**Tests on the topic: "Disseminated intravascular coagulation syndrome, diagnosis, differential diagnosis"**

# Antithrombin III is:
= primary anticoagulant
secondary anticoagulant
platelet factor
fibrinolytic agent
plasma coagulation factor

# The central place in the pathogenesis of DIC is:
= hyperthrombinemia
thrombocytopenia
thrombocytopathy
increased antithrombin III

# To assess the effectiveness of antithrombotic therapy with warfarin, determine
= bleeding time
international normalized attitude
thrombin time

# Violations of platelet-vascular hemostasis can be detected
in determining prothrombin time
= when determining bleeding time
in determining thrombin time

# Duration of development of fulminant form of DIC - syndrome
= several tens of minutes
several hours
a few days
few weeks

# The following mechanisms are the cornerstone of the development of DIC - syndrome:
coagulation system activation
decrease in antithrombotic potential of endotheliocytes
severe secondary endogenous intoxication with products of proteoliosis and tissue destruction
= all answers are correct

# The main initiator of the blood coagulation process is most often:
= tissue thromboplastin
antithrombin III
plasminogen

# In case of DIC, use is contraindicated:
heparin
= epsilon-aminocaproic acid
transfusion of freshly frozen plasma

# The objectives of the hemostatic thrombopenia therapy program are:
immunocorrective therapy
= elimination of platelet deficiency, normalization of the vascular component of hemostasis, increased adhesive and aggregation properties of platelets
von Willebrand factor deficiency correction
decrease in fibrinolytic blood activity

# For von Willebrand disease is characteristic:
recessive type of inheritance, spotty-petechial type of bleeding
= dominant type of inheritance, mixed type of bleeding
recessive type of inheritance, recurrent hemarthrosis
dominant type of inheritance, spotty-petechial type of bleeding

# The main objectives of the hemostatic therapy program for hemorrhagic vasculitis:
increase in activated plasma recalcification time
= immunocorrective therapy, elimination of capillary permeability factor deficiency
von Willebrand factor deficiency correction
prevention of DIC

# In severe hemophilia A, the level of factor VIII in the patient is
= 0-1%
0-3%
3.1-5%
5.1-10%
10-15%

# In moderate forms of hemophilia A, the level of factor VIII in a patient is:
0-1%
0-3%
= 3.1-5%
5.1-10%
10-15%

# With a mild form of hemophilia A, the level of factor VIII in a patient is:
0-1%
0-3%
3.1-5%
= 5.1-10%
10-15%

# Vascular platelet hemostasis characterizes the test:
blood coagulation time
= Duke bleeding duration
thrombin time
euglobulin clot lysis
amount of fibrinogen

# What method is used to judge the resistance of microvessels:
fibrinogen concentration determination
determination of the activity of factor VIII
= cuff test
fibrinolytic activity test
blood coagulation time

# Antifibrinolytic drugs are all but:
amben
pantripin
= cryoprecipitate
contrikal
proudox

# Diagnosis of hemophilia A is based on:
peripheral blood test
= anamnestic data, clinical manifestations, coagulogram analysis
myelogram
all of the above is true

# For hemophilia A in the coagulogram, changes will be characteristic:
= increase in blood coagulation time, increase in activated plasma recalcification time, degree of thrombosis I-III;
decrease in blood coagulation time, decrease in fibrinogen level, degree of thrombotest I-III;
increase in blood coagulation time, decrease in activated plasma recalcification time, degree of thrombosis IV-V;
decrease in blood coagulation time, decrease in activated plasma recalcification time, degree of thrombosis IV-V

# Coagulogram allows you to evaluate:
= coagulation mechanism of hemostasis
vascular wall resistance
platelet functional activity
vascular platelet mechanism of hemostasis
all of the above is true

# Treatment of hemophilia A:
= transfusion of FFP, cryoprecipitate or factor VIII
transfusion of cryoprecipitate or factor VII
platelet transfusion or factor VIII
native concentrated plasma transfusion and factor IX

# Cryoprecipitate will be an effective hemostatic agent:
with hemophilia B
= von Willebrand disease
with thrombocytopenia
with hemophilia C
with antifibrinogenemia

# Ascorbic acid as a hemostatic agent is most effective for violations:
coagulation mechanism of hemostasis
= vascular component of hemostasis
platelet mechanism of hemostasis
Disseminated Intravascular Coagulation Syndrome - II
with all hemorrhagic diathesis

