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Evaluation of Postoperative Prognosis on Carotid Endarterectomy Single Center Experience

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ABSTRACT

AIM: To determine the prognostic value of routine hematological indices in patients undergoing carotid endarterectomy.

MATERIAL and METHODS: As a retrospective single center study, we measured the systemic immune inflammation index (SII) and other systemic inflammatory parameters to estimate the mortality and morbidity of patients undergoing CEA. These parameters include inflammatory markers which are included in routince preoperative haematologic tests like CBC.

RESULTS: After the analysis of the collected datas from 72 patients, the results showed that inflam-matory indices were significantly different in patients with different clinical coruse.

CONCLUSION: Inflammatory parameters calculated from routine preoperative hematologic parameters proved to be important predictive parameters that can be used in mortality/morbidity estimation of patients scheduled for CEA.

KEYWORDS: Carotid artery, Stenosis, Cerebrovascular, Endarterectomy

INTRODUCTION

Despite the advances in treatment strategies, carotid artery atherosclerosis remains a major cause of stroke and a prominent contributor to overall mortality and morbidity (21,32,44). Carotid artery stenosis accounts for approximately 30% of all cases of stroke (4). The estimated prevalence of carotid artery stenosis in the age group of 30–70 years is 1.8% in males and 1.2% in females (41). Atherosclerotic stenosis of cervical segment of the internal carotid artery is present in approximately 1%–2% of the general adult population; however, it affects more than 10% of individuals between the ages of 60 and 79 years (29,40).

One option for treating the carotid artery stenosis is Carotid endarterectomy (CEA) while the other is being carotid artery stenting (CAS). These two vascular treatments recommended for mitigating the risk of major stroke and disability (1,5). CEA was first reported by Eascott and colleagues in 1954 (15). Over the last 60 years, the techniques for CEA have remarkably evolved (8,30,34). Essentially, CEA is performed to minimise the risk of stroke by surgically removing the atherosclerotic plaque from both the common carotid artery bifurcation and the internal carotid artery (45). The reported rates of restenosis after endarterectomy vary between 5% and 37%, depending on the definition of restenosis and the follow-up period (11,24). Local and systemic inflammatory responses, the presence of risk factors (such as obesity, and dyslipidemia and smoking), and the characteristics of atherosclerotic plaques are the main parameters influencing post-endarterectomy restenosis (6,18,20,35,36).

The key role of atherosclerotic-inflammatory processes in the pathogenesis of carotid stenosis and the ability to

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readily measure the inflammatory markers using routine hematological indices have introduced a new perspective on post-endarterectomy prognosis. Inflammatory markers such as blood cell counts and also ratios such as platelet-to-lymphocyte ratio (PLR), or the systemic immune inflammation index (SII) are associated with vascular events (12,22,46), including carotid atherosclerosis (9,36), and cancer prognosis (17,33,43,53).

This study demonstrates the value of the SII in predicting post-carotid endarterectomy stroke and mortality.

MATERIAL and METHODS

Study Design

We reviewed 83 patients retrospectively who underwent carotid endarterectomy between 2017 and 2022 in our clinic. All patients were aged > 18 years, symptomatic, and had at least 50% stenosis. 11 patients were excluded due to active tumors, infection, rheumatological-hematological diseases, or a previous history of CEA. The remaining 72 patients were included and analyzed.

Epidemiologic data as age, sex, and hospitalization period were extracted from the hospital's electronic database. Risk factors, comorbidities, the degree of stenosis, and the side of stenosis were documented.

Inflammatory Markers

Data regarding the following indices were extracted from the laboratory tests: complete blood count including cells of lymphocytes, neutrophil, and platelet. Systemic immune inflammation index (SII), blood cell counts and systemic inflammatory response index (SIRI) were calculated using the following formulae.

SII = (neutrophils * platelets)/lymphocytes

SIRI = (monocytes * platelets)/lymphocytes

CEA Protocol

Patients underwent CEA under general anesthesia using the classic method, encompassing the preparation of the common, external, and internal carotid arteries, followed by clamping of all three arteries. Prior to clamping, 5000 IU of intravenous heparin was administered. Subsequently, CEA was performed through a longitudinal arteriotomy, which included the removal of atherosclerotic plaques. The excised plaque was then sent for histological evaluation. Finally, arterial reconstruction was carried out using 6-0 Prolene sutures and intradermal stitches (Ethicon, Norderstedt, Germany).

