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# MODERN VIEWS ABOUT THE STRUCTURE, FUNCTION AND BIOLOGICAL ROLE OF THE VASCULAR ENDOTHELIUM

**Umarov B.Ya., Khamdamov I.B.**
Bukhara Medical University named after Abu Ali Ibn Sina. Uzbekistan.

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**Abstract:**

In order to improve the results of diagnosis and surgical treatment of patients with atherosclerotic lesions of carotid arteries we investigated endothelial dysfunction in patients with atherosclerotic stenosis. A methods for diagnosis of endothelial dysfunction was assessment of changes in brachial artery diameter before and after a short limb ischemia and plasma homocysteine levels. Object of the study: 72 patients, 38 of which were undergone standard carotid endarterectomy, eversion carotid endarterectomy was performed in other 34 persons. Results and discussion. 57(79,2 %) patients had impaired vasodilation, 68 (94.4 %) persons had increased homocysteine concentration. After classical carotid endarterectomy homocysteine concentration was significantly increased from 15,90 ± 4,26 to 118,4 ± 4,2 μmol/l ($p < 0.05$), after eversion carotid endarterectomy — from 13,30 ± 3,27 to14,2 ± 5,6 μmol/l. According to the results of both tests more intensive endothelial dysfunction was observed in patients with diffuse atherosclerotic lesions. In the postoperative period, the growth of the homocysteine concentration was observed in the group after classical carotid endarterectomy in comparison with eversion carotid endarterectomy

**Keywords:** Atherosclerotic stenosis, endothelial dysfunction, carotid endarterectomy, homocysteine, endothelium-dependent vasodilation

Despite the measures taken, in developed countries stroke already ranks second among the causes of death, being between cardiac and oncological pathologies. Every tenth person in the world dies from a stroke, and only every third stroke patient returns to their previous work activity. Cerebrovascular diseases, and ischemic stroke in particular, remain one of the most pressing problems of modern neurology. According to some authors, a further increase in mortality from stroke is predicted in the future [1,11,17,19]. In 80% of cases, the lesion is ischemic in nature and in most patients it develops due to narrowing of the vessels supplying blood to the brain [2,4,18,20]. Most often, stenosis is caused by an atherosclerotic plaque in the internal carotid artery, which leads to a decrease in cerebral blood flow and creates conditions for the development of ischemic changes in the brain tissue. In order to prevent the development of ischemic disorders of cerebral circulation, a carotid endarterectomy operation is performed from the affected segment of the carotid artery. Despite the continued interest in the problem of choosing a method for surgical revascularization of the carotid arteries, risk factors whose correction will reduce the number of complications have not yet been systematized, and a modern prognostic model has not been developed regarding the outcomes of surgical treatment of carotid stenosis. Particularly relevant is the study of endothelial function in patients with cerebrovascular insufficiency, as the main pathogenetic link in arterial hypertension and atherosclerosis. [5, 16]. The vascular endothelium is a thin semi-permeable membrane lining the heart and blood vessels from the inside, which continuously produces a huge amount of the most important biologically active substances and controls many important functions [6-10]. It plays a very important role in angiogenesis due to the fact that it has vasomotor, antiplatelet, anticoagulant, thrombolytic, anti-inflammatory, antioxidant and antiproliferative activity [12, 13,21]. The barrier role of the vascular endothelium determines its main role in the human body: maintaining homeostasis by regulating the equilibrium state of opposing processes:

a) vascular tone (vasodilation/vasoconstriction);

b) anatomical structure of blood vessels (synthesis/inhibition of proliferation factors);

c) hemostasis (synthesis and inhibition of fibrinolysis and platelet aggregation factors);

d) local inflammation (production of pro- and anti-inflammatory factors.

The metabolic activity of the endothelium is manifested in the great vessels, exerting an additional influence on the entire circulatory regulation system. Vascular hypertonicity, increased peripheral resistance and compensatory arterial hypertension (AH) with all possible complications arise due to failures of vasodilation mechanisms, in particular, insufficiency of

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endothelium-dependent dilatation or endothelial dysfunction. Vasoconstriction, spasm of the muscle components of the vascular wall occurs much faster and more vigorously than their relaxation.

