PATHOGENESIS AND CLINICAL-MORPHOLOGICAL FEATURES OF DVC SYNDROME

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Relevance. Disseminated intravascular coagulation (DIC) is a rapidly progressing and life-threatening condition characterized by a high mortality rate and the development of multiple organ failure. It commonly occurs in severe clinical states such as sepsis, trauma, burns, shock, and obstetric complications. Studying DIC from a pathological and anatomical perspective is essential for a deeper understanding of its mechanisms, as well as for improving early diagnosis and treatment strategies.

Objective. To investigate the pathogenesis, clinical presentation, and morphological features of DIC syndrome, and to enhance early diagnostic approaches and treatment strategies based on clinical and laboratory findings.

Materials and Methods. The pathogenesis of DIC is primarily associated with excessive thrombin generation, which leads to widespread activation of the coagulation cascade throughout the vascular system. As a result, fibrin formation increases, causing the development of microthrombi within the microcirculation, particularly in capillaries. These microthrombi impair tissue perfusion and contribute to organ dysfunction.

Simultaneously, the excessive consumption of platelets and coagulation factors leads to a condition known as consumption coagulopathy, which predisposes patients to bleeding complications.

DIC may develop in response to various triggers, including severe infections (sepsis), trauma, toxic conditions, hemodynamic disturbances, and obstetric complications.

Clinically, DIC typically progresses through two main phases:

Thrombotic phase – characterized by microthrombus formation leading to tissue ischemia and organ dysfunction.

Hemorrhagic phase – caused by depletion of clotting factors, resulting in widespread bleeding.

Patients may present with petechiae, hemorrhages, internal bleeding, acute respiratory failure, and renal insufficiency. Acute DIC is often associated with hypotension, shock, and multiple organ dysfunction syndrome, whereas chronic or subclinical forms may only be detected through laboratory findings.

Morphologically, DIC is characterized by fibrin thrombi in small and medium-sized vessels, leading to ischemic necrosis, as well as degenerative and dystrophic changes in affected organs.

Conclusion. DIC syndrome is a complex pathological process resulting from increased thrombin generation, impaired anticoagulant mechanisms, and imbalance in the fibrinolytic

system. Clinically, it manifests as bleeding, shock, and multiple organ dysfunction. Morphological findings include microthrombosis, ischemic necrosis, and cellular degeneration. Early diagnosis and timely treatment are crucial for improving patient outcomes and survival.