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Short Communication

The Cause of Transient Ischemic Attacks

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Goal

Medicine has been facing the question for several decades: what is the cause of transient ischemic attacks? To answer "we don't know" is very embarrassing, so doctors need to say something about atherosclerosis, which everyone has.

Method

Study of numerous sources of information posted on the Internet. Discussion of proposed ideas at conferences, publication of original articles in Russian and English-language medical journals.

Results

Transient ischemic attacks (TIA) account for about 80% of all strokes. TIA occurs when micro vessels and capillaries in any part of the brain become clogged as a result of one of two mechanisms - either thrombosis or embolism.

So far, doctors have not answered the question, where do these obstacles to blood flow disappear 15 - 60 minutes after the TIA attack passes? Even such a question is not raised. In fairness, some modern doctors note: in a certain percentage of cases [1], even with a thorough examination of a patient who has suffered a transient ischemic attack, it is not possible to find out its cause, these TIA belong to TIA of unclear genesis.

Medicine as a science, of course, does not stand still, nevertheless, for more than two hundred years it has not answered the questions: why does atherosclerosis develop? What is the cause of ischemic brain attacks? Meanwhile, too many able-bodied people are losing developed countries due to strokes. Let's figure out what is the cause of transient ischemic attacks?

Of course, there must be one reason, not a whole list. The list that medicine demonstrates is related factors, but not the cause or mechanism.

So, why do TIAs of ordinary genesis arise? The doctors' answer is as follows. The main cause of TIA is atherosclerosis, which contributes to thrombosis, or with TIA, blood clots with blood flow can move from other parts of the body, for example, from the heart. And why does atherosclerosis develop? The answer is also not one, but 20 pieces. According to the author of the new theory, such an answer regarding TIA is not completely complete, which means it is wrong.

Our question as it was, and remains: where do microthrombs go after 15 - 60 minutes after TIA? And why does blood pressure rise very quickly to critical values at these minutes (and usually before this event)?

According to the new theory [2-4], TIA occurs due to insufficient arterial blood volume in the arteries. As an undesirable and unnoticeable event, blood can quickly flow from the arteries to the veins through open large arteriovenous anastomoses (AVA), while lowering

blood pressure. Under normal conditions, the AVA is in a closed state. The opening of the AVA lumen may be associated with physical or psychological stress. For example, when working continuously for several hours in a sitting position, at a computer or other device. Usually stress, low physical activity, excess weight, fatigue leads to an increase in blood pressure, but not to critical values.

With a significant leakage of blood, the internal volume of the arteries should adequately decrease by the same amount, and the venous volume should increase. But since the walls of large arteries usually become stiffer with age due to atherosclerosis and plaques, the decrease in the internal volume of the arterial bed is not due to the aorta and large arteries near the heart, but due to the small and most distant arteries from the heart. It can be assumed that in young people, the reaction to blood loss due to open anastomoses will proceed more gently, imperceptibly.

Everyone can observe: if your hands and feet have become "cold" in a warm room, it means that a spasm of small arteries has occurred. According to physical laws (gravity), the spasm primarily affects the vessels located in the upper parts of the body, in particular in the brain (even taking into account the adjustment of the pressure of the cerebrospinal fluid). The optimal pressure difference between arterioles and venules in some parts of the brain becomes critically reduced. The average pressure between them at this time also reaches a minimum.

With an increased glucose content in the blood, tissue perfusion also worsens, as blood fluidity decreases. The perfusion slows down or stops. At this time (at the moments of critical pressure reduction) gas bubbles, fat bubbles and micro emboli begin to form in the capillaries and in the brain tissue. All this is similar to the caisson effect when divers quickly ascend from the depths of the sea. Groups of small "balls", or emboli, do not occur at one or several points, but in a certain volume of brain tissue, on a group of capillaries most distant from a large artery. The organismal system, which monitors the normal perfusion of brain tissue, reacts to these events and requires an increase in blood pressure to nourish the brain, since the brain is the main human organ. But since the volume of arterial blood is already insufficient by this time, an increase in blood pressure can lead to even greater losses of arterial blood through the AVA and greater spasm. It is becoming increasingly difficult to restore normal perfusion. The situation is escalating. The arterial pressure exerted by the heart and large arteries continues to rise at these minutes, if possible. At such critical moments, TIA or a brain stroke may occur. It all depends on the effectiveness of emergency medical care.

For example, injection of a magnesia solution (or other medication) forcibly dilates arterioles, venules and other vessels in all organs. The volume of arterial blood increases rapidly due to the reabsorption of a certain amount of blood and tissue fluid through arterioles into large arteries. Gas emboli are spontaneously eliminated (collapse) when the pressure in the arterioles of the brain increases again. After a while, systemic pressure and arterial circulation are restored. If help is provided on time, the symptoms of TIA gradually disappear, memory is restored.

Now we can say that the main course of acute cerebrovascular accident is the loss of arterial blood volume - hence hypertensive crises, TIA and, ultimately, strokes. In 2018, the author himself experienced a hypertensive crisis, TIA and rehabilitation. The bitter experience suggested what could happen to the cardiovascular system and the human brain.

And doctors do not know this theory and are not guided by it…

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