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Acute Kidney Failure: Suspicion Falls on Arteriovenous Anastomoses (AVA)

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Abstract

In official medicine, it's nowhere said that uncontrolled leakage of arterial blood through arteriovenous anastomoses (AVA) is very dangerous for human health. The purpose of AVA, apparently, is the regulation of heat exchange of the human body, limiting the maximum blood pressure, regulating the flow of arterial and venous blood by opening a direct communication between the arterial and venous basins. Because of the high pressure difference between the basins (an average of about 50-60 mmHg or 680-820 mm H2O), the blood flows intensively from the arteries to the veins. Depending on stress and charges, the AVA lumen may change, i.e., AVA periodically can be in both open and closed positions. The very presence of AVA of different calibers in the cardiovascular system of man is not denied by anyone, but no one has studied in details the effect of AVA on blood circulation in various organs of man. At least until 2011.

Introduction

It is the complex effect of blood leakage that can explain many cardiovascular diseases (CVD). We emphasize: leaks from the arterial system and, accordingly, overflow of the venous system. Almost all of my previous work published since 2011 [1-17] are just related with the effect of blood leakage through large arteriovenous anastomoses (AVA). Proceeding from simple logic, the main purpose of large AVA is an emergency lowering of peak values of arterial pressure in the system.

The AVA is opened – the blood pressure (BP) falls rapidly, the AVA closed - the pressure rises again. Thus there are so-called "jumps of blood pressure". The BP jumps are the syndrome of "Ermoshkin-Lukyanchenko" [3]. But sometimes, in an unhealthy way of life, the AVA's wrong manipulations are possible: after opening, they can close with more delays than necessary, or be constantly pathologically open. Over time, leakage of arterial blood into the venous system through AVA can overflow the venous bed.

According to the New Arrhythmia Theory [3], the overcrowded hollow veins (vena cava) can lead to the fullness of the liver and spleen. In addition, overcrowded hollow veins can serve as a "conductor" of mechanical impulses that suddenly begin to run many times in the same circle: the left ventricle, the aorta, the artery, the AVA, the liver, the vein, the hollow vein, the heart and trigger the mechano-induced excitation of the cardiomyocytes either from the atria or from the ventricles. As a result, either atrial or ventricular paroxysmal tachycardias are possible with equal QQ intervals on the ECG, respectively with narrow or wide QRS dents. Intervals are equal to each other because the traveling times of a mechanical wave along the same vessels are almost the same. Note that under other conditions, there are only single extrasystoles, or other types of arrhythmia.

Discussion

Further study of the effect of open AVA or AVA with a violation of their functionality showed that these small connective vessels may be involved not only in arrhythmia, but in other diseases [2-4,15,16]: for pelvic organs, metabolic disorders, to systemic inflammation, to venous insufficiency of the brain, to heart failure and many others.

And now let's consider the mechanism of chronic (CKI) and acute kidney injury (AKI). It is believed that the mechanism of kidney injury (KI) is currently unknown. The searches have been conducted for more than 3000 years, intensively - about 100 years. The AKI is a sudden (within a few hours or days) potentially reversible renal dysfunction due to the effects of various exogenous or endogenous factors with a delay in the products of nitrogen metabolism, a dysfunction of the water-electrolyte and acid balance [18]. The AKI is a state that results from a disorder of the kidney blood flow, damage of the glomerular membrane of the nephron, sudden obstruction of the ureters, while the levels of urea and creatinine in blood plasma are rapidly increasing. The CKI are the same disorders that pass a little noticeably and at a much slower pace.

Annually about 150 people out of 1 million need urgent help for acute renal insufficiency. As a rule, two-thirds of them need hemodialysis and hemosorption. Even against the background of treatment, mortality in all forms of acute renal insufficiency reaches 20-50%. The usual recommendation of doctors: we must seek and treat the underlying disease, then treat renal insufficiency. More complete information about the current state of the problem of renal insufficiency, diagnosis and treatment can be found in the open literature [19-25].

But there is a new theory, a new mechanism for the emergence of cardiovascular and other human diseases is proposed.

The present article proposes the selection and consideration of arguments in favor of the new mechanism of renal insufficiency in the form of questions and answers.

- Renal insufficiency can be acute and chronic. Why?

Reason: (Here and below, the answers are given in accordance with the New Theory). If, after the opening of the large AVA, venous pressure in the entire venous pool and, under certain conditions, increases rapidly in the renal vein, the AKI may occur. If the leakage of arterial blood through AVA is small, but prolonged with periods of remission, then the CKI arises. The speed of the pathological process depends on the topology of the arteries and veins, their throughput, the availability of collaterals completely different for each individual, the presence and proper operation of venous valves, atherosclerosis. The open AVA create an increased venous pressure in large veins. This pressure, spreading through the renal veins, reaches the renal venules, partially or completely blocks the capillary circulation in the kidneys. The formation of urine is disturbed. Necrosis of kidney cells and KI is gradually developing.

