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The Practical Significance of the VEGF Factor in the Acute Period of Ischemic Stroke

Tashkenov E. M.

Andijan State Medical Institute, Andijan, Uzbekistan

Abstract Stroke disease has a profound effect on the economy and health of the whole world and our country. In recent times, the diagnosis of stroke is focused on the dilution of laboratory methods, that is, it is introduced as a new approach in the study of the pathogenesis of the disease.

Keywords Ishemik insult, VEGF omillari, Ion tomirlar endoteliysi, Metabolizm

1. Introduction

Concepts about metabolism pathways expand and biomarkers involved in pathological processes lead to an increase in the amount of therapeutic targets and the opening of new doors in the future in medicine [1,2,3]. Biomarkers found in cerebral stroke include S100 protein, neuronal specific enolase (NSE), NMDA receptor protein or antibodies, basic myelin protein (MBP), s reactive IQs (SRP), interleukin 6 (IL6), Matrix metalloprotease (MMR2 and MMR9), and X. The production of an endothelial growth factor in the vascular endothelium increases when the brain infartis lacks oxygen to the tissue. Vascular endothelial growth factor or VEGF-A is part of the most studied group of proangiogenic factoryl. The endothelial growth factor is a potent stimulant of angiogenesis among the waxes of the Kup-numbered VEGF family. The VEGF gene is located on the short shoulder of chromosome 6, with receptors divided into 4 classes: VEGFR1, VEGFR2, VEGFR3, VEGFR4. These receptors are located primarily in endothelial cells, with small amounts in megacariocytes, monocytes, neurons, spindle hematopoietin, and tumor cells. VEGFR1 has been little studied, preventing it from binding to VEGF and VEGFR1, blocking the intracellular reaction cascade, lowering vascular permeability, reducing inflammation and angiogenesis [7,8,9]. VEGFR2 is the primary receptor, carrying out the transfer of the activated signal. This receptor binds to VEGF and stimulates intracellular proteinkinase. VEGFR2 activation increases endothelial cell differentiation, migration and proliferation, increases vascular permeability, promotes changes in inflammation, binds to neoangiogenesis and atherosclerosis, mobilizes endothelial cells. VEGFR3 is involved in lymphogenesis. In a mature organism, VEGF is located in all vascular tissues and is involved in the support of vascular hemostasis. This feature is the VEGF vasculoprotective effect: ensuring the formation and survival of endothelial cells, production of NO and prostacyclin, vasodilation, antithrombosis and proliferation of smooth muscle cells, activation of chemotaxis, support changes in the inflammatory process [16,17,18]. Under mechanical forces, as a result of the transformation, inflammation, hypoxia of the structure of the cell, the production of the VEGF factor begins under the influence of vasoactive hormones angiotensin II and vasopressin. Under tissue hypoxia conditions, VEGF does not wait for expression, but imposes an increase in capillary density and a low drop in arterial pressure. The VEGF factor ensures not only the formation of normal blood vessels, but also the formation and survival, and is also involved in the regulation of blood pressure. Prolonged vact causes decreased survival of endothelial cells, decreased terminal capillary and arterioles in tissues, increased blood pressure as a result of reduced VEGF levels in the blood or VEGFR receptor blocks. VEGF is a potent angiogenic glycoprotein, producing from a vascular endothelial cell, macrophages, neurons, and neuroglia in response to cerebral hypoxic damage [1,2,3,9,10].

The purpose of the work: to study the role of the VEGF factor in the acute period of ischemic stroke.

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2. Materials and Methods

We selected patients in a private hospital affiliated with Asaka neurology hospital LLC, conducted their clinical neurological examinations, laboratory tests of the Andijan region AIDS karshi wrestling Center in the study of blood VEGF levels. Venous blood serum of selected patients was taken. 3ml of venous blood was taken between 9.00-11.00 am of the day of the collected venous blood, and the Vakutayner containing the Clot Activator was delivered to the examination laboratory for 1.5-2 hours at the same time as turgi adhered to the transportable coids. The laboratory

vacuum cleaner was made up of a marked composition, then passed through 3000 speed/min centrafuga for 15 minutes, after which the serum was separated into an Epindorff plate test tube and-200s was frozen and sung in a sacking burn maxad for the IFA inspection method. To perform the IFA examination, we used the" VEGF-IFA-Best " test kit to anise the amount of VEGF (Vascular endothelial growth factor/endothelial growth factor) from acute cranial ischemia and blood serum from healthy group patients. The norm also equates to 691 me/ML under 20-50 years of age in the blood and the examination was carried out using Multiskan™ FC type 357 aparati (Thermo Fisher Scientific). It was also examined with patients ' NIHSS scale, Sollin index and Glasgow scale. A total of 89 patients were selected. We have divided into three groups of all patients. they are 25-76 years old with an average age of 56±1.5. The first group of patients consists of 32 patients who are hastened by the type of acute ischemic atherothrombotic stroke, of which 7 patients are women, 25 patients are men. The second group consisted of 29 patients with acute cerebral ischemia, 15 of them female patients, 14 male patients as well as a third group of 28 healthy individuals with 14 female and 14 male selectively.

3. Results

We used the manna-Uitna method in maksadi to compare the results of the three groups obtained.

