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ORIGINAL ARTICLE

Usefulness of the platelet-to-lymphocyte ratio in predicting the severity of carotid artery stenosis in patients undergoing carotid angiography

Ceyhun Varım [a,*], Perihan Varım [b], Bilgehan Atılgan Acar [c], Mehmet Bülent Vatan [b], Mehmet Sevki Uyanık [d], Tezcan Kaya [a], Turkan Acar [c], Ramazan Akdemir [b]

[a] *Department of Internal Medicine, Sakarya University Medicine Faculty, Sakarya, Turkey*
[b] *Department of Cardiology, Sakarya University Medicine Faculty, Sakarya, Turkey*
[c] *Department of Neurology, Sakarya University Medicine Faculty, Sakarya, Turkey*
[d] *Department of Hematology, Sakarya University Medicine Faculty, Sakarya, Turkey*

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Abstract Carotid artery stenosis (CAS) is primarily caused by atherosclerotic plaque. Progressive inflammation may contribute to the rupture of an atherosclerotic plaque. The platelet-to-lymphocyte ratio (PLR) is a new and simple marker that indicates inflammation. In this study, we aimed to investigate the use of the PLR to determine the severity of CAS. One hundred forty patients were chosen from among patients who underwent carotid angiography in our institution. Symptomatic patients with stenosis >50% in the carotid arteries and asymptomatic patients with stenosis >80% were diagnosed via carotid angiography as having critical stenosis. Patients were classified into two groups. Group 1 included patients who had critical CAS, whereas Group 2 included patients with noncritical CAS, as determined by carotid angiography. Correlations between the PLR and the severity of CAS were analyzed. There were no significant differences in sex and age between the two groups. The PLR was 162.5 ± 84.7 in the noncritical CAS group patients and 94.9 ± 60.3 in the critical CAS group patients ($p < 0.0001$). The PLR value of 117.1 had 89% sensitivity and 68% specificity for CAS [95% confidence interval, 0.043 −0.159; area under the curve, 0.101 ± 0.03)]. In this study, we have shown that PLR values may be associated with critical stenosis in at least one of the carotid arteries. Furthermore, PLR values may be used to predict critical stenosis in the carotid arteries.

Conflicts of interest: All authors declare no conflicts of interest.

* Corresponding author. Sakarya University Internal Medicine, Adnan Menderes Street, Number 195 Adapazari, 54000 Adapazari, Sakarya, Turkey.
E-mail address: ceyhunvarim@sakarya.edu.tr (C. Varım).

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Introduction

Carotid artery stenosis (CAS) is an important arterial occlusive disease that may lead to the formation of cranial ischemic infarction and stroke. Atherosclerosis has a role in 90% of the etiologies of all extracranial carotid artery diseases [1–4]. Atherosclerosis is a systemic chronic inflammatory disease of the arterial intima. It affects overall systemic arterial circulation [3]. Increased inflammatory status is related to a poor prognosis for atherosclerosis [4].

Increased platelet activation has an important role in the initiation and progression of atherosclerosis [4]. Inflammatory mediators, such as interleukin 1 and 6, stimulate megakaryocytic proliferation and cause thrombocytosis. Thus, the platelet count may indicate inflammation. Studies have shown a relationship between coronary artery diseases and high platelet count [5–7]. In addition, lymphopenia is an indicator of physiologic stress and poor general health [8]. In this study, we aimed to reveal the relationship between CAS and platelet-to-lymphocyte ratio (PLR) values.

Materials and methods

Between January 2014 and May 2015, 140 patients who underwent carotid angiography were included in this study. An independent consultant neurologist evaluated all patients before the carotid intervention. Patients were considered symptomatic if they had experienced a cerebral infarct, transient ischemic attack, or amaurosis fugax attributable to a lesion of the ipsilateral carotid artery within the preceding 6 months. Clinical data such as hypertension, previous stroke, diabetes mellitus, hyperlipidemia, current smoking status, coronary artery disease, peripheral vascular disease/abdominal aortic aneurysm, previous carotid stenting, previous carotid endarterectomy, and chronic kidney disease were recorded. Demographic data such as age and sex were also assessed. All patients underwent carotid Doppler ultrasound, magnetic resonance angiography, or computed tomography angiography of the carotid arteries before the angiography.

