ClinicSearch

Clinical Genetic Research

Vladimir E *

Open Access

Research Article

Age-related deformity of the arteries, inflammation and atherosclerosis. What is primary, what is secondary?

Vladimir Ivanovich Ermoshkin

Russian New University (RosNOU), Street Radio, Russia

Corresponding Author: Vladimir E, Russian New University (RosNOU), Street Radio, Russia.

Received Date: September 08, 2022; **Accepted Date**: September 15, 2022; **Published Date**: September 26, 2022.

Citation: Vladimir E, (2022) Age-related deformity of the arteries, inflammation and atherosclerosis. What is primary, what is secondary? *Clinical Genetic Research*, 1(1); Doi:**10.31579/2834-8532/003**

Abstract

After the publication of several articles on the possible cause and mechanism of atherosclerosis requires s some explanation for the better understanding of this problem. Readers can arose pull the question of why atherosclerosis is accompanied by inflammation? Or is it on the contrary, is inflammation accompanied by atherosclerosis? What does official medicine say about this? What is primary, what is secondary?

Keywords: inflammation; atherosclerosis; arteriovenous anastomoses

Objectives

After the publication of several articles on the possible cause and mechanism of atherosclerosis [1- 4] requires s some explanation for the better understanding of this problem. Readers can arose pull the question of why atherosclerosis is accompanied by inflammation? Or is it on the contrary, is inflammation accompanied by atherosclerosis? What does official medicine say about this? What is primary, what is secondary?

Method

Studying information on the Internet, participating in medical conferences, publishing articles.

Results

Earlier in N s th theory atherosclerosis [1- 4, 7] was show but that the cause of atherosclerosis of the arteries and aorta can be periodic arterial blood loss. Recall that the normal ratio of the blood volume of the track th present: arterial blood in humans 15-20% of venous - 80-85%. It is believed that the flow capacities of the two pumps - the left and right ventricles of the heart - are the same under normal conditions. Previously, it was about tmecheno that during stressful situations and rise in blood pressure (BP) can be opened large arteriovenous anastomoses. [5] This leads to a temporary leak through the arterial anastomosis of blood directly from the arteries into the veins, etc. and this AD with lowers the. An additional factor in increasing the imbalance in blood volumes is the Earth's gravity. Prolonged position of the spine in an upright position (with walking), and concomitant lack of exercise aiding w t increase the venous blood volume in the lower half of the body.

Of course, there are volume ratios recovery mechanism, and the imbalance between the average weaning venous and arterial blood gradually in a few hours eliminates is Busy. Replenishment of the volume of arterial blood is carried out by additional pumping of blood through the pulmonary circulation. This naturally occurs during physical exertion, through special breathing practices, while resting in a horizontal position of the body. In young people this happens quickly and imperceptibly, in the elderly slowly, ailments and diseases develop. By f course, much depends on the way of life of a particular person, such as the nervous system and other factors.

It can be assumed that in many people, periodic blood leaks and further volume replacement occur regularly and imperceptibly. It can also be assumed that gradually, along with the past years, the ratio of the volumes of venous and arterial blood in each person gradually changes. Most importantly, the average volume of arterial blood decreases. This means, that the physical body's capabilities also reduced, in some there is ischemia of organs. The volume of the arterial bed decreases in approximately the same proportion. Along with this, the stiffness of the arteries increases and the development of atherosclerosis occurs, the growth of plaques.

Now in more detail. P ri rubbed s, relative to the nominal volume, a volume of arterial blood arises forced reduction of the arterial bed. In this context, we consider the statics, but not the dynamics of the blood pumping process. Those. we do not consider the mechanics of heartbeats (pulse) in the arteries, laminarity or turbulence of flows in the arteries. From the viewpoint of static e the receptacle acit that all arteries with blood loss occurs some "constant spasm " artery walls, because blood practically does not change its volume either when stretched or

compressed. For example, the magnitude of the "constant spasm" of the walls of the arteries in a particular place can be determined by averaging measurements over a certain interval, for example, for 10-100 seconds. Averaging should subjecting be smiling and the thickness of the arterial wall, and exterior, and the inner diameter of the arteries. But such measurements have never been carried out, there are no corresponding instruments yet. While this is only virtual attempts something to measure and p have.