# Introduction of vikasol will be an effective hemostatic agent:
with hemophilic bleeding
= with complex deficiency of K-vitamin-dependent factors
with DIC
with local fibrinolysis
with idiopathic thrombocytopenic purpura

# Solutions of calcium chloride should be used as a hemostatic agent for violations:
platelet and coagulation component of hemostasis
= platelet and vascular component of hemostasis
coagulation component of hemostasis
fibrinolytic bleeding
with all violations of hemostasis

# What is the basis for the diagnosis of hemophilia B:
on the analysis of peripheral blood and myelogram
= history, clinical manifestations, coagulogram analysis
on myelogram and biochemical analysis of blood

# Treatment of hemophilia B:
albumin transfusion
= transfusion of freshly frozen plasma, factor IX
platelet transfusion and factor XI
red blood cell transfusion and factor VIII

# Transfusion therapy during operations in patients with hemophilia B:
= transfusion of freshly frozen plasma, transfusion of factor IX
platelet transfusion, factor XI transfusion
red blood cell transfusion, factor IX transfusion
albumin transfusion, factor VIII transfusion

# The von Willebrand factor forms a complex with the coagulation factor:
X
V
XII
= VIII
VII

# Fibrinogen content is normal:
2-4 mmol / l
2-4 mg%
= 2-4 g / l
200-300 mg%
2-3 g / l

# The clinical type of bleeding for platelet component disorders of hemostasis will be:
hematoma
= spotty-petechial
vasculitis purple
mixed
angimatous

# In which disease the number of megakaricytes in the bone marrow is increased:
aplastic anemia
multiple myeloma
= thrombocytopenic purpura
chronic lymphocytic leukemia
megaloblastic anemia

# For which disease is thrombocytopenia detected:
von Willebrand disease
Hageman's disease
= acute leukemia
Iron-deficiency anemia
hemorrhagic fever with renal syndrome

# The most common cause of hemorrhagic diathesis is:
hereditary coagulopathies
disseminated intravascular coagulation syndrome
= thrombocytopenia, thrombocytopathy
disovarial purpura
hemorrhagic fever

# To detect thrombocytopenia, it is necessary to examine:
platelet adhesion-aggregation function
= platelet count
fibrinogen
thrombin time
beta thrombomodulin

# To identify thrombocytopathy, it is necessary to examine:
platelet aggregation function
platelet adhesion function
platelet factor III
bleeding time
= all of the above

# What determines the severity of the clinical course of immune thrombocytopenic purpura:
peripheral blood hemoglobin content
white blood cell count
degree of hemorrhagic syndrome
platelet count in peripheral blood

# What is the basis for the diagnosis of immune thrombocytopenic purpura:
on a biochemical blood test and a coagulogram
= on analysis of peripheral blood, myelogram, clinical picture
hemoglobin content in peripheral blood, coagulogram

# The main diagnostic sign of thrombocytopenic purpura:
mixed type bleeding
= petechial spotted type of bleeding
vasculitis-purple type of bleeding
positive cuff test
negative can test

# The main diagnostic sign of thrombocytopenic purpura:
= platelet count less than 100 \* 10 9 / l and an increase in the duration of bleeding;
platelet count less than 200 \* 10 9 / l and an increase in plasma recalcification time;
platelet count less than 150 \* 10 9 / l and increased blood coagulation time
red blood cell count and fibrinogen level

# The main types of treatment for immune thrombocytopenic purpura:
platelet transfusion ~ corticosteroids
immunosuppressants
immunotherapy
splenectomy
= all of the above

# With idiopathic thrombocytopenic purpura, the treatment program will be ineffective:
prednisone
= vikasol
splenectomy
cyclophosphamide

# Transfusion therapy for advanced hemorrhagic syndrome in patients with immune thrombocytopenic purpura:
= transfusion of freshly frozen plasma and platelets
platelet and albumin transfusion
transfusion of albumin and potassium chloride
red blood cell and platelet transfusion

# In case of massive blood loss during surgery in patients with immune thrombocytopenic purpura, red blood cells should be transfused:
individually selected
washed red blood cells
thawed red blood cells
= standard red blood cells

# In the absence of platelet growth after splenectomy in patients with immune thrombocytopenic purpura with threatening hemorrhagic syndrome, the following are used:
= transfusion of freshly frozen plasma, platelets + hormone therapy
transfusion of cryoprecipitate, red blood cells + hormone therapy
red blood cell transfusion, cryoprecipitate + cytostatics

