FEATURES OF THROMBOPHILIA IN COVID-19

[1]Kiyamova Laziza Botirovna, [2]Raxmatova Dilnoza Doniyarovna
211 group students of the medico-pedagogical faculty Samarkand State Medical Institute, Uzbekistan

---

ABSTRACT

From this article we see that why COVID-19 causes thrombosis, A thrombus is a mixture of platelets, erythrocytes and fibrin protein, which begin to accumulate on the wall of the damaged vessel. gradually it thickens and hardens, but in general it resembles a viscous substance, similar to jelly, it can be unambiguously stated that with this virus, from the very beginning, there is an activation of hemostasis, intravascular blood coagulation and thrombus formation in small-caliber vessels of vital organs.

Key words: COVID-19, thrombosis, thrombophilia, symptoms, virus, treatment,

Thrombophilia is a predisposition to the development of recurrent vascular thrombosis (mainly venous) of various localization. The disease is caused by genetic or acquired pathology of blood cells or defects in the blood coagulation system. Clinically, thrombophilia is manifested by multiple thrombosis of very different localization. Repeated thrombosis in the patient's history should lead the doctor to think about the presence of thrombophilia, which can be confirmed by blood and coagulation studies. Treatment of thrombophilia depends on its type. As a rule, thrombolytics, anticoagulants and disaggregates are used.

In some situations, it can come off and move to another part of the body. Such blood clots are called emboli.

Thrombosis symptoms:

- severe cough (often with blood);
- throbbing or cramping pain in the limbs;
- sharp chest pain; sudden shortness of breath;
- swelling, darkening of the skin and redness around the clot.

Why, in your opinion, did this feature of the course of the disease manifest itself immediately?

- I believe that all this manifested itself immediately, but was not adequately assessed by doctors initially: there has not yet been such a number of autopsies and widespread testing for hemostasiological markers. I must say that we have been studying this problem for a long time, almost from the very beginning of the epidemic. At the very beginning of April, we published a paper based on the first observations of our Chinese colleagues. The work was titled "COVID-19 and disseminated intravascular coagulation syndrome." It had an extremely wide resonance, since even then doctors began to understand the role of the blood coagulation system in the infectious process.

What is the mechanism of thrombus formation in covid-19 and does it differ from this process in other pathologies?

- This is a very difficult question. Today, it can be unambiguously stated that with this virus, from the very beginning, there is an activation of hemostasis, intravascular blood coagulation and thrombus formation in small-caliber vessels of vital organs. In this case, not only the lungs are damaged, but the blockade of microcirculation and its irreversible nature determine the outcome of the disease. But there are many people with blood clotting disorders. Now, during the epidemic, difficult times have come for them.

This is true. In our population there are people not only with obvious, but also with latent disorders of hemostasis, predisposing to thrombosis - genetic thrombophilia, antiphospholipid syndrome and a number of other diseases, accompanied by excessive activation of the hemostasis system; as well as people with a high readiness for a super-inflammatory response (congenital factors and a number of rheumatological and immune diseases). Now it is important for them to control their condition, and doctors do not forget about it.

Finally, COVID-19 is blood clotting. This is, in fact, the virus-mediated NET-os model, which characterizes the close relationship of biological processes such as inflammation and thrombus formation. Neutrophils and their extracellular neutrophil traps (NET) play a huge role in the development of so-called immune-thrombosis. This is one of the priority research areas today, which we are now developing together with our students, including foreign colleagues.

It so happened that, largely and thanks to our efforts (lectures and publications), most obstetricians today are aware that pregnancy is a state of so-called physiological hypercoagulability, and these patients are often prescribed anticoagulants during pregnancy. However, further research is required to judge the incidence of thrombosis in pregnant women with COVID-19.

In general, it must be said that most of the complications of pregnancy are either caused or combined with a high thrombogenic potential. Genetic factors of blood clotting, especially antiphospholipid syndrome, are risk factors for a huge number of pregnancy complications - intrauterine fetal death, IVF failures, intrauterine fetal growth retardation, and premature placental abruption, which leads to severe thrombohemorrhagic complications, and finally, thrombosis and thromboembolism.

Therefore, of course, one can expect that in the context of COVID-19, these complications could pose an even greater danger. After all, the virus can be a factor that activates blood clotting factors. Of course, generalizing studies are needed here, but even now our individual observations indicate that the risk of such complications is increasing.

The problem of risk factors is very important here. The fact is that, in addition to visible diseases such as diabetes or hypertension, there are invisible diseases that we often do not even suspect about. In recent years, the doctrine of genetic thrombophilia has become widespread. Globally, it is up to about 20 percent of people who carry some form of genetic thrombophilia.

You can live with this for a hundred years, but if an infection, an injury occurs, an operation is performed, the patient may die from thromboembolism, even if the operation is performed at the highest technical level. The reason for this is latent genetic thrombophilia, a mutation that makes the carrier at high risk for blood clots. One of the forms of thrombophilia - the so-called hyperhomo-cysteinemia, which can be either acquired or genetically determined, can also be an important factor in thrombosis, heart attacks, and strokes. And now there is evidence that hyperhomo-cysteinemia is aggravated by SARS-CoV2 infection.