Of course, the amount of reduction of the internal "diameter" of the arteries along the vessels of the cardiovascular system of all time personal. In addition, according to the laws of hydromechanics positive and negative voltages in the walls of arteries are unevenly distributed, more significant - in places bending s vessels and at the bifurcation of the arteries. Exactly the same picture of the distribution of plaques and the degree of atherosclerosis is observed with a significant experience of cardiovascular diseases. First of all, such vessels include vessels located above the large venous valves, and, moreover, above the level of the heart. These vessels are aortas and, coronary, vertebral, carotid and cerebral arteries.

So, when the arterial blood leakage due to the opening of arteriovenous anastomoses, etc. oiskhodit strain I artery walls. Specifically, first of all, the inner diameter of the arteries decreases, while the outer diameter is relatively constant, i.e. relatively rigid adventitia. Simultaneously, compression of the unicellular layer of the endothelium occurs, because with spasm of the artery, the inner surface of the vessel (the surface area of the endothelium) decreases. Endothelial cells are displaced, compressed, intercellular clefts are reduced. In other words, dysfunction (alteration) of the endothelium occurs, the production of nitric oxide is disrupted , on which the vascular tone depends . The stiffness of the arteries increases somewhat - this is the initial stage of atherosclerosis.

And now a key factor of atherosclerosis, a key phenomenon, Kotor nd tried to find, but did not find doctors and researchers of atherosclerosis in the last 150 years. Consider a section of a common artery and its cross section. Because first of all, the inner layers of the artery wall are subject to spasm (compression) during blood leaks, while the endothelial layer in the form of a circle shifts (approaches) to the axis of the artery, and the outer layer (adventitia) practically remains without deformations and its diameter does not change , then the muscle layer the walls of the arteries (media) are forced to be stretched in the transverse direction .

Periodic, but with e -inflammatory bubbled periods of compression s endothelium and tensile s in the transverse direction the media - this is a physical cause for the occurrence of inflammation. Recall that according to all medical instructions, temporary, and even more permanent injury (damage) to an organ leads to its inflammation.

When tension and spasm of arteries media in the thickness of the artery (between the adventitia and endothelium) in oznikaet negative pressure, or in other words force " choke " causing sticking (adhesion) of platelets and light fractions of cholesterol to the wall am Arter and minutes. " Choke " liquids it is only possible either through the endothelium and of the arterial lumen, or through the adventitia of the zones of perivascular adipose tissue. Calculations show that the suction forces are more powerful in the adventitia zone. There are medical works confirming that often the first signs of atherosclerosis arise precisely near the adventitia. Some m -pocket medical researchers indicate that the first signs of atherosclerosis begin in layer e adventitia [8]. Now these researchers have arguments "for" a New Theory of atherosclerosis. Infiltration occurs through the endothelium of blood fragments from the main flow, primarily cholesterol low density, h Erez adventitia - fragments of perivascular adipose tissue. Therefore, in a layer of media may be provided any fractions of blood or tissue may be conceived n p oliferatsiya new cells. In parallel, oznikaet Calc and nirovanie and fibrosis of the artery walls. Plaques form in areas with the largest and most damaged endothelium. Negative pressure or suction force in the media, involving w T cells from adjacent layers of arteries or of adjacent organs. By notch from other tissues, which should not be in the media, can drive it and to inflammation. Therefore, it should be considered:

1) the primary link of atherosclerosis is the gradual deformation of the inner layers of the arteries due to a lack of blood in the arterial bed and the occurrence of suction forces.

2) and infiltration of foreign cells or substances in the media, vascular stenosis, inflammation and atherosclerosis is a secondary phenomenon!

