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PATHOANATOMICAL BASIS OF BLOOD COAGULATION SYSTEM DISORDERS IN COVID-19 INFECTION

Ganiyev Sardor Saminjonovich
Fergana medical institute of public health

Abstract

COVID-19 infection manifests not only as respiratory system damage but also as a systemic disease with profound hemostasiological changes in the body. As a result of SARS-CoV-2 virus infection, acute activation of the blood coagulation system, endothelial dysfunction, microthrombosis, disseminated intravascular coagulation (DIC syndrome), and multiple organ failure develop. This article extensively covers the pathoanatomical basis of blood coagulation system disorders in COVID-19 infection[1,2,3], morphological features, microcirculatory changes, mechanisms of thrombotic complications, and their clinical significance.

Keywords

COVID-19, SARS-CoV-2, hemostasis, thrombosis, microthrombosis, disseminated intravascular coagulation, endothelial dysfunction, pathoanatomical changes, multiple organ failure.

INTRODUCTION

The COVID-19 pandemic has placed an unprecedented burden on healthcare systems worldwide. It has been established that the main cause of death in severe and critically severe forms of the disease is associated not only with respiratory failure but also with thromboembolic complications. Autopsy and pathoanatomical studies have shown that disorders of the blood coagulation system play a leading role in COVID-19. Therefore, an in-depth study of the pathogenesis and morphological basis of the disease, especially the analysis of changes in the hemostasis system, is of great importance for early detection of the disease, prevention of complications, and improvement of treatment strategies[4,5,6].

Disorders of the blood coagulation system in COVID-19 infection develop based on complex and multifactorial pathogenetic mechanisms. When the SARS-CoV-2 virus enters the body, it damages endothelial cells through ACE2 receptors. While the endothelium under normal conditions has anticoagulant, vasodilator, and anti-inflammatory properties, its protective function is disrupted by the virus. As a result, a procoagulant state is formed, creating conditions for the development of thrombosis.

Disorders of the blood coagulation system in COVID-19 infection develop based on complex, multi-stage, and interconnected pathogenetic mechanisms. The SARS-CoV-2 virus enters the body through the respiratory tract and binds to angiotensin-converting enzyme-2 (ACE2) receptors located on the cell surface. These receptors are widespread not only in alveolar epithelial cells but also in vascular endothelial cells throughout the body, serving as the primary morphological substrate determining the virus's systemic effect. As a result of the direct cytopathic effect of the virus on endothelial cells, the integrity of the endothelium is disrupted, cells swell and become vacuolated, and in some cases, apoptotic and necrotic changes develop[7,8,9].

Under normal conditions, the vascular endothelium is the main regulatory structure that maintains hemostasis balance in the body, possessing anticoagulant, antithrombotic, and fibrinolytic properties. Endothelial cells produce biologically active substances such as nitric oxide (NO), prostacyclin, thrombomodulin, heparan sulfate, and tissue plasminogen activator[10,11]. These substances inhibit platelet aggregation, dilate blood vessels, and prevent intravascular thrombus formation. However, during COVID-19 infection, as a result of viral infection of endothelial cells, these protective mechanisms are severely weakened or completely disrupted.

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In the context of viral endotheliitis development, the expression of tissue factor, which has procoagulant properties, sharply increases on the endothelial surface. Simultaneously, the activity of thrombomodulin and the protein C system decreases, leading to insufficient functioning of physiological anticoagulant mechanisms[12,13]. Consequently, the blood coagulation system is activated, thrombin formation intensifies, and fibrin polymerization accelerates. Pathoanatomical examinations reveal intravascular thrombi rich in fibrin threads, platelet aggregates, and erythrocytes.

Endothelial dysfunction in COVID-19 infection is closely linked to the inflammatory process, and the cytokine storm further deepens this process. Pro-inflammatory cytokines, such as interleukin-6, interleukin-1β, and tumor necrosis factor-α, activate the coagulation cascade and increase fibrinogen synthesis. Concurrently, the fibrinolysis process is inhibited, which slows down the breakdown of thrombi[14,15,16]. From a pathoanatomical perspective, this condition manifests as the formation of stable, dense fibrinous thrombi within the blood vessels.

