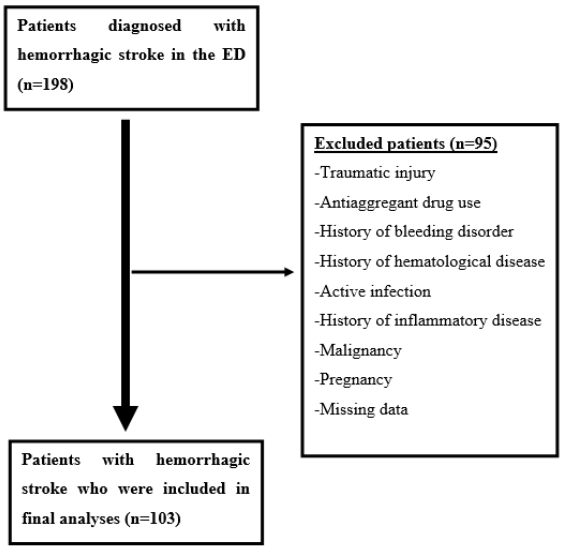

Figure 1. Flowchart of patients

Data/ Parameter	n (%) or *median (IQR 25-75)*
Male gender, *n (%)*	61 (59.22)
Age, *median (IQR 25-75)*	64 (53-84)
Comorbidities, *n (%)*	
Hypertension	55 (53.39)
Diabetes mellitus	21 (20.38)
Chronic renal failure	8 (7.76)
Coronary artery disease	18 (17.47)
Chronic heart failure	9 (8.73)
Cerebrovascular disease	8 (7.76)
Stroke type, *n (%)*	
Intracranial Hemorrhage (ICH)	58 (56.31)
Subarachnoid Hemorrhage (SAH)	45 (43.68)
Location, *n (%)*	
Bilateral	11 (10.67)
Basal ganglia	35 (33.90)
Frontal lobe	30 (29.12)
Parietal lobe	44 (42.71)
Temporal lobe	32 (31.06)
Occipital lobe	10 (9.70)
Cerebellum	6 (5.82)
Secondary Injury, *n (%)*	
Cerebral edema	48 (46.60)
Midline shift	11 (10.67)
Intraventricular hemorrhage	31 (30.09)
GCS, *median (IQR 25-75)*	15 (7-15)
Laboratory parameters, *median (IQR 25-75)*	
White blood cell	11.20 (8-13.70)
Hemoglobin	13.80 (12.30-15.22)
Hematocrit	40.90 (36.30-45.30)
Platelet count	214 (189-250)
MPV	8.58 (7.42-9.66)
RDW	15.20 (14.10-16.30)
PMI	1814.40 (1517-2282.40)
Glucose	138 (112-186)
Urea	36.60 (25.69-51.38)
Creatinine	0.86 (0.75-1.04)
Surgical intervention, *n (%)*	13 (12.62)
Length of hospital stay, *days median (IQR 25-75)*	6 (2-16)
In-hospital mortality, *n (%)*	26 (40.62)
Discharge after hospitalization, *n (%)*	38 (59.37)
Overall 30-day mortality, *n (%)*	32 (31.06)

Table1. Demographic data and clinical data of patients and laboratory parameters

GCS; Glasgow coma scale, MPV; Mean platelet volume, PMI; Platelet mass index,

While no significant difference was observed in other laboratory parameters showing platelet count and inflammation, PMI was significantly higher in the surviving patients (1912 [IQR 25–75%: 1544.20-2468.28]) compared to the non-surviving ones (1722.10 [IQR 25–75%: 1332.26-2114]) (p=0.039) (Table 2). Subgroup analyzes for secondary injury, surgical intervention and laboratory parameters in both hemorrhagic stroke types were also given in Table 3.

There was no significant difference in PMI, platelet count, MPV and RDW levels in patients with and without secondary injuries (p> 0.05 for all). Similarly, when patients with or without surgical intervention were compared, no significant difference was found in terms of parameters showing platelet count and inflammation, including PMI (p>0.05 for all) (Table 4). Subgroup analyzes for PMI in both patients' groups with and without secondary injury were also given in Table 5.