Table 1. Tested patients on the NIHSS scale

< / Score >	I group	II group	III group	Reliability of results (r)
VEGF мЕ/мл	739±37.1	688±21.1	478±17.9	<0/001

When the first group (n=32) tested patients on the NIHSS scale, 2 (6%) Scored 1, 7(24%) scored 2, 2(6%) scored 4, 2(6%) scored 5, 4(12%) scored 6, 6(18%) scored 7, 2(6%) Scored 8, 5(16%) scored 11ball, 2(6%) scored 13 (Table 1).

The second group (n=29) scored 0 points in 8(30%) patients on the NIHSS scale, 1ball in 3(10%) patients, 2 points in 5(20%) patients, 3 points in 2(5%) patients, 4 points in 3(10%) patients, 6 points in 2(5%) patients, 7 points in 4(15%) patients, 8 points in 2(5%) patients.

The third group (n=28) scored 0 points in 16(65%) patients when examining patients on the NIHSS scale, 1ball in 3(10%) patients, 2 points in 3(10%) patients, 3 points in 2(5%) patients, 4 points in 2(5%) patients, 6 points in 2(5%) patients (Table 2).

When we evaluate the Colling index in the first group of patients. 5(18%) scored 0, 4(12%) scored 1, 2(6%) Scored 2, 4(12%) scored 3, 4(12%) scored 4, 2(6%) scored 6, 4(12%) scored 13, 5(18%) scored 14, 2 (6%) scored 15 (Table 2).

In the second group of patients, when assessed on the Colling index, it scored 2 points in 3(10%) patients, 6 points in 2(5%) patients, 11ball in 3(10%) patients, 13 points in 2(5%) patients, 14 points in 11(40%) patients, and 15 points in 8(30%) patients (Table 2).

Table 2. Results on the NIHSS scale in patients of the first, second and third groups

	First team (n=32)	Second team (n=29)	Third team (n=28)
0 points	-	30%	65%
1 point	6%	10%	10%
2 points	24%	20%	10%
3 points	-	5%	5%
4 points	6%	10%	5%
5 points	6%	-	-
6 points	12%	5%	5%
7 points	18%	15%	-
8 points	6%	5%	-
11 points	16%		-
13 points	6%		-

In a third group of patients, when assessed on the Colling index, 2 points were scored in 2(5%) patients, 11ball in 2(5%) patients, 13 points in 2(5%) patients, 14 points in 7(25%) patients, and 15 points in 15 (60%) patients (Table 3).

Table 3. Results on the Colling index in patients of the first, second and third groups

	First team (n=32).	Second team (n=29).	Third team (n=28).
0 points	18%	-	-
1 point	12%	-	-
2 points	6%	10%	5%
3 points	12%	-	-
4 points	12%	-	-
6 points	6%	5%	-
11 points	-	10%	5%
13 points	12%	5%	5%
14 points	18%	40%	25%
15 points	6%	30%	60%

Also, in the first group of patients, brachycephalic artery UZDS examination (N=32) 100% of patients in the second group are diagnosed with atherosclerotic plaques in 45% to 82% of cranial blood vessels, while in the second group of patients, in the Brachycephalic artery UZDS examination (n = 29) 65% of patients are diagnosed with atherosclerotic plaques in 25% to 64% of cranial blood vessels. In the third group of patients, in the examination of brachycephalic arteries UZDS (n=28), 40% of patients in the second group are diagnosed with atherosclerotic plaques in 21% to 32% of cranial blood vessels (Figure 1).

Analysis of the results obtained: in the first group of patients, the load on the amount of VEGF in the blood compared to the second and third groups of patients was anicized. In the NIHSS scale and the Colling index, the neurological deficit appears to be superior to the second and third group in the first group of patients. A cranial UZDS examination found that over 50% of atherosclerotic plaques were present in the second group of patients. That is, an

increase in the volume of plaque of the cranial blood vessels leads to an increase in the likelihood of developing a stroke, an increase in weight and a deepening of the neurological deficit. Also, the tugri to wait for the amount of VEGF in the blood is propartional.

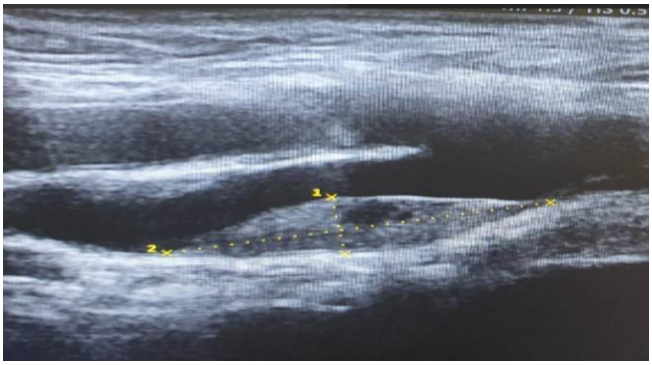

Figure 1. Left-sided common sleep artery in the intima and medial floors of low-density atherosclerotic plaque occlusion 77%

4. Conclusions

In the etiopathogenesis of acute cerebral ischemia, VEGF plays a role in neurospecific oxyl muxime, that is, an increase in the amount of VEGF in the blood during the acute period of patients with acute stroke is due to the chukurity and exacerbation of the disease process. It is also an important factor in assessing and predicting the effectiveness of the treatment of the disease.

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