

Evaluation of mean platelet volume and platelet distribution width in patients with asymptomatic intermediate carotid artery plaque

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Abstract

Background: Platelets play a significant role in the pathogenesis of atherosclerosis. In atherosclerotic plaques, the risk of plaque rupture is more crucial than the severity of the stenosis they cause. Non-calcified carotid artery plaques are more unstable than calcified plaques, and they are associated with a higher risk of rupture, thromboembolism, and consequently, stroke.

Aim: The purpose of the present study is to compare calcified and non-calcified plaques that cause intermediate carotid artery stenosis, with respect to mean platelet volume (MPV) and platelet distribution width (PDW).

Methods: A total of 139 asymptomatic patients with 50–70% stenosis of the carotid artery were included in this study. Carotid Doppler ultrasound imaging and computed tomography angiography were performed to divide the carotid artery plaques into two groups as calcified and non-calcified. Patients included in the calcified (n = 73) and non-calcified (n = 66) plaque groups were compared with respect to MPV and PDW.

Results: Mean platelet volume was statistically significantly higher in the non-calcified plaque group compared to the calcified plaque group (MPV in non-calcified/calcified plaque groups [fL]: 10.0/9.0, respectively) ($p < 0.01$). PDW was not significantly different between the two groups ($p = 0.09$). Platelet count was statistically significantly higher in the calcified plaque group compared to the non-calcified plaque group (platelet count in calcified/non-calcified plaque groups [$10^3/\text{mm}^3$]: $250 \pm 63/226 \pm 56$, respectively) ($p = 0.019$). Multivariate regression analysis showed that MPV was independently associated with non-calcified carotid artery plaque (odds ratio 5.95, 95% confidence interval 2.63–13.45, $p < 0.001$).

Conclusions: Mean platelet volume is increased in the presence of non-calcified carotid artery plaques that cause asymptomatic intermediate stenosis. Increased MPV can be used as a marker to predict the risk of rupture of the non-calcified carotid artery plaques.

Key words: atherosclerosis, carotid artery stenosis, mean platelet volume, platelet distribution width, non-calcified plaque, stroke

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INTRODUCTION

Platelets play a significant role in the pathogenesis of atherosclerosis. In addition to acting as mediators of thrombus development, platelets also induce inflammation [1]. Increased platelet reactivity causes thrombogenic activation, which in turn increases the risk of cardiovascular diseases [2]. Increased platelet activity was demonstrated in patients with carotid ar-

tery stenosis [3]. As thrombocytes are activated, they increase in size and become more reactive [4].

Mean platelet volume (MPV) is a commonly used biomarker, and it indicates platelet functions such as platelet size, inflammation, and thrombosis [2]. Increased MPV is paralleled by a shorter bleeding time and a high level of plasma thromboxane B2 [5]. MPV values correlate with platelet function and

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activation. High MPV value is a reliable indicator of increased platelet activity. MPV is an independent risk factor for recurrent strokes in patients with a history of stroke or transient ischaemic attack (TIA) [6].

Platelet distribution width (PDW) represents the variation in platelet size. Large PDW can be an indicator of prothrombotic status [7]. There is only one study in the literature that has established a correlation between PDW and the severity of carotid artery lesion; in that study, PDW was found to be an independent predictor of the severity of carotid lesions [8]. So far, no study has investigated the relation between PDW and the risk of stroke or carotid artery plaque rupture.

The risk of rupture of atherosclerotic plaques is more crucial than the severity of the stenosis they cause [9]. Histopathological examinations of carotid endarterectomy samples demonstrated that calcified plaques are more stable and they are associated with a lower risk of rupture and thromboembolism [10]. Conversely, non-calcified and ulcerous plaques are more fragile and they carry a higher risk of rupture; therefore, they are associated with increased risk of embolism and stroke [11].

There is currently no test that can be used to predict the risk of stroke in asymptomatic patients with intermediate (50–70%) carotid artery stenosis. We believe that the non-calcified plaques that cause asymptomatic intermediate carotid artery stenosis are more active in terms of atherothrombosis, and they may be associated with a higher risk of stroke than calcified plaques. The present study compares calcified and non-calcified plaques that cause asymptomatic intermediate carotid artery stenosis with respect to MPV and PDW.