A ROC analysis was performed, and the AUC was calculated to find out the cut-off level for PMI level between surviving and non-surviving patients on the 30th day. AUC value was found to be 0.628 (95% CI: 0.517-0.738) for PMI in discriminating surviving patients (Figure 2). When the PMI threshold value was taken as 1827.50, PMI levels above this value could predict surviving patients with sensitivity of 54.92% (95% CI: 42.64-66.7), specificity of 71.80% (95% CI: 49.92%-83.80%), positive likelihood ratio of 1.76 (95% CI: 1.01-3.07), negative likelihood ratio of 0.66 (95% CI: 0.47-0.93), positive predictive value of 81.20% (95% CI: 69.10%-87.12%), and negative predictive value of 41.80% (95% CI: 32.72%-49.30%).

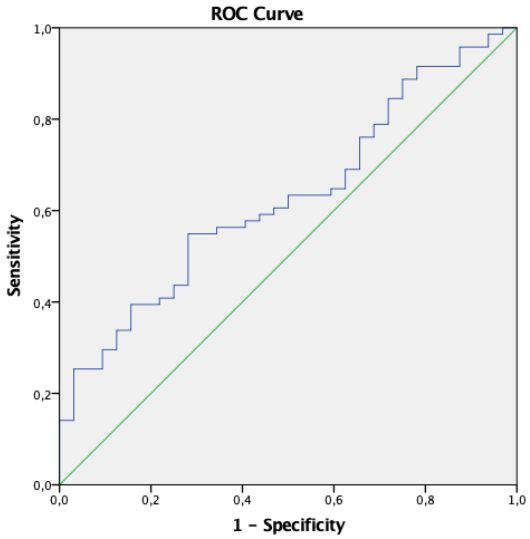

Figure 2. ROC curve for PMI in discriminating surviving patients on the 30th day

Discussion

In our study, in which we investigated the relationship between PMI and 30-day prognosis in patients with hemorrhagic stroke, we obtained three important findings. First, unlike other platelet function markers and inflammation indicators (platelet count, MPV, RDW), PMI levels showed statistically significant difference between the surviving and non-surviving patients. This result supports the idea that PMI, a novel marker, may be a more effective indicator for both platelet functions in bleeding pathophysiology and inflammatory response. Second, we found that platelet function and inflammation markers, including PMI, did not differ significantly in patients who developed acute central damage secondary to hemorrhage (cerebral edema, midline shift and intraventricular hemorrhage) and required urgent surgical intervention. Finally, we found that the predictive accuracy of PMI for 30-day survival was poor (AUC = 0.628). Although PMI can give an idea to the clinician in conjunction with other variables, we believe that it cannot be used alone to predict prognosis due to the complex pathophysiological process of hemorrhagic stroke and patient-dependent multifactorial variable course.

	Survivor (n=71)	Non-survivor (n=32)	p
Age, *median (IQR 25-75)*	64 (53-80)	70 (53-87.70)	0.280
Male gender, *n (%)*	43 (60.56)	18 (56.25)	0.680
Stroke Type, *n (%)*			
ICH	43 (60.56)	15 (46.87)	0.195
SAH	28 (39.43)	17 (53.12)	
GCS, *median (IQR 25-75)*	15 (13-15)	5 (3-10)	<0.001
Secondary Injury, *n (%)*			
Cerebral edema,	28 (39.43)	20 (62.50)	0.030
Midline shift	2 (2.81)	9 (28.12)	<0.001
Intraventricular hemorrhage	17 (23.94)	14 (43.75)	0.043
Laboratory parameters, *median (IQR 25-75)*			
Platelet count	220 (189-263)	205(189-247)	0.175
MPV	8.82 (7.44-9.86)	8.24 (7.40-8.86)	0.131
RDW	15 (14-16.20)	15.22 (14.46-17.28)	0.248
PMI	1912 (1544.20-2468.28)	1722.10 (1332.26-2114)	0.039
Surgical intervention, *n (%)*	7 (9.85)	6 (18.75)	0.217