Accordingly, the risk group includes all those who have elevated levels of homocysteine in the blood, but the person may not know about it. Therefore, we have now begun a large-scale study to identify these risk groups, to isolate various forms of thrombophilia in patients with COVID-19. Our goal is to find out if these people are at risk for developing severe complications of the new coronavirus infection.

In this regard, preventive (victimological) work should be carried out with a wide coverage of the population, for example, dividing it into groups according to age criterion: minors, young people, adults, elderly people.

Depending on the characteristic behavioral characteristics determined by the possible needs in conditions of self-isolation and potential criminal threats for these age categories, it is necessary to develop and implement individual measures of victimological prevention.

Another important feature of the implementation of these measures during the period of self-isolation will be the predominantly remote method of their implementation using the capabilities of information and telecommunication systems, as well as the media.

The substantive side of the implemented measures of victimological prevention is to inform citizens, first of all, about the possible spheres and methods of committing crimes, the characteristic behavioral characteristics of criminals used in the commission of criminal acts by means.

It seems that in order to implement effective victimological prevention in the context of the spread of coronavirus infection, it is necessary to timely predict the emergence of potential criminal threats to citizens

When a blood clot forms, normal blood flow is disrupted, which can lead to the development of dangerous complications. Thrombosis of cerebral vessels can cause ischemic stroke, thrombosis of coronary vessels causes the development of myocardial infarction, with thrombosis of the main arteries of the extremities, there is a threat of tissue necrosis, leading to amputation.

Thrombosis of the veins and arteries of the intestinal mesentery (mesenteric vessels) causes intestinal necrosis and the development of peritonitis due to perforation of the necrotic area of the intestine. Micro thrombosis of the placenta vessels can lead to malnutrition of the ovum and spontaneous abortion. With deep vein thrombosis of the lower extremities, there is a risk of blockage of the pulmonary artery by a detached thrombus (pulmonary embolism), and in the long-term period, the risk of developing chronic venous insufficiency increases. According to international statistics, the number of deep vein thrombosis of the lower extremities is constantly growing.

Currently, the frequency of treatment for phlebothrombosis is 150 cases per 100 thousand people per year. The risk of developing thrombophilia increases in the presence of a genetic predisposition (hereditary thrombophilia), certain acquired blood diseases (thrombocytosis, etitremia, antiphospholipid syndrome), atherosclerosis, malignant neoplasms, atrial fibrillation, varicose veins of the lower extremities, a number of autoimmune diseases, arterial hypertension. The likelihood of developing thrombophilia increases after a stroke or heart attack. The risk of blood clots increases after injuries and surgical interventions, with prolonged inactivity, with obesity, during pregnancy, when taking hormonal contraceptives. Smoking is a risk factor. Thrombophilia develops more often in patients of older age groups.

There are two main groups of thrombophilia.

1. Thrombophilia caused by a violation of the cellular composition and rheological properties of blood. The following conditions lead to the development of this group of thrombophilia:

- thickening of the blood, excess of corpuscular elements (thrombocythemia, erythrocytosis, polycythemia);
- change in the shape of red blood cells (sickle cell anemia);
- increased plasma viscosity (cryoglobulinemia, Waldenstrom's disease, myeloma).

2. Thrombophilia resulting from primary hemostasis disorders.

This group includes conditions caused by an excess or deficiency of coagulation factors. All types of blood disorders and defects in the blood coagulation system can be either congenital or acquired. The clinical manifestations of thrombophilia in the formation of a specific thrombus depend on the site of the lesion, the degree of circulatory disorders and the presence of concomitant (background) pathology. Thrombophilia is evidenced by multiple thrombosis of various localization, organ infarctions that develop in relatively young patients, thromboembolism in the pulmonary artery basin.

The diagnosis of thrombophilia is made on the basis of a tendency to recurrent formation of blood clots of different localization, family history (hereditary predisposition), characteristic laboratory test results. Patients with venous thrombosis usually seek help from a phlebologist.

The presence of repeated thrombosis in the anamnesis allows the doctor to suspect thrombophilia and to prescribe appropriate laboratory tests. Laboratory tests are carried out in two stages. First, the most common types and groups of thrombophilia are determined, then confirmatory highly specialized studies are performed.In the treatment of thrombophilia, it is often necessary to simultaneously solve two problems: to take measures to combat thrombosis and to treat the underlying disease that led to the recurrent formation of blood clots. thrombosis treatment consists in the appointment of thrombolites.

REFERENCE

1. Salwa Khanr and Joseph D. Dickerman. Hereditary thrombophilia/'/ Thrombosis Journal. — 2006.
2. Greaves M. What neurologists need to know about outside neurology thrombophilia // Pract. Neurol. — 2002.
3. Cattaneo M. Hyperhomocystelnemla, atherosclerosis and thrombosis // Thromb. Haemost. —1999.
4. Cobo-Sorlano R., Sanchez-Ramon S., Aparicio M. J. et al. Anti-phosphollpld antibodies and retinal trombosis in patients without risk factors: a prospective case-control study II Am. J. Ophthalmol. — 1999.
5. Cortlnovls A., CrippaA., Crippa M. Fattore Vlll/Ag (von Wlllebrand) e protelna C Deformablllta e aggregablllta erltrocltarla. Parametrl dl rlschlo di occlusione venosa retlnlca II Mlnerva-Med. — 1994.