At the microcirculatory bed level, the most significant morphological feature characteristic of COVID-19 is widespread microthrombosis in capillaries and venules. These microthrombi are particularly prevalent in the lung alveolar capillaries, leading to gas exchange disturbances, alveolar hypoxia, and the development of respiratory distress syndrome. In lung tissue, thickening of interalveolar septa, capillary engorgement with blood, fibrin exudates, and hemorrhagic foci are observed, which pathoanatomically characterize the thrombotic angiopathy specific to COVID-19[17,18].

Endothelial damage is observed not only in the lungs but also in the vessels of the heart, kidneys, liver, and central nervous system. In the heart, thrombosis of myocardial capillaries, interstitial edema, and small hemorrhages lead to impaired myocardial contractile function. In the kidneys, thrombosis and endothelial swelling of glomerular capillaries cause disruption of the filtration process, leading to the development of acute renal failure. Thrombosis and stasis in the liver sinusoids result in hypoxic damage to hepatocytes.

Thus, in SARS-CoV-2 infection, a stable hypercoagulable state is formed in the body as a result of the mutually reinforcing effects of endothelial cell damage, inflammation, and the hemostasis system[16,20]. This condition, pathoanatomically manifested by intravascular thrombosis, microcirculatory disorders, and ischemic and hemorrhagic changes in parenchymal organs, is considered the main morphological cause of severe COVID-19 progression and high mortality rates.

Pathoanatomical examinations reveal widespread alveolar capillary microthromboses in the lungs of patients who died from COVID-19. These microthrombi are composed of fibrin, platelets, and erythrocytes, leading to disruption of alveolar gas exchange. This condition has led to the concept of "COVID-19-specific thrombotic microangiopathy." Thromboembolic changes in the pulmonary arteries and veins are frequently observed, resulting in the development of acute cor pulmonale and severe hypoxia.

In cardiac pathoanatomy, microthromboses, interstitial edema, hemorrhages, and necrotic foci are detected in the myocardial capillaries. These findings indicate the important role of hemostatic disorders in the pathogenesis of COVID-19-associated myocarditis and acute heart failure. Endothelial damage to the coronary vessels accelerates thrombus formation and can lead to the development of acute coronary syndrome.

In the kidneys, thrombosis of glomerular capillaries, fibrinoid necrosis, endothelial edema, and hemorrhages are observed. These changes manifest as acute renal failure, which significantly increases the risk of death in COVID-19. The detection of sinusoidal thromboses in the liver tissue, necrosis around the central vein, and signs of steatosis are also directly related to disorders of the hemostasis system.

Microthromboses, hemorrhagic infarctions, and ischemic foci are detected in the vessels of the central nervous system. This pathoanatomically explains the high incidence of strokes, impaired consciousness, and neurological complications in patients with COVID-19.

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Disseminated intravascular coagulation (DIC syndrome) is common in severe forms of COVID-19, occurring simultaneously with thrombosis and hemorrhagic syndrome. Pathoanatomically, intravascular fibrin threads, thrombi, and multiple hemorrhages in parenchymal organs are detected. DIC syndrome is one of the main causes of multiple organ failure.

Increased platelet activity, complement system activation, and cytokine storm in COVID-19 further stimulate the blood coagulation system. IL-6, TNF-α, and other pro-inflammatory cytokines enhance the synthesis of coagulation factors and suppress anticoagulant mechanisms. As a result, a "hypercoagulable state" develops in the body.

Thus, disorders of the blood coagulation system in COVID-19 infection are considered the central link in the pathogenesis of the disease, which morphologically manifest as thrombosis, microcirculatory disturbances, necrosis, and hemorrhages in multiple organs. These pathoanatomical changes explain the severe course of the disease and high mortality rates.

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