Primary Endpoint and Follow-up

Patients were retrospectively reviewed at least one year back and medical histories were collected together with clinical outpatient examinations were conducted. Any occurrence of stroke and/or mortality during this timeframe was documented. Mortality and postoperative mobidity was the main endpoint of this study. We defined stroke as a neurological deficit that persisted for more than 24 hours. Major adverse cardiovascular and cerebrovascular events (MACCE) included mortality, stroke, and myocardial infarction.

Statistical Analysis

The study universe was divided into three main groups (T1, T2, and T3) based on tertiles of SII level at admission. Baseline characteristics were compared among these three groups. Statistical analyses were conducted for the observed mortality, stroke, and MACCE within each sub-group.

The SPSS 22.0 software program for Windows has been utilised for statistical analyses. Categorical variables were expressed as frequency and differences between groups were analysed by using the Chi-Square Test. Continuous variables were expressed as median and mean ± standard deviation. Inter-group differences were assessed with Kruskal-Wallis test; p values <0.05 were considered as statistically significant.

Ethical approval: This study is performed under the regulations of the institutional research committee (Date: 02/11/2023, Decision Number: i09-643-23).

RESULTS

The study included 72 patients (35 female, 37 male; median age: 71 years) who underwent CEA. Clinically characteristics of the patients categorized according to their SII levels are presented in Table I.

Patients in T2 were significantly younger than patients in the T1 group (p=0.015). There were not any significant inter-group differences with respect to gender. When groups were examined based on ipsilateral stenosis, the proportion of patients with the stenosis of 70%–89% in the T2 group was higher than T1, while the proportion of patients with with a stenosis of 50%–69% in the T1 group was remarkably higher than that in T2 (p=0.020). Hyperlipidemia was more frequently observed in the T3 group compared to the T1 (p=0.020). Datas were statistically insignificant between the groups in terms of contralateral stenosis, body mass index (BMI), diabetes mellitus (DM), hypertension (HT), smoking, previous myocardial infarction, percutenaus coronary intervention (PCI), and coronary artery bypass grafting (CABG).

As one progresses from T1 to T3 among the groups, leukocyte counts (p=0.001), neutrophil counts (p<0.001), platelet counts (p=0.001), monocyte counts (p=0.001), and SIRI (p<0.001) increased, while the lymphocyte counts (p<0.001) decreased.

In Table II, the incidence of in-hospital stroke showed an increasing trend from T1 to T3, but the pattern was statistically insignificant due to only three cases. Regarding long-term outcomes, the rates of stroke (p=0.005), MACCE (p=0.001), and death (p=0.002) increased from T1 to T3, and higher SII levels were associated with a higher incidence.

DISCUSSION

Atherosclerosis is a major cause of cerebrovascular disease associated mortality. Inflammatory process is a pivotal step in the pathogenesis of atherosclerosis and significantly contributes to the progression of these diseases (28). Atherosclerosis

Table I: Baseline Characteristics of Patients Stratified by Systemic Immune-Inflammation index Tertiles

	T1	T2	T3	p-value
	SII < 419	419 <SII <696	SII > 696	
	n = 24	n = 24	n = 24	
Age	72.54 ± 5.69	66.92 ± 7.07	71.25 ± 8.59	**0.015**
Sex, female, n (%)	10	13	12	0.566
Stenosis % ipsilateral				
%50–69	3	13	9	**0.020**
%70–89	16	7	13	
>%90	5	4	2	
ICA stenosis >70%, contralateral	0	1	2	0.768
Body mass index				
<18.5	0	0	0	
18.5–24.9	5	5	6	0.676
25–29.9	11	16	11	
>30	8	3	7	
Diabetes mellitus	13	13	16	0.383
Hypertension	18	17	19	0.741
Hyperlipidemia	2	7	9	**0.020**
Smoking	9	12	8	0.770
Previous myocardial infarction	8	9	10	0.554
Previous PCI	7	7	10	0.362
Previous CABG	2	2	7	0.092
Laboratory parameters				
White blood cell count/μL	7.65 ± 2.47	7.20 ± 1.20	9.38 ± 3.33	**0.001**
Monocyte count, cells/μL	0.68 ± 0.34	0.59 ± 0.10	2.91 ± 10.25	**0.001**
Neutrophil count, cells/μL	3.96 ± 0.71	4.32 ± 0.82	7.01 ± 3.08	**<0.001**
Lymphocyte count, cells/μL	2.82 ± 1.63	2.05 ± 0.46	1.73 ± 0.49	**<0.001**
Platelet count, cells/μL	222.08 ± 55.09	269.21 ± 55.87	283.17 ± 47.65	**0.001**
Systemic immune inflammation index (SII)	339.25 ± 91.39	564.00 ± 83.83	1390.44 ± 1410.97	
Systemic inflammatory response index (SIRI)	0.01 ± 0.005	0.01 ± 0.005	0.07 ± 0.227	**<0.001**