Table 2. Comparison of surviving and non-surviving patients

	Survivor (n=71)	Non-survivor (n=32)	p
Secondary Injury, *n (%)*			
ICH patients:			
Cerebral edema	17 (39.5)	8 (53.3)	0.353
Midline shift	2 (4.7)	4 (26.7)	0.034
Intraventricular hemorrhage	11 (25.6)	9 (60)	0.016
SAH patients:			
Cerebral edema	11 (39.3)	12 (70.6)	0.042
Midline shift	0 (0)	5 (29.4)	0.005
Intraventricular hemorrhage	6 (21.4)	5 (29.4)	0.722
Laboratory parameters, *median (IQR 25-75)*			
ICH patients:			
Platelet count	214 (186-266)	207(185-250)	0.450
MPV	8.90 (7.30-10.10)	8.58 (8.10-8.90)	0.582
RDW	15.2 (14.1-16.2)	15.0 (13.9-17.1)	0.817
PMI	1912 (1536-2377)	1783 (1539-2114)	0.389
SAH patients:			
Platelet count	224 (190-261)	201 (189-241)	0.223
MPV	8.87 (7.49-9.80)	7.80 (7.02-9.05)	0.117
RDW	14.9 (13.3-15.6)	15.3 (14.6-17.3)	0.149
PMI	1932 (1610-2552)	1687 (1289-1997)	0.039
Surgical Intervention, *n (%)*			
ICH patients	N/A	N/A	N/A
SAH patients	7 (9.85)	6 (18.75)	0.217

Table 3. Subgroup analyzes for secondary injury, surgical intervention and laboratory parameters in both hemorrhagic stroke types

	Patients with Secondary Injury (n=64)	Patients without Secondary Injury (n=39)	p	Need for Surgical intervention (n=13)	No need for surgical intervention (n=90)	p
PMI	1807.20 (1522.50-2334.72)	1827.56 (1502.12-2227)	0.962	1820 (1510.30-2207.32)	1791.70 (1516.90-2299.32)	0.990
Platelet count	214 (189-254)	217 (188-250)	0.691	215(192-253)	214 (187-251)	0.781
MPV	8.40 (7.43-9.60)	8.77 (7.40-9.63)	0.563	8.20 (6.60-9)	8.63 (7.57-9.73)	0.125
RDW	15.05(13.95-16.52)	15.20 (14.22-16.20)	0.943	15 (14.30-15.66)	15.22 (13.90-16.34)	0.509

Table 4. Comparison of laboratory parameters in patients with and without secondary injury or surgical intervention

	Survivor (n=26)	Non-survivor (n=38)	p
Patients with Secondary Injury			
PMI, *median (IQR 25-75)*	1943.97 (1605.60-2637.60)	1706.52 (1302.26-1876.05)	0.014
	Survivor (n=33)	Non-survivor (n=6)	p
Patients without Secondary Injury PMI, *median (IQR 25-75)*	1827.56 (1487.06-2299.56)	1989.81 (1581.98-2255.95)	0.876

Table 5. Comparison of PMI in survivor and non-survivors in patients' groups with and without secondary injury

In recent years, the effects of inflammation and coagulopathy on the outcome of many pathophysiological conditions have been discovered, and accordingly, the prognostic value of blood parameters associated with both processes in various inflammatory diseases including ICH has begun to be investigated (10). Srinivasan et al. found that the leukocyte count is associated with clinical outcome in cerebrovascular diseases/SAH; Tao et al. reported similar findings for platelet/lymphocyte ratio (PLR), Chen et al. for MPV, and Siegler et al. for RDW (11-14). It has been suggested that PMI, which has recently started to find a place in the literature, may be a more effective prognostic marker than other platelet-related inflammatory parameters. While Krecak et al. reported that PMI is associated with thrombotic risk in polycythemia vera, Gao et al. associated PMI values with liveoid vasculopathy, Özbalcı et al. with reduced progression in chronic lymphocytic leukemia, Wang et al. with liver fibrosis in chronic hepatitis B and Akpınar et al. with slow coronary phenomenon (15-19). Although PMI continues to be investigated in the literature for many different conditions belonging to many different systems, studies on hemorrhage are quite limited. In a study conducted by Okur et al. in preterm infants, it was reported that low PMI is an important hemostatic parameter associated with a series of morbidities, including intraventricular hemorrhage (7). Öztürk et al. reported that, unlike MPV, pre-delivery PMI values are not associated with postpartum hemorrhage (20). Korkmaz et al. found that low PMI measured in very-low-birth-weight newborns at risk of intraventricular hemorrhage on postpartum of 5 to 7 days can provide clinicians with knowledge regarding IVH. The study also reported that the mortality rate, which was 18.6% in mild stage IVH patients, was 33.9% in severe stage IVH patients with significantly lower PMI values (21). In our study, platelet count, MPV and RDW did not show any significant relationship with 30-day mortality in ICH patients, while PMI was statistically significantly lower in non-surviving patients. Low PMI may indicate an inability to limit bleeding secondary to impaired platelet function. Additionally, low PMI can also be considered one of the indicators of inadequate neuroinflammation. In the literature, it has been reported that post-bleeding neuroinflammation is more than a non-specific stress-related reaction and plays a role in triggering the post-ICH coagulative response (13). Although excessive inflammation carries the risk of vasospasm-related cerebral damage, its deficiency, as indicated by low PMI values, may be another possible cause of the negative prognostic effect through coagulation failure, which was found in our study.