METHODS

Study population

A total of 164 patients were evaluated to be enrolled in this prospective study. Twenty-five patients were excluded from the study due to various reasons (nonsteroidal anti-inflammatory drugs or antibiotics usage [five patients], acute coronary syndrome [five patients], high creatinine values [five patients], active infection [five patients], malignancy [one patient], and heart failure [four patients]), and the analyses were performed on the data obtained from 139 patients. The patients were divided into two groups: those having calcified (73 patients) and non-calcified (66 patients) plaques. The study was approved by the ethics committee of our institution. Initially carotid Doppler ultrasound (CDUS) and then computed tomography angiography (CTA) were performed for all patients. Patients with 50–70% stenosis in the carotid artery were included in the study. The study cohort consisted of patients who had been assessed by a council (including a neurologist, a cardiologist, a radiologist, and a cardiovascular surgeon), and who had been confirmed to be asymptomatic with respect to the carotid artery lesion. Symptomatic patients were defined as those who experienced an ischaemic cerebrovascular event

with or without sequel, a TIA, or amaurosis fugax within the last six months, and they were excluded from the study. Patients with diabetes mellitus were included in the study after their blood glucose levels had been regulated. Patients were included in the study at least two months after initiation of their antiaggregant, antihyperlipidaemic, and antihypertensive therapies. Blood samples were obtained at two different time points, 15 days apart. The laboratory parameters that have been analysed in this study represent the mean value of two measurements taken at different time points and evaluated using the same device. Patients with a history of ischaemic or non-ischaemic stroke, systemic inflammatory disease, cancer, acute coronary syndrome, previous myocardial infarction, heart failure, significant valvular disease, chronic obstructive pulmonary disease, renal or liver failure, a haematological disease, an active infection, and patients who were using anti-inflammatory drugs or antibiotics were excluded from the study.

Blood samples

Fasting blood samples were drawn from a large antecubital vein of each patient for determination of biochemical and haemostatic parameters. EDTA-tubes were used for automatic blood count. The blood counts were measured by a Beckman Coulter LH 780 Haematology Analyzer. Total cholesterol, low-density lipoprotein, triglyceride, and high-density lipoprotein levels were measured by colorimetric method (Abbott Laboratories; Illinois USA).

Definitions

Hypertension was defined as a systolic/diastolic blood pressure of $\geq 140/90$ mm Hg or patients using antihypertensive medications. Diabetes mellitus was defined as a fasting plasma glucose level of ≥ 126 mg/dL or patients actively using oral antidiabetics and/or insulin. Patients who smoke regularly were considered as smokers. Hyperlipidaemia was defined as a total cholesterol level of ≥ 200 mg/dL. Coronary artery disease was defined angiographically as the presence of a plaque resulting in 50% or more stenosis in a major coronary artery. Body mass index (BMI) was calculated by dividing the body weight (kg) by the square of height (m).

Doppler ultrasonography and computed tomography angiography assessments

Carotid artery stenosis was first assessed by CDUS and then by CTA. CDUS examination was performed using an Esaote s.p.a MyLabClass C (Florence-Italy) device and a linear arrayed probe that allowed selection of frequencies between 3–11 MHz. CTA was performed using a CT device with Philips Brilliance 64 detector (Holland). After venous access was established through the antecubital vein and 80 mL non-ionic contrast agent was administered at a rate of 4.5 mL/s, axial-plane CT images of the carotid and cerebral arteries were

Table 1. Comparison of the demographical, clinical, and biochemical characteristics between the groups

	Calcified plaque group (n = 73)	Non-calcified plaque group (n = 66)	P
Age [years] median (25 th –75 th percentile)	70 (63–75)	71 (63–77)	0.420
Men	46 (63%)	50 (76%)	0.105
Diabetes mellitus	29 (40%)	28 (42%)	0.747
Hypertension	51 (70%)	53 (80%)	0.157
Coronary artery disease	47 (64%)	49 (74%)	0.209
Hyperlipidaemia	51 (70%)	42 (64%)	0.436
Smoking status	22 (30%)	18 (27%)	0.710
Total cholesterol [mg/dL] (25 th –75 th percentile)	198 (166–235)	182 (156–216)	0.198
Triglycerides [mg/dL] (25 th –75 th percentile)	150 (97–180)	144 (106–202)	0.802
HDL-C [mg/dL] (25 th –75 th percentile)	44 (37–53)	43 (36–48)	0.216
LDL-C [mg/dL] (25 th –75 th percentile)	115 (87–155)	111 (88–137)	0.286
BMI [kg/m ²] (25 th –75 th percentile)	26 (24–28)	26 (24–27)	0.731
Statin	38 (52%)	38 (58%)	0.514
ASA	58 (79%)	56 (85%)	0.408
Clopidogrel	6 (8%)	4 (6%)	0.623
ASA + clopidogrel	5 (7%)	2 (3%)	0.304