Endothelial dysfunction (ED) is manifested by a violation of the neurohumoral regulation of vascular tone, their remodeling and activation of the processes of thrombogenesis and inflammation in the vascular wall. In particular, it can cause vascular spasm, increased thrombus formation and increased adhesion of leukocytes to the endothelium, which is accompanied by impaired regional blood circulation and microcirculation. ED has been identified in metabolic syndrome and dyslipidemia and may be associated with obesity, hyperhomocysteinemia, sedentary lifestyle and smoking in [1,22]. After nicotine consumption, the number of circulating endothelial cells in the peripheral blood increases, which is a sign of increased endothelial desquamation. According to the hypothesis of atherogenesis, called "response-to-injury," it is damage to endothelial cells and the development of endothelial dysfunction that become the first stage of the development of atherosclerosis.

According to the results of a number of clinical studies, a test with EDVD according to D.S. Celermajer has been recognized as an adequate diagnostic test to diagnose ED [2,12,23]. Despite intensive study of ED, there is only limited data on the relationship between ED and markers of atherosclerotic peripheral vascular disease. Decreased brachial artery dilatation response in patients with peripheral vascular atherosclerosis has been associated with worse prognosis in several clinical prospective studies.

The endothelium has vasomotor, antiplatelet, anticoagulant, thrombolytic, anti-inflammatory, antioxidant and antiproliferative activity; it plays an extremely important role in the development of atherosclerotic changes in the vascular wall, vascular remodeling, and angiogenesis [1, 4, 15].

Endothelial function can be defined as the balance of opposing principles - relaxing and constrictor factors, anticoagulant and procoagulant factors, growth factors and their inhibitors. In turn, ED is a violation of this dynamic balance [6, 7]. Most biologically active substances secreted by endothelial cells, as well as those involved in the process of interaction with the endothelium, can be indicators of its function. ED, as the earliest phase of damage to the vascular wall, can be one of the sensitive markers of cardiovascular diseases [2].

**PURPOSE OF THE STUDY:** to improve the results of surgical treatment of patients with atherosclerotic lesions of the carotid arteries based on the study of endothelial dysfunction.

**MATERIALS AND METHODS.** The study was carried out on 72 patients who were treated in the department of surgery of the Bukhara branch of the Russian Research Center for Emergency Medicine. The main group included patients with hemodynamically significant atherosclerotic stenosis of the carotid arteries. Also, 48 (66.7%) of them had clinical manifestations of damage to another vascular system.

According to the type of surgical intervention on the brachiocephalic zone, patients were divided into two groups: group 1 - patients who underwent classic carotid endarterectomy (CEE) with artery plastic surgery with an autovenous patch (38 patients, 52.8%), group 2 - patients who underwent eversion CEE (34 patients, 47.2%). Ultrasound diagnostics were performed using an SSI 5000 Sono Scape device (made in China). To locate and measure the lumen and thickness of the intima-media complex and blood flow parameters in the common carotid artery, a linear sensor with a main frequency of 7.5 MHz was used. The lumen measurement was carried out in duplex mode with color Doppler mapping (CDC) of flows.

IMT was determined using ultrasound duplex scanning of vessels in B-mode. In accordance with the recommendations of the American Association for the Study of Arterial Hypertension, an IMT of less than 0.9 mm was considered normal, and intimal thickness equal to or exceeding 0.9 mm was considered thickening. [15]. All patients underwent laboratory tests, which included general clinical blood and urine tests.

Homocysteine was determined in blood plasma by the enzyme immunoassay method using high-performance liquid chromatography using Axis Homocysteine EI fluorimetric detectors (norm 8–12 μmol/L).

Statistical processing of data from the results of the experimental study was carried out using the licensed software package Statistica 6.0. The hypothesis about the normal distribution of the corresponding data in each group was tested using the Shapiro-Wilk test. In the case of normal distribution, Student's t-test was used to compare two independent groups for one characteristic; Pearson correlation analysis was used to analyze the relationship between two characteristics. If the hypothesis of normal distribution was not confirmed, the Mann-Whitney method and Spearman correlation analysis were used, respectively.