Recent studies have reported that some peripheral blood inflammatory biomarkers such as leukocytes and MPV may be associated with bleeding severity and complications in ICH (22-24). However, it has been underlined in different studies that proinflammatory markers may be independent of certain parameters determining the short-term prognosis, such as the infarct area (25-26). For example, while MPV to platelet ratio was found valuable in the prognosis of ICH in some studies, it was found unrelated to prognosis in a recent study by Chen et al. (13, 24, 27). The heterogeneity of the study populations, the diversity of parameters investigated, and the differences in prespecified clinical outcomes seem to be the most likely causes of inconsistent results reported in the literature. We did not find any statistically significant relationship between the development of acute central damage (cerebral edema, midline shift and intraventricular hemorrhage) secondary to hemorrhage or the need for urgent surgical intervention and PMI, contrary to the 30-day prognosis. In bleeding models, it has been shown that many pathways involved in the inflammatory and coagulative processes in ICH develop in association with vasospasm (28). It is known that most of these bleeding-related triggering effects in both macrovascular and microvascular areas occur especially in the hyperacute phase (22). Accordingly, our results suggest that PMI is not related early acute inflammatory response and early complications.

In the literature, different discriminative values ranging from poor to good (0.648 to 0.803) have been reported for AUC values related to inflammatory parameters in patients with ICH (12, 13). In our study, we determined that the AUC value (0.628) for PMI had a poor performance for the prediction of 30-day survival. To the best of our knowledge, our study is the first to investigate the prognostic value of PMI in adult patients with hemorrhagic stroke. PMI is one of the parameters easily obtainable from inexpensive, fast and widely available routine blood tests. However, due to this result in our study, we believe that PMI cannot be used alone in predicting prognosis in hemorrhagic stroke due to its complex pathways and multifactorial variable course that are yet to be clarified.

Limitations

First of all, our study was a single-center study and enrolled a relatively small number of patients; thus, our results cannot be generalized to all centers. Due to our study's retrospective nature, incorrect and missing data may have affected our study results. In addition, we took the first PMI values taken after ED admission as basis, but we did not investigate the time interval these PMI values correspond to after bleeding. Moreover, we may not have accessed all the

records of drugs or herbal supplements that could potentially affect some inflammatory parameters investigated in our study. Additionally, since we did not have the complete data on the day of death for all mortal patients with secondary injuries, we could not be able to interpret the in-hospital mortality time. Subgroup analyzes were also not suitable for interpretation because they included a relatively small number of patients. Finally, we did not evaluate long-term follow-up of our patients after 30th day, and the course of PMI levels.

Conclusion

Our study results revealed that the patients who died by 30th day after hemorrhagic stroke had lower PMI levels and when considered according to the stroke types, this difference was due to the patients with SAH. However, the predictive accuracy of PMI for 30-day survival was poor. Therefore, we believe that PMI cannot be used alone in predicting prognosis of hemorrhagic stroke, but in combination with other markers of platelet function and inflammation, it may contribute to clinicians in patient risk assessment.

Conflict of Interest: The authors declare that there is no conflict of interest.

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Authors' Contribution: Conception – SD, EE; Design and Supervision – SD, EE, OK, YÇ; Data Collection and Processing – SD, HU, ŞKÇ; Analysis and Interpretation – SD, OK, HU; Literature Review – SD, HU, ŞKÇ; Writing – SD, EE, HU; Critical Review – EE, ŞKÇ, YÇ.

Ethical Approval: Ethical approval for this study was obtained from Kecioren Training and Research Hospital Ethics Committee (No: 2012-KAEK-15/2142).

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