ASA — acetylsalicylic acid; BMI — body mass index; HDL-C — high-density lipoprotein cholesterol; LDL-C — low-density lipoprotein cholesterol

obtained using the tracking method. Acquired slices were transferred to the work-station (Philips IntelliSpace Portal) and multi-plane images, maximum intensity projection and volume rendering three-dimensional images were developed by post-processing the original slices via appropriate software. These images were reviewed with respect to the vascular plaques and stenosis. The stenosis level was assessed in CDUS [12]. Characteristics of carotid internal artery plaques were evaluated by 64-slice CTA. Measurements were recorded from the points with maximum thickness of plaque and greatest luminal diameter of stenosis. Hyperdense plaques (plaques containing > 50% calcific components and having a CT density > 120 Hounsfield unit [HU]) were defined as calcified plaque, while hypodense plaques (plaques containing < 50% calcific components and having a CT density < 120 HU) were defined as noncalcified plaque [13]. Based on the CDUS and CTA examinations, plaques that were fully soft and calcified by less than 50% were categorised as non-calcified plaques, whereas those that were fully hard and calcified by more than 50% were categorised as calcified plaques.

Statistical analysis

Data were analysed using SPSS software version 15.0 for Windows (SPSS Inc., Chicago, IL, USA). Categorical variables were presented as frequency and percentage. The χ^2 test and Fisher's exact test were used to compare categorical variables. The Kolmogorov-Smirnov test was used to assess the distribution of continuous variables. Student's t-test was

used for variables with normal distribution, and the values were presented as mean \pm standard deviation. Continuous variables without normal distribution were analysed using the Mann-Whitney U test, and the obtained values were presented as median (50th) values and interquartile ranges (25th and 75th). Multivariate logistic regression analysis was used to evaluate the independent associates of the risk of non-calcified plaque. The odds ratios (OR) and 95% confidence intervals (CI) were calculated. A two-tailed p-value of < 0.05 was considered statistically significant.

RESULTS

Table 1 summarises the demographical and clinical characteristics in addition to laboratory results of 139 patients (73 with calcified plaque, 66 with non-calcified plaque) who were included in the study. No significant difference was noted between the two groups in terms of the baseline parameters.

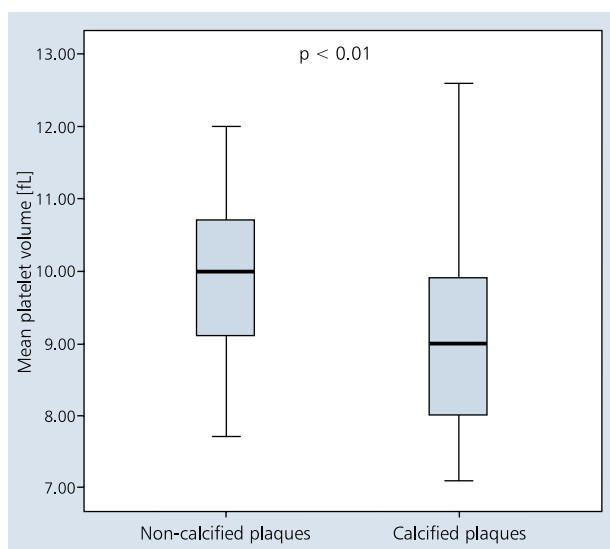
Table 2 shows the comparison of haematological data between the two groups. The platelet count was statistically significantly higher in the calcified plaque group. MPV was higher in the non-calcified plaque group, and the difference in MPV between the two groups was statistically significant (Fig. 1). There was no significant difference between the two groups with respect to PDW ($p = 0.09$).