- In renal failure, the pressure in the renal veins, as well as in other veins of the great circle of blood circulation increases. In this connection, the question arises whether renal venous hypertension is associated with abnormal water and sodium retention.

Reason: Yes, there is a connection, because the progenitor (source) of increasing venous pressure is the open AVA, overflow of the venous pool, increased venous pressure in large veins only. Of course the CNS also plays a role, but not the main one. Venous blood mixed with arterial blood moves not only to the right atrium, but also starts moving towards small veins and venules, i.e. moves in the opposite direction relatively to normal motion. The fluid moves where the pressure is less. With open AVA, the pressure gradient between arterioles and venules in some organs becomes insufficient. The capillary circulation slows down, stops, or pendulum movement of blood occurs in the capillaries due to residual arterial pulsation. Disorder occurs primarily in those organs that are located in the lower half of the body, where there are damages of venous valves. These are the organs of the small pelvis and legs. In a person who is in a vertical position, only working muscles and periodic sharp movements of the diaphragm upwards (for example, during running, swimming or performing special exercises) can pump venous blood. Further, because of blood stasis, there are a limiting expansion of veins, the yield of blood from the vascular swelling, varicose and thrombosis As a rule, first of all, pathological changes occur in organs that have been below the heart for a long time (for example, all the time of wakefulness during the day). And the lower the organ, the more essential pathology is the more significant delays of liquids.

- The AKI often occurs in patients in hospital who have already been hospitalized with severe illness or injury. The hospital mortality rate may exceed 50% [26].

Reason: The AKI in weakened patients develops quickly because they have to stay in the lying position for a long time. Besides that, there may be additional causes of KI – the iatrogenic causes, due to medication and surgical intervention. In the lying position with open AVA venous blood predominantly does not flow, as usual, into the legs and into the pelvic organs. In these organs, when the position is lying, the hydrostatic pressure component disappears and the venous valves begin to work better in easy conditions. This means that the pathological venous pressure can spread not only down, but also to any other organs located near the AVA. Such organs can primarily include organs of the upper body, including kidneys and renal veins, especially because those veins do not have valves. Further, blockage of capillary circulation in the kidneys and sudden development of arterial arrest may occur.

- Usually, with the development of renal insufficiency (and with some other diseases too), doctors say: it is necessary to treat a "basic" disease [23,24].

Reason: This is a cunning medical course due to a lack of understanding of the mechanism of almost all cardiovascular diseases (CVD). Doctors are looking for a "basic" disease and often "find" it. From the point of view of the New Theory of CVD, such a move can be justified by the fact that open or not functioning AVAs are the cause of not just one single CVD, but many diseases at once. The opened AVA increase venous pressure not only in the vena cava, but also in many other veins, depending on the topography of the middle and small veins, their throughput, the presence of collaterals, the state of the venous valves. In some veins before, in some later. The lifestyle and commitment to moderate physical activity are also important for the organism's "choice" which disease will be the first, what's next. This is confirmed by the statistics of morbidity: usually in old age there is not one, but several diseases at once. And every person, except for hereditary diseases, has its own sequence of various diseases. But arrhythmia, heart failure, systemic inflammation, renal insufficiency, cancer, leg diseases, pelvic organs diseases are always in the forefront, as they, from my point of view, are most likely with an increase in venous pressure.

- First place in the frequency of development of the AKI occupy severe somatic (shock) conditions, accompanied by a decrease in cardiac output [27].

Reason: With open AVA, blood flows freely from the arterial basin into the venous pool. This leads to an imbalance in the volume of arterial and venous blood. And with a decrease in the volume of arterial blood, i.e., with hypovolemia, naturally, cardiac output is reduced.

- The AKI and CKI are characterized by a slowing of blood flow in the kidneys, and the slowdown, according to medical observations due to significant blood loss, for example, during surgery or injury.

Reason: Not only in operations and injuries, arterial blood loss occurs. With open AVA, the blood flows freely from the arterial basin into the venous pool. This is also a loss of blood, or hypovolemia. And the hypovolemia is the cause of acute and chronic KI.

- The criterion for the presence of the KI is the total volume of urine released by the body, for example, per day [24].

Reason: With open AVA the blood flows from the arterial basin to the venous pool. This can lead to an increase in venous pressure in the kidneys. There is a blockage of blood circulation. The excretion of urine in patients in comparison with healthy people, naturally, decreases.

- As the liquids and the final metabolic products formed in the body accumulate in the blood, the acute renal insufficiency can lead to serious consequences, including the development of infection (sepsis) [25].