We detail discussed the mechanism of atherosclerosis from the viewpoint of new theory of atherosclerosis [1 7]. The whole process of the pathogenesis of atherosclerosis at the biochemical, biophysical and genetic levels is very complex, diverse, its consideration, perhaps , will be only in the following articles. My task as a physicist was as follows: search and substantiation of the cause and mechanism of atherosclerosis at the physical, at the macro level. It is this level that has been inaccessible to doctors and researchers of atherosclerosis for more than 150 years, during which time about ten quasi theories of atherosclerosis were born in medicine. Now almost all these theories are united by the new theory, for all these theories a superstructure has been created that easily explains the causes of this disease. Let me remind you that atherosclerosis, according to the most conservative estimates, shortens a person's life by 10-20 years.

And now let's turn to the scheme of "Pathogenesis of atherosclerosis" by Professor V. Gurevich of Dpt them. And. And. Mechnikov, Department of Cardiology, Center for Atherosclerosis and Lipid Metabolism Disorders "Clinical Hospital No. 122 named after LG Sokolova " for 2010. Apparently, this scheme is an average assessment of the causes of atherosclerosis on the part of official medicine.

Here's what he writes. “Atherosclerosis is a systemic disease associated with damage to large and medium arteries of the muscle type, which is a combination of changes in all layers of the vascular wall, accompanied by local inflammation, endothelial dysfunction, proliferation and changes in the contractility of smooth muscle cells, the development of fibrous tissue with subsequent stenosis or occlusion, leading to hemodynamic disorders in the area of responsibility of the affected segment of the vessel. Morphological changes in the arteries are represented by atherosclerotic plaques with a lipid core and a fibrous capsule. "

In this definition of atherosclerosis, almost everything that is characteristic of atherosclerosis is said, and this statement is in good agreement with the data from the New Theory of Atherosclerosis. Only the cause and mechanism of atherosclerosis is absent.

The new theory named both the cause and the mechanism of atherosclerosis, and this is the main thing!

Conclusions

The author of this article and the hypothesis believes that the cause and mechanism of atherosclerosis have been named. No criticism is not yet so. The preliminary conclusion is that in order to prevent atherosclerosis, it is necessary to avoid stress, to prevent large leaks of arterial blood from the arteries into the veins. To this end, Mr. Parts Required regular physical activity and breathing exercises to increase the va be pumping in a small circle of blood circulation and replenishing I be arterial blood loss. Perhaps, there exists another solution to fill the volume of arterial blood to normal levels, for example, the direct effect of a temporary increase in blood pumping through the right ventricle.

How to maintain the ratio of arterial and venous blood volumes? That is the question now. This is the main challenge for the future.

One more task. How to reach out to medical leaders? How to reach out to academicians and doctors, on whose opinion the development of medicine depends? The New Theory of Atherosclerosis will be one year old in September 2021. None of the leaders wrote to me, no one called. How to reach out to the leaders of medicine and inform them that the era

of lack of knowledge about the causes of atherosclerosis is (apparently) over? It seems, from the leaders of other priorities ... Letters to the requests of new articles from the editors of medical journals from different countries are continuously every day. But I am alone, and there are many editors. How to convey to every doctor and researcher the New Theory, information that the cause of atherosclerosis has already been clarified and a serious joint discussion is required?

References:

1. Ermoshkin Vladimir Ivanovich. “The Mechanism of Atherosclerosis and Pathological Spasms of Human Arteries”. EC Cardiology 7.9 (2020): 69-74.
2. Vladimir Ermoshkin. “Possible Cause of Atherosclerosis from the Point of View of a Physicist”. EC Cardiology 8.6 (2021): 24-28.
3. Ermoshkin VI "Eurasian Scientific Association", "Atherosclerosis occurs due to loss of arterial blood", No. 9 (67) • September, 2020.
4. V.I. Ermoshkin, Atherosclerosis. There are several theories, can they be combined? Can! 2021.
5. Internet resource. Clinical case. Russian.
6. Yu.A. Romanov. Structural and functional features of human endothelium in normal conditions and in atherosclerosis. 2003. Dissertation.
7. IN AND. Ermoshkin. The main processes that occur in the walls of arteries in atherosclerosis, 25.06. 2021.
8. Di Wang, et. Roles of Cells from the Arterial Vessel Wall in Atherosclerosis. 2017.