ICA: *Internal carotid artery,* ***PCI:*** *Percutaneous coronary intervention,* ***CABG:*** *Coronary artery bypass grafting.*

Table II: In-Hospital and Long-Term Outcomes According to SII Tertiles

	T1	T2	T3	p*
In-hospital outcome, n (%)				
Stroke	0	1	2	0.768
Long-term outcomes, n (%)				
Stroke	1	1	8	**0.005**
All-cause death	2	3	11	**0.002**
MACCE	3	4	19	**0.001**

SII: *Systemic immune-inflammation index;* ***MACCE:*** *major adverse cardiac and cerebrovascular events. *Fisher's exact test.*

is characterized by the accumulation of lipids, proliferation of smooth muscle cells plus local inflammation. Also, immune system play a pivotal role in this cascade (10).

Several studies have highlighted the involvement of neutrophils and lymphocytes in the adverse outcomes of patients with coronary artery disease through the progression of coronary atherosclerotic plaques (14,16,27,38,42). Furthermore, various inflammatory markers are associated with the restenosis or neurological complications following CEA or carotid stenting, as well as with an unfavorable prognosis in acute ischemic stroke (3,7,13,20,23,39).

We investigated SII levels in patients who underwent CEA. The SII was initially introduced to assess the immune-inflammatory status and evaluate morbidity and mortality risk in cancer

patients. SII is calculated using the platelet, neutrophil, and lymphocyte counts and is considered as a modification of the platelet-to-lymphocyte ratio (PLR) and neutrophil-to-lymphocyte ratio (NLR) (47,50-52). Several studies have documented the prognostic value of SII in cardiovascular and cerebrovascular diseases (26,48). However, as pointed out, only one previous study has specifically assessed its potential as a prognostic indicator in patients who have undergone CEA(49).

Atherogenesis is a chronic inflammatory process involving an important interaction of damaged arterial wall cells, inflammatory cells, and cholesterol (27,31,38). Similar to that observed in coronary arteries, intimal carotid plaques are formed by the infiltration of inflammatory cells associated with a central lipid core covered by a fibrin cap (38). The development of lipid plaques and their subsequent expansion into the arterial lumen typically culminates in carotid artery stenosis, the potential for plaque rupture, atherothrombosis, and eventual distal embolization.

Neutrophils play a pivotal role in all atherosclerotic processes, including plaque development, remodeling, rupture, and reperfusion injury (19). They are among the initial inflammatory cells involved in arterial plaques, attracting leukocytes and promoting foam cell formation (25). Due to widespread inflammation and the redistribution of lymphoid cells, a decreased lymphocyte count is often associated with immune system depression and cardiovascular disorders (37). Increased platelet counts, levels of inflammatory mediators, platelet activation, a prothrombotic state, and a thrombus formation rich in platelets within atherosclerotic plaques can contribute to worse outcomes (2).

In this study, we underlined the SII level, a readily accessible and cost-effective test for CEA postoperative morbidity/mortality index.

CONCLUSION

This study indicates that routine hematological indices can provide valuable prognostic insights in patients undergoing CEA. Elevated SII levels are shown to have increased risk of stroke, mortality, and MACE in CEA patients. This index provides a simple and easy-to-apply method in clinics engaged in CEA.

Conflict of Interest: All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers; bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent- licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge or beliefs) in the subject matter or materials discussed in this manuscript.

Declarations

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Availability of data and materials: The datasets generated and/or analyzed during the current study are available from the corresponding author by reasonable request.

Disclosure: The authors declare no competing interests.

AUTHORSHIP CONTRIBUTION

Study conception and design: BCA
Data collection: EBM, OO, OMO
Analysis and interpretation of results: MZ, BCA
Draft manuscript preparation: MZ, BCA, OO
Critical revision of the article: MZ, UE
Other (study supervision, fundings, materials, etc...): MB, EBM
All authors (MZ, BCA, EBM, OO, OMO, MB, UE) reviewed the results and approved the final version of the manuscript.

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