**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.