Receiver-operating characteristic curve analysis showed that the MPV cut-off value was ≥ 9.9 fL for the prediction of calcified and non-calcified plaques that cause intermediate carotid artery stenosis (area under curve: 0.74, 95% CI

Table 2. Comparison of the haematological parameters between the groups

	Calcified plaque group (n = 73)	Non-calcified plaque group (n = 66)	P
Haemoglobin [g/dL]	13.5 ± 1.5	12.8 ± 1.4	0.013
Platelets [10 ³ /mm ³]	250 ± 63	226 ± 56	0.019
MPV [fL] median (25 th –75 th percentile)	9.0 (8.0–9.9)	10.0 (9.0–10.7)	< 0.01
PDW [%] median (25 th –75 th percentile)	11.4 (11.0–12.9)	12.0 (11.3–13.0)	0.09

fL — femtolitre; MPV — mean platelet volume; PDW — platelet distribution width

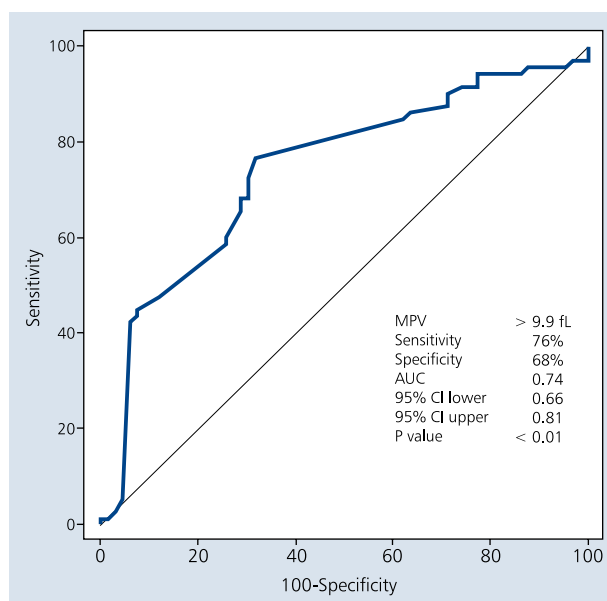
**Figure 1.** Box-plot demonstration of the distribution of mean platelet volume in the calcified and non-calcified groups

0.66–0.81, $p < 0.01$). MPV values ≥ 9.9 fL predicted calcified and non-calcified plaques with 76% sensitivity and 68% specificity (Fig. 2).

The multivariate logistic regression analysis including age, hypertension, diabetes mellitus, coronary artery disease, hyperlipidaemia, statin use, acetylsalicylic acid use, BMI, MPV, PDW, and platelet count showed that the MPV was independently associated with non-calcified carotid plaque (OR 5.95, 95% CI 2.63–13.45, $p < 0.001$) (Table 3).

DISCUSSION

This is the first study comparing calcified and non-calcified plaques that cause asymptomatic intermediate carotid artery stenosis with respect to MPV and PDW. In the present study, carotid artery plaques were assessed by CDUS and CTA, and the calcified plaque group was compared with the non-calcified plaque group in terms of MPV and PDW. The findings of this study indicate that the non-calcified plaques that cause intermediate carotid artery stenosis are associated with a significantly higher MPV value compared to the calcified plaques, whereas there is no difference between the two groups with respect to PDW values.

**Figure 2.** The receiver-operating characteristic curve of mean platelet volume for predicting non-calcified carotid plaque; AUC — area under curve; CI — confidence interval; fL — femtolitre; MPV — mean platelet volume**Table 3.** Independent predictors of non-calcified carotid plaque in multivariate logistic regression analysis

	Odds ratio (95% confidence interval)	P
Age	1.63 (0.94–1.04)	0.76
Hypertension	0.79 (0.30–2.08)	0.64
Diabetes mellitus	1.16 (0.49–2.73)	0.71
Coronary artery disease	0.81 (0.32–2.08)	0.67
Hyperlipidaemia	1.28 (0.52–3.12)	0.58
Statin	0.83 (0.36–1.92)	0.67
Acetylsalicylic acid	0.50 (0.15–1.62)	0.25
Body mass index	1.01 (0.83–1.24)	0.86
MPV ≥ 9.9 fL	5.95 (2.63–13.45)	< 0.001
PDW $\geq 11.4\%$	2.15 (0.92–5.02)	0.07
Platelets > 232 10 ³ /mm ³	1.63 (0.725–3.70)	0.23

fL — femtolitre; MPV — mean platelet volume; PDW — platelet distribution width

Atherosclerotic lesions are the leading cause of ischaemic stroke worldwide [14]. Previous studies have suggested that the leukocyte increase in subendothelial space may be a result of platelet activation [15]. Thrombocytes release various cytokines and growth factors that play significant roles in maintaining endothelial integrity. Platelets that are large and metabolically active have an important place in the pathophysiology of ischaemic stroke [16].

