Federal State Budgetary Educational Institution of Higher Education

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Guidelines for conducting a practical lesson with 5th year students of the Faculty of Medicine on the topic:

HEMORRHAGIC DIATHESIS. DIC

Vladikavkaz 2022

Guidelines for conducting a practical lesson with 6th year students of the Faculty of Medicine on the topic:

DIC SYNDROME IN THE CLINIC OF INTERNAL DISEASES. DIAGNOSIS, TREATMENT AND PREVENTION OF THROMBOSIS.

PURPOSE OF THE LESSON: in the process of clinical analysis of the patient to increase the level (quality) of knowledge and skills of students in the diagnosis (differential diagnosis) of DIC - syndrome.

Students should be able to: Make a differential diagnosis of DIC-syndrome, taking into account the etiology, pathogenesis, clinical course. Assign timely pathogenetic therapy, taking into account the stage of the disease. Assign preventive measures for a group of patients with a high risk of developing DIC - syndrome.

Motivation of the relevance of the topic. DIC-syndrome is a non-specific pathology of hemostasis, which is based on disseminated blood coagulation in the circulation with the formation of many microclots and aggregates of blood cells, which causes blockade of microcirculation and deep dystrophic changes in organs, followed by the development of hypocoagulation and consumption thrombocytopenia and often bleeding.

Disseminated intravascular coagulation is a general pathological reaction (internal accident) of the body to various factors, which occurs much more often than it is diagnosed. Moreover, the underestimation of this syndrome in the pathogenesis of many diseases leads to the appointment of therapy that is adequate to the nosological form, but not adequate to DIC. In turn, this leads to aggravation of intravascular coagulation and determines the lethal outcome.

Determining the level of students' preparation. Second level of knowledge:

control methods - written survey (20 min). Students should know the main points of etiology, pathogenesis, classification and clinical manifestations of DIC; students should be able to possess propaedeutic skills.

Report of student curators in the Chamber. When reporting a patient, students should pay special attention to the fact that:

The main causes of the development of DIC - syndrome:

1. Infections and septic conditions (bacteremia, viremia).

2. All types of shock (anaphylactic, traumatic, burn, septic, with prolonged crush syndrome, etc.).

3. Acute intravascular hemolysis (transfusion of incompatible blood, hemolytic crises, hemolytic anemia, various thrombocytopathies).

4. Malignant tumors, leukemias.

5. Massive tissue damage (injury, including traumatic brain injury, burns, cruch-syndrome); extensive surgical interventions, including the use of a heart-lung machine.

6. Obstetric - gynecological pathology.

7. Cardiovascular diseases.

8. Acute and subacute inflammatory processes (in the lungs, liver, pancreas, kidneys, etc.).

9. Systemic connective tissue diseases, systemic vasculitis.

10. Acute and chronic immunoinflammatory diseases of the kidneys (glomerulonephritis).

11. Poisoning by hemocoagulating snake venoms.

12. Treatment with drugs that cause platelet aggregation, increase blood clotting and reduce its anticoagulant and fibrinolytic mechanisms (α-adrenergic stimulants, glucocorticoids, estrogen-progestive drugs, ε-aminoparonic acid), etc.

13. Other causes: diabetes mellitus, radiation sickness, hyperthermia, etc.

Pathogenesis. Under the influence of etiological factors, blood coagulation is activated, thrombin formation, widespread fibrin deposition and thrombus formation in the microvasculature, which causes an increased consumption of the main anticoagulants (antithrombin III, proteins C, S) with their subsequent deficiency.

Widespread intravascular coagulation leads to intensive consumption of coagulation factors and platelets (coagulopathy and consumption thrombocytopenia), activation of the fibrinolysis and proteolysis system with the development of hemorrhagic syndrome.

1. Tissue damage and the entry of procoagulants into the bloodstream (with obstetric and gynecological pathology, malignant tumors, leukemia).

The initiator of coagulation processes is tissue thromboplastin (factor III). According to the external and internal mechanism of coagulation under the influence of thromboplastin, factor Xa is activated.

Tissue thromboplastin enters the bloodstream from damaged tissues in obstetric and gynecological pathology (the placenta, amniotic fluid are rich in thromboplastin), in malignant tumors, and leukemia.

2. Hyperproduction of procoagulants by the system of mononuclear phagocytes.

In severe infections, sepsis, malignant neoplasms, monocytes and macrophages release thromboplasty and procoagulant substances into the blood.

3. Damage to the endothelium and activation of the internal pathway of activation of blood coagulation.

With widespread atherosclerosis of the arteries, systemic vasculitis, prosthetics of blood vessels and heart valves in the pathogenesis of DIC, the main role is played by endothelial damage, exposure of collagen and activation of the internal pathway for activating blood coagulation.

4. Aggregation of blood cells.

An important link in the pathogenesis is an increase in the adhesive-aggregation function of platelets (as a result, blood clots are formed) and changes in erythrocytes. There is aggregation of erythrocytes, the formation of "sludges" (clumps of Er, shrouded in fibrin threads), fragmentation, deformation of Er and intravascular hemolysis. Thromboplastin is also released from the destroyed ER.

The above mechanisms are reduced to the formation of active thromboplastin and refer to the I phase of the development of DIC - syndrome.

5. Intensive formation of thrombin and development of disseminated intravascular coagulation.

Phase II - the transition of prothrombin to thrombin under the influence of active thromboplastin and the participation of calcium ions.

Phase III - the formation of fibrin - polymer. Under the influence of thrombin formed in large quantities, fibrinogen is hydrolyzed to insoluble filaments of fibrin, a polymer, which subsequently precipitate into stable fibrin clots. Microthrombi are formed, causing microcirculation obstruction and multiple organ failure.