**RESULTS AND DISCUSSION**. One of the most practical methods for diagnosing endothelial dysfunction is assessing the change in the diameter of the brachial artery before and after short-term ischemia of the limb. Atherosclerotic lesion of the brachial artery is closely related to similar changes in the coronary and carotid arteries: impaired flow-dependent dilatation of the brachial artery is a predictor of cardiovascular

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diseases [9]. The technique used was developed in 1992 by D.S. Ceiermajer. To assess changes in the diameter of the brachial artery, a 7.5 MHz linear transducer of an ultrasound machine (SSI 5000 Sono Scape.) was used. The brachial artery was located in a longitudinal section 2-15 cm above the elbow, the image was synchronized with the R wave of the ECG. The study was carried out in triplex mode (B-mode, color Doppler flow mapping, spectral analysis of Doppler frequency shift). The reactive hyperemia test was performed in 72 patients. In two groups of patients studied, a comparative analysis of changes in the diameter of the brachial artery was performed 30 and 60 seconds after decompression (endothelium-dependent vasodilation - EDV). A normal reaction of the brachial artery was considered to be its dilatation against the background of reactive hyperemia by more than 10% of the initial diameter. A lower value or vasoconstriction was considered a pathological response.

When analyzing the functional state of the endothelium, a pronounced decrease in EDVD was determined in both groups. In patients of the 1st group of EDVD of the brachial artery, the average increase in the diameter of the brachial artery at the 30th second was 5.67 ± 1.54%, at the 60th second - 5.51 ± 1.64%, which turned out to be significantly lower. than in patients of group 2: 6.85 ± 1.70% and 6.13 ± 1.75% at 30 and 60 seconds, respectively ($p < 0.05$).

The results obtained show a significant degree of impairment of endothelium-dependent vasodilation in patients with stenotic atherosclerosis of the carotid arteries and confirm its greater severity in patients with diffuse atherosclerotic lesions.

Univariate correlation analysis of EDVD in both groups and some risk factors for cardiovascular diseases showed that a decrease in EDVD is significantly and inversely associated with diabetes, increasing age, body mass index, and thickness of the intima-media complex of the brachial artery (Table 2). Associations between EDVD and risk factors persisted when patients with diabetes were excluded from the analysis. Endothelial dysfunction is an early event in the development of atherosclerosis. The value of EDVD is reduced in individuals at high risk of developing atherosclerosis, although these associations are not always strong.

Another marker of endothelial dysfunction that we used was homocysteine levels. Homocysteine directly damages the internal arterial lining and also inhibits the synthesis of nitric oxide (NO) [3].

Homocysteine concentrations were determined in all study patients. Based on exceeding the threshold homocysteine concentration (> 10 mmol/l), endothelial dysfunction was identified in 56–64 patients.

Taking into account that an excess of homocysteine concentration of more than 10 μmol/L is considered by the authors [8] as an independent risk factor for thrombovascular disease, we used this value as a cutoff value. The average homocysteine concentration in group 1 (classical CEE) was 15.80 15.90 ± 4.26 μmol/l. In 12 16 (40 42.1%) of 38 patients, homocysteine levels exceeded 15 μmol/l. In group 2 (eversion CEE), the average homocysteine content was 13.10 13.30 ± 3.27 μmol/l. In 7 9 (26.5%) patients, the homocysteine concentration was above 15 μmol/l.

Thus, the concentration of homocysteine in the 1st group of patients was significantly higher than in the 2nd group ($p < 0.05$).

Correlation analysis of the relationship between homocysteine levels in both groups and some risk factors for cardiovascular diseases showed that homocysteine concentration is directly proportional to the level of total cholesterol and intima-media thickness.

In group 1 (classical CEE), the homocysteine concentration after surgery increased compared to the preoperative level to 18.4 ± 4.2 μmol/L. This difference from the preoperative level is statistically significant ($p < 0.05$). In group 2 (eversion CEE), the postoperative concentration was 14.2 ± 5.6 μmol/L and was not statistically different from the preoperative one.

Thus, our studies revealed that in the postoperative period, patients who underwent traditional CEA showed a significant increase in homocysteine concentration, in contrast to patients after eversion CEA, in whom the homocysteine level remained at the same level.

**CONCLUSION.** In patients with stenotic atherosclerosis of the carotid arteries, significant dysfunction of the vascular endothelium is detected, determined using functional and laboratory tests. In 57 (79.2%) patients with stenosing atherosclerosis of the carotid arteries, a violation of edvd was found. in 68 (94.4%) patients, plasma homocysteine levels exceeded 10 mmol/l.

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