As thrombocytes are activated, they increase in size and become more reactive [8]. Compared to the thrombocytes of normal size, large thrombocytes are metabolically and enzymatically active, display faster binding to collagen, produce a higher amount of thromboxane A₂, and express more glycoprotein Ib, IIb/IIIa receptors [17]. MPV is widely used to assess the platelet size and functions.

Mayer et al. [18] performed a study on atherosclerotic plaque progression and clinical outcomes in patients with asymptomatic carotid stenosis and concluded that MPV is an independent risk factor for predicting adverse cardiovascular outcomes in patients with asymptomatic carotid stenosis. Interestingly, a statistically significant relation could not be established between MPV and the severity of carotid artery stenosis in that study [18]. Similarly, Adam et al. [8] also could not demonstrate a correlation between MPV and the severity of carotid artery stenosis. A common conclusion of the previous studies is that rather than indicating the severity of carotid lesions, MPV constitutes a risk factor for the development of complications associated with an already existing plaque. In the present study, mean MPV values of the non-calcified and calcified plaque groups were 10.0 fL and 9.0 fL, respectively. MPV cut-off value was ≥ 9.9 fL for the prediction of calcified and non-calcified carotid artery plaques. These findings confirm that, independently of the severity of stenosis, the non-calcified carotid plaques with higher MPV values are associated with an increased risk of stroke.

There is only one study in the literature that investigated the relation between PDW and carotid intima-media thickness, and that study did not reveal a correlation between carotid intima-media thickness and PDW [19]. PDW is correlated with the severity of carotid artery lesions. In the present study, PDW was not significantly different between the patients with calcified and non-calcified asymptomatic intermediate carotid artery plaques.

In this study, the platelet count was lower in the non-calcified plaque group than in the calcified plaque group. Some studies have previously established an inverse relationship between MPV and platelet count; however, the reason for this inverse relationship is still unclear. One study demonstrated that the stimulations that result in an increase in platelet count and volume during thrombopoiesis are regulated by independent mechanisms [20, 21].

Plaque morphology is an independent risk factor for prognosis in coronary artery patients. Non-calcified plaques are

associated with worse clinical outcomes. After 78 months of follow-up, the rate of mortality among patients with calcified coronary plaques, mixed-plaques, and non-calcified plaques was shown to be 1.4%, 3.3%, and 9.6%, respectively. Coronary CTA can provide reliable information about plaque morphology [22].

An important factor that distinguishes the present study from previous studies is that in this study, carotid stenosis and plaques were assessed by both CDUS and CTA. CTA is a more convenient method than magnetic resonance angiography and Doppler ultrasonography for assessing the severity of carotid artery stenosis [23]. CTA has also been shown to identify plaque ulceration, calcification, and lipid cores with an overall agreement of about 75% between CTA findings and histology [24]. Occasionally, the degree of stenosis as demonstrated by CDUS and by CTA may be different. Assessing the carotid plaques by both of these methods reduces the error margin. CTA is a particularly reliable method to evaluate the nature of a plaque and the extent of plaque calcification in the carotid arteries; however, measurements by intravascular methods (intravascular ultrasound and optical coherence tomography) could have provided more sensitive results [25].

Our study group consisted of asymptomatic patients who had intermediate stenosis in the carotid artery. This patient group is currently being followed-up by medical means in light of available information. It is difficult to predict who among these patients will become symptomatic during follow-up. The nature of an atherosclerotic plaque is an important determinant of its symptomatic characteristics. Non-calcified carotid plaques are more unstable than the calcified plaques, and therefore they are associated with a higher risk of rupture, thromboembolism, and stroke [8]. In the present study, MPV was higher in the non-calcified plaque group compared to the calcified plaque group. Based on this finding, it may be argued that among the non-calcified plaques that cause asymptomatic intermediate carotid artery stenosis, those that are associated with increased MPV may reflect a higher risk of stroke, and such lesions should be closely monitored.

