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Research Article

WHEN THE WHOLE WORLD, STARTING FROM DECEMBER 2019, IS CONCERNED ABOUT THE "CORONAVIRUS" -COVID-2019, HOW CAN BIOPHYSICS HELP?

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ABSTRACT

This paper analyzes modern methods and technologies for diagnosing and treating patients with coronavirus. And so, when the coronavirus settles in the endothelial cells (the inner wall of the blood and lymphatic vessels), a thrombus (thrombosis) is created. Thrombosis - intravital formation of blood clots (thrombi) inside the blood vessels, preventing the free flow of blood through the circulatory system.

KEYWORDS

Coronovirus, thrombus, thrombosis, hemocoagulation , viscosity, hemostasis, coagulation, vasospasm, adhesion, fibrinogen.

INTRODUCTION

When a blood vessel is damaged, the body uses platelets and fibrin to form a blood clot to prevent blood loss. Under certain conditions, blood clots can form in the bloodstream even without vascular

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damage. The cell is held together by membranes. 10-6 cm2 membrane plane has from 10 to 500 channels. Coronavirus clogs these channels, creates a blood clot and the cells begin to die. The body at this moment does everything to restore these damages as quickly as possible. This is due to the process of blood clotting, when a lot of viruses enter the body at the same time and they infect a large number of blood vessels. Blood coagulation (hemocoagulation) is the most important stage of the hemostasis system, responsible for stopping blood loss in case of damage to the vascular system of the body. Clotting leads to imbalance and starts to create a lot of blood clots and this is the main cause of death. By determining the viscosity and coagulability of blood in humans, it is possible to determine in what form a person will have an infection - in a mild or severe form.

At such moments, so that the disease does not proceed in a severe form, it is necessary to regulate the activity of the blood coagulation system, preventing the coagulation system from being strongly activated, i.e. suppress it for a while, thereby providing the lungs with a few days of respite until the immune system defeats the virus. To prevent negative consequences, it is required to fully decipher the mechanism of blood coagulation. Blood coagulation is preceded by the stage of primary vascular-platelet hemostasis. This primary hemostasis is almost entirely due to vasoconstriction and mechanical blockage of platelet aggregates at the site of damage to the vascular wall. Actually, blood coagulation (hemocoagulation, coagulation, plasma hemostasis, secondary hemostasis) is a complex biological process of formation of fibrin protein strands in the blood, which polymerizes and forms blood clots, as a result of which the blood loses its fluidity, acquiring a curdled consistency.

METHODS

The process of hemostasis is reduced to the formation of a platelet -fibrin clot. Conventionally, it is divided into three stages: 1. temporary (primary) vasospasm 2. platelet plug formation due to platelet adhesion and aggregation; 3. retraction (contraction and compaction) of the platelet plug. Vascular injury is accompanied by immediate activation of platelets. Adhesion (sticking) of platelets to connective tissue fibers along the edges of the wound is due to Glycoprotein von Willebrand factor. Simultaneously with adhesion, platelet aggregation occurs: activated platelets attach to damaged tissues and to each other, forming aggregates that block the path of blood loss. A platelet plug appears. From platelets that have undergone adhesion and aggregation, various biologically active substances (ADP, adrenaline, norepinephrine, and others) are intensively secreted, which lead to secondary, irreversible aggregation. Simultaneously with the release of platelet factors, thrombin is formed, which acts on fibrinogen to form a fibrin network in which individual erythrocytes and leukocytes get stuck - a so-called platelet -fibrin clot (platelet plug) is formed.

RESULTS

Thanks to the contractile protein thrombosthenin, platelets are pulled towards each other, the platelet plug contracts and thickens, and its retraction occurs. In its simplest form, the process of blood coagulation can be divided into three phases: 1-phase activation includes a complex of successive reactions leading to the formation of prothrombinase and the transition of prothrombin to thrombin; 2- phase of coagulation - the formation of fibrin from fibrinogen; 3-phase retraction - the formation of a dense fibrin clot. The lungs may be the epicenter of Covid-19, but scientists believe it can also enter the circulatory system by infiltrating our

