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Research Article

The Main Processes that Occur in the Walls of the Arteries in Atherosclerosis

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Abstract

After the publication of several articles on the possible cause and mechanism of atherosclerosis, it is necessary to make some additional arguments for a better understanding of the problem. It is known that over the past hundred years, many theories of atherosclerosis have been put forward. But there is still no consensus, although the authors of the theories have already been awarded at least three Nobel Prizes. On the other hand, most honest researchers believe that at the moment the mystery of the mechanism of atherosclerosis has not been solved yet. Only a certain group of factors of atherosclerosis is known.

Keywords: Arteries; Atherosclerosis; heart

Introduction

After the publication of several articles on the possible cause and mechanism of atherosclerosis [1-4], it is necessary to make some additional arguments for a better understanding of the problem. It is known that over the past hundred years, many theories of atherosclerosis have been put forward. But there is still no consensus, although the authors of the theories have already been awarded at least three Nobel Prizes. On the other hand, most honest researchers believe that at the moment the mystery of the mechanism of atherosclerosis has not been solved yet. Only a certain group of factors of atherosclerosis is known.

Method

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

Results

A new theory of atherosclerosis [1-4] has shown that the cause of increased stiffness of the arteries, including the aorta, may be periodic loss of some volume of arterial blood. Recall that the normal ratio of blood volumes is as follows: venous blood in humans is 80-85%, arterial blood is 15-20%. It is believed that the throughput capacities in terms of blood pumping volumes for the left and right ventricles of the heart are the same. It is noted that during stressful situations and an increase in blood pressure (BP), large arteriovenous anastomoses can open [5]. This leads to leakage of arterial blood through the anastomoses directly from the arteries to the veins. An additional factor in increasing the imbalance of blood volumes is the gravity of the Earth. Prolonged position of the spine in an upright position (standing or sitting) and inactivity also contributes to an increase in the volume of venous blood, mainly in the lower half of the body. Of course, there is a mechanism for restoring the volume ratio, and the imbalance between the average weaning of venous and arterial blood is gradually eliminated. Replenishment of arterial blood volume is carried out by additional pumping of blood through the small circle of blood circulation. This naturally occurs during physical exertion, massage, through breathing practices, while resting in a horizontal position of the body, plus a healthy sleep at night. In young people, this happens quickly and imperceptibly, in older people it is slow. Of course, a lot depends on the lifestyle of a particular person, the type of nervous system and other factors.

It can be assumed that many people have periodic blood leaks and further replenishment of volumes occur regularly and imperceptibly. It is also possible to make assumptions that gradually, along with the years lived, the ratio of venous and arterial blood volumes gradually changes in each person. The main thing is that the average volume of arterial blood decreases. Approximately in the same proportion, the volume of the arterial bed decreases, the stiffness of the arteries increases, and the development of atherosclerosis and the growth of plaques occurs. The installation of stents cannot solve the problem at the root, because a global lack of arterial blood after some time can lead to spasm of the same artery, but in a slightly different place, or other arteries will be subjected to atherosclerosis.

What can happen with a single, but significant leakage of arterial blood in a young person? It is obvious that the average volume of the arterial bed decreases by the same amount. If the leakage is significant, for example, due to severe stress, then the so-called forced spasm of the arteries occurs, i.e. the walls of the arteries try to get closer towards the axis of the vessel by a certain amount, in other words, the "diameter" of the artery and the circumference of their lumen become smaller. During

these periods of time, the walls of the arteries deviate from their average non-stressed position. At the same time, the main load on the walls of the arteries is as follows: there are forces of separation of the endothelium from the intima due to a lack of arterial blood, these forces are transmitted through the middle elastic layers of the wall to the hard outer layer of the adventitia. But the adventitia usually does not spasm, it is much harder than the other layers of the vessel and holds the outer layer of the arteries almost unchanged.

Note here that the forces of separation of the endothelium from the media are additionally increased when this vessel is located in a vertical position relative to the Earth's surface. This mechanism is described in more detail in the article [2]. The maximum values of these forces are noted in the upper parts of these arteries or the aorta. It is these forces that can lead to a rupture (dissection) of the aorta. All this can be shown and proved using the laws of physics, specifically hydrostatics and hydromechanics.

Depending on the degree of reduction of the "diameter" of the artery, the degree of reduction of the length of the "circle" and the area of the lumen of the artery in a particular place is easily calculated. Over the years, changes occur in all arteries, and in large ones especially, all this leads to a significant compression of endothelial cells. The lumen of the artery is proportionally reduced. Intercellular interaction in a dense row of endothelial cells (EC) is disrupted. To keep the process of reducing the diameter of the arteries, the inner layers of the artery walls become tougher and tougher over the years.

Now it may be clear that it is not sessions of high blood pressure that lead to atherosclerosis, but the consequences after them. In fact, atherosclerosis can progress both with normal and with low blood pressure. The main reason is the lack of arterial blood in the arterial bed.

What happens directly in the artery wall? When stretching the inner layers of the artery wall (increasing the distance between the rigid adventitia and the endothelium), a pressure drop is created inside the elastic media layers, i.e. in the thickness of the artery walls, in the space between the endothelium and the rigid adventitia. As a result, areas with negative pressure appear inside the multilayer wall. Right in the thickness of the vascular wall, the forces of "suction" arise.

(This is equivalent to stretching the bellows on an accordion. When stretching the accordion without pressing the buttons, a negative pressure is formed inside the bellows, and the surrounding air, along with fine dust, rushes into the bellows through any holes! This is the" infiltration " of air together with dust into the accordion!)