Reason: In addition to what has been said, the cause of sepsis and systemic inflammation can be not only the kidneys in which necrosis due to stasis occurs, but also other organs that have been previously more affected by the pathological action of open AVA anastomoses, for example the organs of small pelvis or liver.

- The AKI may occur with severe heart rhythm disturbances.

Reason: There is such a connection as the only cause and mechanism of AKI and severe cardiac rhythm disturbances are open AVA. The AKI occurs due to overflow of renal veins, and arrhythmia - because of the run of mechanical impulses over congested hollow veins.

- Diuretics have no effect on prognosis and mortality from AKI [24].

Reason: According to the New Theory, diuretics can either worsen a patient's condition, or at best have no effect. The fact is that with open AVA there is either a constant or periodic leakage of arterial blood from the arterial pool into the venous bed. This violates the required ratio of volumes in the two basins. When venous pressure rises, excessive venous blood can stagnate in all organs of a person, depending on the prevailing posture of a person, but more often in the pelvic organs and lower extremities. Additional losses of arterial blood with the help of diuretics can cause only harm. To somehow observe the balance of blood volumes, venous blood in people with venous fullness and excess weight is eliminated from the body with the help of abundant sweat at low loads or even without it.

- Most often, the AKI (prerenal) develops as a result of impaired renal hemodynamics due to severe hypotension. When the systolic blood pressure drops to 50 mmHg. The glomerular filtration stops and anuria develops.

Reason: Naturally, hypotension occurs due to prolonged leakage of arterial blood into the venous bed. Most often it can be in the elderly. Non-permanently opened large AVA, or gaping AVA, lead to periods of prolonged hypotension. In such cases anuria can fail to arise, because there is no driving force for the formation of urine and its excretion from the kidneys.

- Sometimes AKI develops with bilateral thrombosis of the renal arteries or veins.

Reason: When capillary renal circulation is blocked due to open AVA, the pressure gradient between arterioles and venules becomes critically small. The blood movement near the kidneys stops both in the arterial channel and in the venous beds. Over time, there is stasis and thrombosis.

- Sometimes there is an increase in venous pressure, there is a lack of capillary circulation (ischemia) in some organs, there is cardiac and/or renal insufficiency, but the arterial cardiac output is normal. Why?

Reason: We can assume that in a healthy person, not counting special cases when any physical or psychological stress is exercised, the average blood flow at four control points of the cardiovascular system is almost the same. These points are: the mouth of the hollow veins at the entrance to the right atrium, the pulmonary trunk, the mouth of the pulmonary veins, and the aorta at the exit from the left ventricle. At the same time the capillary circulation is normal in all human organs. But if in a person the AVA open for a "long" time, it means that some organs will lack circulation, deterioration in the perfusion of some organs due to the rise in venous pressure in the veins and to the blockage of blood circulation. This is physics.

The average blood flow in the aorta may remain the same or even slightly increase due to hypervolemia, but some organs may experience ischemia due to stasis. Why? Because a part of the blood is not sent to the organs, but passes directly to the large veins through the AVA. This is how partially "useless" heart work is carried out. That is why cardiac output can be normal, and capillary circulation in some organs is impaired.

- Sometimes the AKI develops due to kidney stones or tumors, to severe vomiting, or to diarrhea [23,24].

Reason: In this case, if such clinical facts are present, a new theory cannot add anything concrete: it is possible.

Conclusion

We cannot hush up the proposed theory - the mortality from CVD and cancer is very high and tends to increase. Modern treatment of almost all CVD is symptomatic. The results are weak. There is no single theory of CVD at all; this task is simply not solved, although, at least for more than 50-100 years, the CVD problem has been one of the major medicine problems.

I think the above arguments in support of the New Theory plus information from other sources about the New Theory [3,15-17] should cause just confusion among the researchers of the cardiovascular system, doctors, people responsible for the development of medicine, primarily for development cardiology. It turns out that if the New Theory is accepted, tested and proven, then the entire mosaic foundation of the old cardiology will simply collapse. Existing provisions in cardiology, methods of treatment, medicines - everything needs to be reviewed. If many human diseases are caused by open arteriovenous AVA anastomoses, then AVA should be treated. But the main task is the timely detection and observation of AVA within various organs. The AVA should work adequately: when the blood pressure is raised above a certain level, it opens, when the blood pressure drops below a certain minimum level, it closes tightly again. An additional way of solving the problem is possible: to develop artificial AVA with adjustable lumens and to carry out the installation of AVA according to indications.

Who from serious medical structures will risk supporting the proposed New Theory and developing it? I have been waiting for more than 6 years. Several times I spoke at conferences. Usually they say: it's interesting, but it has to be proven. Only ordinary cardiologists write letters to me and support me. Except them, editors of medical journals and organizers of medical conferences are showing great interest: every day I receive up to 5-10 letters with offers via e-mail from different countries. So far, that's it…

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