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arteries. How exactly the virus attacks the heart and blood vessels remains a mystery, but experts believe the infection can cause blood clots and heart attacks because the virus binds to ACE2 receptors on the cells that line our blood vessels and heart. Patients with Covid in the hospital show signs of serious heart and vascular problems. And We see that in a significant proportion of patients, blood clots form in the arteries entering the heart. One study published in the scientific journal JAMA Cardiology found that 20 percent of 416 patients hospitalized in Wuhan with the coronavirus had heart damage. An increased likelihood of blood clots appears to be one of the features of severe Covid disease. This may explain why patients with diabetes and cardiovascular disease are at risk for severe illness with Covid-19, as their vascular system is already under additional stress. Biologically, the process of thrombosis is the norm - this mechanism is designed to protect a person from heavy bleeding, and natural thrombolysis is sufficient to destroy small blood clots, platelets and proteins. But with coronavirus, the process of thrombolysis will be difficult, the vessels (and the heart) experience a very high load, including toxic viral, cytokinin , and the risk of thrombosis increases by 3-6 times! If a patient suffers from severe viral pneumonia, has a history of atherosclerosis, aneurysms, chronic cardiovascular pathologies, then coronavirus can turn into a disaster. The vascular walls become thinner, and at the same time, blood clots are formed, which dissolve with great difficulty, especially at high temperatures. In some patients with coronavirus, a cytokinin storm occurs - an excessive, pathological immune response to the virus with the release of a huge amount of protective cytokine molecules. However, they kill not only the virus, but also destroy healthy cells of the human body, including the same endothelial ones. Any infection, viral and especially bacterial, affects blood clotting. The severity of the consequences, including thrombotic, depends on the characteristics of the pathogen and the patient's body. Moreover, for example, the owners of the first blood group have a lower risk of thrombosis, while in people with the second and third this risk is almost 30% higher. There are patients who naturally have thinner vascular endothelium. There are patients with genetically determined thrombophilia (a mutation that significantly increases the risk of severe thrombosis). And thus we come to three conclusions: 1) the cause of death is not always pneumonia or respiratory failure; 2) the cause of death is often thrombosis (heart attack, stroke, pulmonary embolism), as well as toxic damage to the heart and blood vessels (myocarditis, heart failure) - the presence of pneumonia is an aggravating factor; 3) the cause of death can be indirect or direct infection of other internal organs - the brain, kidneys, gastrointestinal tract, and the presence of chronic diseases and pneumonia is an aggravating factor. In some of the dead, the virus was found even in the cerebrospinal fluid. Therefore, the primary task of treatment for coronavirus is to destroy the infection, while the high temperature must be brought down and heparin preparations are prescribed to anticoagulate the blood and detoxify the body. Late initiation of anticoagulant therapy is an unfavorable factor that increases the risk of death. The most common and severe complication after coronavirus is thrombosis, especially with stenosis - vasoconstriction, and aneurysm ruptures (protrusion of the vessel wall with its thinning). It should be borne in mind that the vascular endothelium damaged by the virus recovers slowly, and the processes occurring in the body give an increased load on the entire circulatory system.

Thrombi, jelly-like clots of blood, cellular components and proteins, are formed due to damage to blood vessels, and even against the background of thickening (clotting disorders) of the blood. They grow over time

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and circulate in the blood without the patient suspecting or feeling that a blood clot is forming. These blood clots can block the arteries of the heart and brain. Therefore, a complication of coronavirus can be a heart attack or stroke. Pulmonary embolism (PE) is no less a threat to life. Moreover, often blood clots that block the vessels of the lungs initially form in the deep veins of the legs, after which they break off and move. Therefore, if there are symptoms such as:

- sudden unexplained shortness of breath
- chest pain,
- blood in cough discharge
- chest wheezing
- fainting, cold sweat, and dizziness,
- a sharp increase in heart rate at rest.

CONCLUSION

The patient is urgently recommended to do a CT scan of the pulmonary artery with contrast. Unfortunately, in most cases, such diagnosis is post-symptomatic, as is the diagnosis of stroke. CT angiography is performed using contrast. It allows you to see blood clots, neoplasms, aneurysms, pathologically narrowed sections of blood vessels. Sometimes ultrasound copes with this task, while the result of the examination is perfectly complemented by CT data. Ultrasound allows you to assess the dynamics of blood flow, to recognize its violation, while CT is the most reliable method of visual assessment of blood vessels. As soon as a large blood clot is detected in the bloodstream, the patient is prescribed special drugs (thrombolytics, statins) to reduce it or an invasive thrombolysis procedure. Therapy and prevention of diseases of the cardiovascular system after coronavirus should be comprehensive. Only a doctor can individually select the necessary diagnostic and therapeutic measures.

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