The development of disseminated thrombus formation leads to the activation of protective anticoagulant mechanisms with their rapid depletion.

6. Decreased function of anticoagulant and antiaggregatory mechanisms.

In the hypercoagulable phase, the anticoagulant system is activated in response to excessive thrombin formation and thrombus formation, however, the depletion of the anticoagulant system quickly sets in - the level of antithrombin III, proteins C, S drops in the blood. And the depletion of the anticoagulant system contributes to further thrombus formation.

7. Development of coagulopathy and consumption thrombocytopenia.

Excessive activation of hemostasis in DIC, intensive formation of microthrombi and platelet aggregates leads to the depletion of coagulation factors and a decrease in the number of platelets due to their intensive consumption in the process of pathological coagulation. Coagulopathy and consumption thrombocytopenia develop. This is manifested by a sharp decrease in the amount of prothrombin, fibrinogen, Tr, coagulation factors V, VIII, XIII in the blood. As a result of the exhaustion of the coagulation system, the hemorrhagic phase of DIC develops.

8. Activation of fibrinolytic and proteolytic systems also plays a role in the development of hypocoagulation syndrome.

Classification

I. Etiological variant (septic, obstetric, traumatic, immunocomplex, etc.).

II. Flow:

1) lightning fast (within several tens of minutes);

2) acute (within several hours, up to a day);

3) subacute (from several days to 3 weeks);

4) chronic (runs for months, years);

5) recurrent;

6) latent.

III. Phases (stages):

1) the phase of hypercoagulation and hyperaggregation of platelets;

2) a transitional phase with multidirectional shifts (a tendency to hypocoagulation according to some tests and hypercoagulation according to others);

3) phase of hypocoagulation and activation of fibrinolysis (hemorrhagic);

4) recovery (with an unfavorable outcome - severe complications and death).

The allocation of phases depending on the state of the hemostasis system is possible only in the acute course of DIC.

The DIC clinic is a reflection of thrombotic and ischemic damage to organs and tissues, on the one hand, and hemorrhagic syndrome, on the other hand.

In addition to the symptoms of the underlying disease, the clinic consists of the following symptoms:

1. Hemocoagulation shock: caused by impaired microcirculation in organs and tissues, the development of ischemia in them, the entry into the blood of a large number of toxic substances, proteolysis products, activation of the kinin system. Clinical symptoms in hypercoagulable states may be combined with manifestations of thrombosis of venous and arterial vessels of various localizations.

2. Violations of hemostasis (from hypercoagulation to deep hypocoagulation). Pronounced hypercoagulability (in the absence of a technical error during blood sampling) is evidenced by the fact that the blood immediately coagulates in a syringe or test tube. Positive procoagulation tests (ethanol, protamine sulfate), fragmentation of red blood cells in smears, etc. confirm DIC-syndrome.

In the II (intermediate) phase, the clinical symptoms characteristic of the hypercoagulable phase persist and dominate, but signs of hypocoagulation appear in laboratory tests.

3. Hemorrhagic syndrome develops in phase III. Petechiae, extensive hemorrhages, ecchymosis, hematomas appear on the skin. Both local bleeding (hemopericardium, hemorrhages in the pleural cavity, peritoneum, intracerebral and subarachnoid spaces) and generalized hemorrhagic syndrome are characteristic.

4. Violations of microcirculation in the organs, their dysfunction and dystrophy (“shock - organs”) - damage to the lungs and the development of acute respiratory failure and distress syndrome, kidneys with the development of acute renal failure, hepatorenal syndrome, damage to the liver, stomach, intestines.

5. Violations of cerebral circulation.

6. Damage to the adrenal glands (in the form of acute adrenal insufficiency), the pituitary gland.

Differential diagnosis of DIC-syndrome is carried out with:

- massive liver necrosis

- vitamin K deficiency

- thrombocytopenic purpura

- hemolytic-uremic syndrome.

Diagnostics.

Diagnosis of DIC-syndrome should be carried out on the basis of the clinical picture of the disease and taking into account

changes in the totality of coagulogram parameters.

Sharply positive cuff test - the number of petechiae over 30, the size of hemorrhages exceeds 1 mm.

Informative from laboratory studies:

1) KLA: thrombocytopenia, anemia, leukocytosis, shozocytosis.

2) Coagulogram:

 ↑ APTT

 ↑ prothrombin time

 ↓ fibrinogen

 ↑ content of fibrin breakdown products

 prolong bleeding time

 ↓ antithrombin III

 ↓ protein C content

 ↓ factors V, VIII (possibly ↑)

 ↓ factors X, XIII.

3) Blood biochemistry:

 ↑ LDH

 ↑ urea

 hemoglobinemia

4) OAM: hematuria.

5) Positive Gregersen's test.

In all stages of DIC, the determination of markers of disseminated intravascular coagulation in the blood is of great importance:

- an increase in the amount of fibrin degradation products (fibrinogen),

- increase in the number of D-D - dimers (reflecting the breakdown of stabilized fibrin),

- increase in the amount of soluble fibrin-monomer complexes (SFMK).

- decreased activity of antithrombin-III.

RFMK are determined using paracoagulation tests. These tests are based on the phenomenon of paracoagulation - the precipitation of fibrinogen pool stratification products - RFMK, formed during the proteolytic degradation of fibrinogen molecules - fibrin under the action of thrombin and plasmin. One of the simple methods for determining RFMK and fibrinogen degradation products is the ethanol test.

The following table presents the most characteristic signs of the hypercoagulable and hypocoagulable phases of acute DIC - syndrome.

Conducting classes in a thematic classroom. Analysis of the features of the etiology, pathogenesis and clinic of DIC - syndrome, diagnosis and prevention of thrombosis.

The final part of the lesson: control of the acquired knowledge - solving situational problems without possible options for correct answers.