Various factors may affect platelet size and functions. In the present study, we aimed to obtain highly reliable results by obtaining two blood samples 15 days apart and by analysing the mean values of the parameters measured from the separate blood samples.

Limitations of the study

All of the comorbidities and environmental factors that might have affected platelet count and functions were not taken into account. Although MPV is believed to be a marker of platelet (re)activity, it is not specific. The aggregometry method may have yielded more sensitive and accurate results. Additionally, our findings may not reflect the outcomes in female patients because the majority of the study cohort were male. Other limitations of this study the fact that it was a single-centre study and included a relatively small sample size.

CONCLUSIONS

Mean platelet volume is increased in the presence of non-calcified carotid artery plaques that cause asymptomatic intermediate stenosis. MPV may be used as a predictor of stroke risk associated with the carotid artery plaques that cause asymptomatic intermediate stenosis.

Conflict of interest: none declared

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Ocena średniej objętości płytek krwi i szerokości rozkładu objętości płytek krwi u chorych z blaszką miażdżycową tętnicy szyjnej powodującą bezobjawowe zwężenie pośredniego stopnia

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Streszczenie

Wstęp: Płytki krwi odgrywają istotną rolę w patogenezie miażdżycy. Ważniejsze znaczenie ma ryzyko pęknięcia blaszki miażdżycowej niż powodowane przez nią zwężenie. Nieuwapnione blaszki miażdżycowe są bardziej niestabilne niż blaszki uwapnione i wiążą się z większym ryzykiem pęknięcia, powikłań zakrzepowo-zatorowych i, w konsekwencji, udaru mózgu.

Cel: Przedstawione badanie przeprowadzono w celu porównania uwapnionych i nieuwapnionych blaszek miażdżycowych powodujących zwężenie tętnic szyjnych pośredniego stopnia w odniesieniu do średniej objętości płytek krwi (MPV) i szerokości rozkładu objętości płytek krwi (PDW).

Metody: Do badania włączono 139 chorych z 50–70-procentowym zwężeniem tętnicy szyjnej bez objawów klinicznych. Wykonano ultrasonografię dopplerowską i angiografię metodą tomografii komputerowej w celu podzielenia blaszek na dwie grupy w zależności od uwapnienia. Grupy pacjentów z blaszkami uwapnionymi (n = 73) i nieuwapnionymi (n = 60) porównano pod względem MPV i PDW.

Wyniki: W grupie chorych z nieuwapnionymi blaszkami MPV była statystycznie istotnie większa niż w grupie z blaszkami uwapnionymi (MPV w grupie z blaszkami nieuwapnionymi i w grupie z blaszkami uwapnionymi wynosiła odpowiednio 10,0 fl i 9,0 fl; p < 0,01). Wartości PDW nie różniły się znamienne między grupami (p = 0,09). Liczba płytek krwi była statystycznie istotnie wyższa w grupie z uwapnionymi blaszkami niż w grupie z blaszkami nieuwapnionymi (liczba płytek krwi w grupie z blaszkami nieuwapnionymi i w grupie z blaszkami uwapnionymi wynosiła odpowiednio $250 \pm 63 \cdot 10^3/\text{mm}^3$ i $226 \pm 56 \cdot 10^3/\text{mm}^3$; p = 0,019). W wieloczynnikowej analizie regresji wykazano, że MPV była niezależnie związana z występowaniem blaszek nieuwapnionych w tętnicy szyjnej (iloraz szans 5,95; 95% przedział ufności 2,63–13,45; p < 0,001).

Wnioski: Stwierdzono zwiększone wartości MPV w przypadku obecności w tętnicy szyjnej nieuwapnionych blaszek miażdżycowych powodujących bezobjawowe zwężenie pośrednie tętnicy szyjnej. Zwiększona MPV może być stosowana jako wskaźnik ryzyka pęknięcia nieuwapnionych blaszek miażdżycowych.

Słowa kluczowe: miażdżycy, zwężenie tętnicy szyjnej, średnia objętość płytek krwi, szerokość rozkładu objętości płytek krwi, nieuwapniona blaszka miażdżycowa, udar mózgu

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