Premedication, which consisted of aspirin (100 mg/d) and clopidogrel (75 mg/d), was administered at least 2 days before the procedure. Experienced interventional cardiologists performed all carotid diagnostic and therapeutic interventional procedures. Vascular access was obtained via a 6-Fr sheath in the common femoral artery. Carotid and cerebral angiography was performed after an arch aortogram (40° left anterior oblique) using a pigtail catheter. A digital subtraction angiogram of the intracranial and extracranial carotid circulation was obtained for at least two projections.

The innominate and left subclavian arteries were engaged using a 6-F Judkins Right (JR) 4 diagnostic catheter. The JR catheter was advanced to the ascending aortic root, torqued counter-clockwise for turning its tip superiorly, and then withdrawn into the vessel ostium. An angled glide catheter was used in a similar manner. In elderly patients and patients with Type III arches, a Simmons catheter (Cook, Bloomington, IN, USA) was used. We advanced the Simmons catheter into the left subclavian artery first with a 0.038-inch guide wire. We then withdrew the guide wire and advanced the catheter, thereby achieving an angled catheter tip in most patients. In some patients, we strangled the tip of the Simmons catheters in the ascending aorta. We repeatedly pulled back the catheter until selective engaging of the innominate and left common carotid arteries.

Standard anteroposterior and lateral projections were used to delineate the carotid bifurcation. Additional projections were used in some patients. Cerebral angiography was performed with standard anteroposterior and lateral projections in all patients. Independent interventional cardiologists retrospectively evaluated all angiographic images.

The degree of stenosis was assessed according to the North American Symptomatic Carotid Endarterectomy Trial (NASCET) 2011 ASA/ACCF/AHA/AANN/AANS/ACR/ASNR/CNS /SAIP/SCA Guideline on the Management of Patients with Extracranial Carotid and Vertebral Artery Disease Criteria [5]. Internal carotid artery (ICA) stenosis of >50% in symptomatic patients and ICA stenosis >80% in asymptomatic patients were defined as critical for carotid angiography.

Patients were divided into two groups. Group 1 had critical ICA stenosis, whereas Group 2 had noncritical ICA stenosis as assessed by carotid angiography.

Data from all patients were collected from hospital medical records. Excluded from the study were patients with any hematologic disease, recent arterial thrombotic disease, cirrhosis, chronic pulmonary disease, chronic renal disease, any diagnosed cancer, chronic inflammatory or autoimmune diseases, active infection, or patients receiving antibiotic treatment. After 12–14 hours of fasting, venous blood samples were obtained from patients for biochemical and hemogram analysis. Complete blood cell counts and automated differential counts were determined via an automated hematology analyzer (Abbott CELL-DYN 3700 System, Ramsey, Minnesota, 55303, USA), which provided total white blood count, platelet, neutrophil, lymphocyte, monocyte, eosinophil, and basophils counts/mL. The baseline PLR was calculated by dividing the absolute platelet count by the absolute lymphocyte count. The institutional ethics committee approved the study protocol.

Statistical analysis

Data analysis was performed using SPSS for Windows 17.0 (Statistical Package for Social Science; SPSS Inc., Chicago, IL, USA). The mean differences of the continuous data were measured by the *t* test and the median differences of the categorical data were measured by the Mann–Whitney *U* and Fisher's exact tests. A p value < 0.05 was accepted as statistically significant. The Chi-square test was used to compare differences between categorical variables. Receiver operating characteristics curves (ROC) were conducted, when appropriate.

Results

One hundred forty patients were included in the data analysis. Patients were divided into two groups. Group 1 ($n = 64$) included patients with critical CAS, whereas Group