But let's return to the arteries. Apparently, the" suction" of fluid is not possible through adventitia, but through the endothelium is possible, because the forces of separation due to the loss of arterial blood volume constantly damage the flat cells of the endothelium. The forces of "suction" through the gaps between the endothelial cells and through damage in the endothelial layer affect the blood flow directly. The lightest and smallest fractions of blood begin to penetrate under the endothelium or under the intima, and there they can accumulate in the form of a so-called "mush". In official medicine, this phenomenon is called "infiltration". The lightest fractions, as plaque researchers have been telling us for many years, are low-density lipoproteins (LDL), or "bad" cholesterol, plus any other light fractions of blood. In parallel, the work of the "vessels of the vessels" is disrupted, there are reasons for inflammation. Plaques grow in places of significant damage to the endothelium.

This is how the development of atherosclerosis occurs, this "development" goes on almost all your life, because the drop in arterial blood volumes can be slowed down or stopped, for example, by changing your lifestyle, by creating incentives for the growth of new arteries, but due to the progressive stiffness of the arteries and the accumulation of "mush", it is not possible to restore the average volume of the arterial bed, which was, for example, ten or more years ago.

Naturally, first of all, the increase in stiffness occurs on the apical side of the epidermal cells in contact with the blood flow. After all, otherwise there may be a critical decrease in the lumen of all the arteries and subsequent ischemia of many organs, which is unacceptable, so the body adapts, the walls become tougher.

Are there any prospects for preventing the development of atherosclerosis? Of course, they are there. But so far it is unrealistic for the purpose of preventing or "treating" atherosclerosis in a particular person to constantly or periodically pump blood into the arteries (observing blood pressure) with the help of an "additional pump" or periodic blood transfusion from the veins into the arteries. These, apparently, will be the developments of the future, there will be many of them. The main thing is to understand the causes and mechanism of atherosclerosis.

From this day the main cause of atherosclerosis has become clear! In addition, if we analyze the old theories of atherosclerosis, then the new theory becomes a theory that unites most of the old theories! It has become a superstructure over previous theories.

It seems incredible that medicine has spent more than 150 years searching for the cause of atherosclerosis, and the mechanism of atherosclerosis (its physical root cause) turned out to be extremely simple and can now be understood even by a high school graduate. On the other hand, it is known that there was and still is a natural selection for future doctors: school graduates who did not like mathematics and physics went to medicine. That's what it means to know physics badly.

So, to preserve health at this stage of the development of medicine, we can still recommend regular physical activity, breathing practices, special exercises, stress minimization, a varied diet, etc.

Now we can say that the main secret of atherosclerosis is no longer there. And cholesterol, consumed in moderation, of course, is not to blame.

It is necessary to outline steps to really help a person, starting from 2021, when this discovery in medicine occurred. And do not now refer to an erroneous, rejected reason. Allegedly, “the cause of atherosclerosis is a chronic disease (!) That occurs as a result of a violation of lipid (!) and protein (!) exchanges."

A few more facts in favor of the new theory of atherosclerosis, found by the author on the Internet.

1. Previously” Oberleithner H. my colleagues and I found out [6, 7] that endothelial cells (EC) can be conditionally "soft "(they produce more nitric oxide) and "hard". Later, this phenomenon was called "rigid endothelial cell syndrome". Author's comment: in areas where the epithelial separation forces from the intima are of greater amplitude and occur more often in time, there is an adaptation of the cardiovascular system in the form of an increase in the stiffness of surface cells in contact with the blood flow.

2. " If we consider the content of multinucleated cells as one of the signs of the upcoming aging of the endothelial population in a particular zone, it can be assumed that in the zones of HPA (high predisposition to atherosclerosis) these processes are more active and are ahead of those in the zones of LPA (low) by an average of 20-30 years [8]." The author's comment: apparently, due to the loss of arterial blood, the appearance of multinucleated endothelial cells is the result of a strong and constant compression of endothelial cells along the inner closed ring (along the circumference of the artery lumen). As a result, there are violations of intercellular interaction, aging, apoptosis and EC dysfunction.

3. " Under normal conditions, endothelial cells are a layer of flat cells. As a rule, they have a polygonal shape. Their length ranges from 20 to 150 microns, width from 10 to 20 microns... Over time, the already flattened endothelial lining becomes thinner, but the intima as a whole thickens. This is primarily due to edema of the subendothelial space. It also accumulates protein substances [6] - both fine-dispersed and coarse-dispersed, decomposition products of the extracellular matrix, swelling and decay of the fibrous component are detected. In addition, smooth muscle cells migrate from the middle shell to the intima, where they continue to proliferate. "Comment of the author of the article: these findings contribute to a convincing confirmation of the new theory of atherosclerosis!

Conclusions

The author believes that the underlying causes and mechanism of atherosclerosis in humans are now becoming revealed and shown. It turns out that there is no violation of lipid and protein metabolism in the artery wall as the root cause of the disease. The main reason is the periodic loss of the necessary volume of arterial blood in people and the accompanying negative pressure and infiltration that occur in the thickness of the artery walls.

The author of the new theory of atherosclerosis has questions for the heads of medicine. These are the questions.

Do specialists have any objections, criticism or additions to the New Theory of Atherosclerosis?

Who will tell and help how to act in this situation to a single physicist, the author of the Theory of atherosclerosis?

What should be done to get this article on the table of the Minister of Health?

Or is it still necessary to "play silent"?

And yet, perhaps, an epoch-making event in medicine has happened, I will not be afraid of this word.

I apologize in advance if I accidentally offended anyone of the academics.

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