# Transfusion of freshly frozen plasma will be an effective hemostatic agent in the treatment of:
idiopathic thrombocytopenic purpura
= von Willebrand disease
Glanzmann thrombasthenia
transimmune thrombocytopenia

# Hemophilia A and B are manifested by the following clinical type of bleeding, according to ZS S. Barkagan:
bruise
= hematoma
mixed
vasculitis purpurea
angiomatous

# With hemophilia A, there is a hereditary synthesis defect and factor deficiency:
V
= VIII
IX
X
XI

# With hemophilia B, there is a hereditary synthesis defect and factor deficiency:
V
VIII
= IX
X
XI

# With angiohemophilia, there is often a factor deficiency
V
VIII
IX
X
= von Willebrand factor

# With hemophilia C there is a hereditary factor deficiency
V
VIII
IX
X
= XI

# What are the main pathogenetic mechanisms of the development of immune thrombocytopenic purpura:
= increased destruction of platelets in peripheral blood and megakaryocytes in the bone marrow by the antigen-antibody complex
platelet destruction due to a decrease in the level of thrombopoietins
platelet destruction due to impaired glycolysis enzyme activity
platelet destruction due to a lack of coagulation factors in plasma

# What are the main clinical symptoms of immune thrombocytopenic purpura:
weakness
fever
= hemorrhages on the skin and bleeding from the mucous membranes
hemarthrosis

# Hemorrhagic syndrome with immune thrombocytopenic purpura manifests itself in the form:
petechiae located symmetrically on the limbs
= skin hemorrhages and nosebleeds
hematoma

# Typical changes in the hemogram with immune thrombocytopenic purpura:
monocytosis
leukopenia
leukocytosis
= thrombocytopenia
pancytopenia

# Additional laboratory tests confirm immune thrombocytopenic purpura:
= positive Coombs test
normal clotting time
the duration of bleeding is slowed down
increased blood clot retraction

# Typical changes in the myelogram for immune thrombocytopenic purpura: erythroid inhibition
= megakaryocytic germ hyperplasia with no lacing forms
plasma cell metaplasia
blast metaplasia
bone marrow emptying

# Immune thrombocytopenic purpura must be differentiated from:
= acute leukemia
capillarotoxicosis
erythremia
hemophilia
aplastic anemia

# The main treatment methods for immune thrombocytopenic purpura:
= hormone therapy + splenectomy + immunosuppressants
polychemotherapy + hormone therapy + erythromass transfusion
radiation therapy + polychemotherapy + hormone therapy

# Violations of platelet-vascular hemostasis can be detected:
when determining coagulation time
= when determining bleeding time
in determining thrombin time
when determining plasminthogen
in determining fibripolysis

# For hemorrhagic vasculitis is characteristic:
hematoma bleeding
= vasculitis purple appearance of bleeding
coagulation time extension
decrease in prothrombin thrombocytopenia index

# Drugs that can cause thrombocytopathy include:
= acetylsalicylic acid
vikasol
cordaron
veroshpiron

# For the diagnosis of hemophilia is used:
= determination of blood coagulation time
determination of bleeding time
determination of plasminogen

# Disseminated intravascular coagulation syndrome may occur with:
= generalized infections
epilepsy
intracellular hemolysis

# For the treatment of disseminated intravascular coagulation syndrome use:
= freshly frozen plasma
dry plasma
cryoprecipitate
erythromass

# If the patient has teleapgioectasia, nosebleeds, and studies of the hemostatic system does not reveal significant violations, you should think about:
hemophilia
= Randu-Oslev disease
von Willebrant disease
Verloff disease

# The most common in the clinic are immune thrombocytopenia:
isomunous, associated with the formation of antibodies during blood transfusions or pregnancy;
immune, associated with a violation of the antigenic structure of the platelet or with the advent of a new antigen;
= autoimmune, in which antibodies are produced against your own unchanged antigen

# Violation of platelet-vascular hemostasis can be detected:
when determining coagulation time
= when determining the duration of bleeding
when determining plasminogen
in determining fibrinolysis

# Thrombocytopenia refers to:
disorders of secondary hemostasis.
= violations of primary hemostasis.
thrombophilia.
thrombasthenia.
coagulopathy.

# Violations of secondary hemostasis are, except:
deficiency of plasma procoagulants.
hyperheparinemia.
= thrombophilia.
fibrinolysis syndrome.
coagulopathy.

# The clinical type of bleeding for platelet component disorders of hemostasis will be:
hematoma.
= spotty-petechial.
vasculitis purple.

mixed.
angiomatous.