SPECIAL PATHOPHYSIOLOGY

ANEMIAS

1. Anemia is defined as:
    1. Reduced count of erythrocytes, leukocytes, reticulocytes in given amount of blood
    2. Reduced count of erythrocytes, trombocytes, leukocytes in given amount of blood
    3. Reduced count of erythrocytes, haemoglobin in given amount of blood
    4. Reduced count of erythrocytes, haemoglobin and hypovolemia
    5. Olygocythemic hypervolemia

2. Main pathogenetic factor in all anemic syndromes is:
    1. Reduced oxygen partial pressure in arterial blood
    2. Reduced oxygen concentration in blood
    3. Impaired oxygen metabolism in the tissues
    4. Non-hypoxically decreased oxygen demand
    5. Increased oxygen consumption, resulting from genetic abnormality

3. Anemia is:
    1. Reduced oxygen transporting capacity of the blood
    2. Reduced oxygen transporting capacity of the blood
    3. Haemotoxic hypoxia of any kind
    4. 1,2
    5. 1,2,3

4. The pathogenetic classification of anemias includes:
    1. Acute and chronic haemorrhagic anemias, acute and chronic haemolytic anemias
    2. Haemorrhagic anemias, anemias due to impaired erythropoesis and haemolytic anemias
    3. Hypo- and Aplastic anemias, sideroachrestic anemias
    4. Hyper-and hyporegenerative anemias
    5. 1,3,4

5. In the first hours after haemorrhage usually there is:

1. No laboratory data, indicating anemia are present
2. Significantly decreasedlevelsof erythrocytes and haemoglobin
3. Marked reticulocytosis
4. Compensatory polyglobulia
5. Increased megalocyte number

6. At the 4th-5th day after significant haemorrhage the following findings may be observed:

1. Olygocythaemic normovolemia
2. Normocytic hypovolemia
3. Normocytic normovolemia
4. Compensatory hypervolemia
5. Compensatory polyglobulia

7. Which of the following anemias are due to impaired erythropoesis:

1. Iron-deficient anemia, B12-folic acid-deficient anemia
2. Hypo- and aplastic anemias, achrestic anemias
3. Chronic anemiasdue to enzyme deficiency
4. 1,2
5. 1,2,3

8. Anemias, due to impaired erythropoesis may be associated with deficiency in:

1. Iron, Vit B12, Folic acid
2. Spectrin, glutamine
3. Erythropoietin, microelements
4. 1,3
5. 1,2,3

9. Iron deficiency in anemia is a results of:

1. Reduced alimentary intake
2. Impaired ionization and resorption in gastro-intestinal tract
3. Impaired transport to the liver and the bone marrow
4. Impaired utilization in the bone marrow
5. Increased demands or increased loss
6. All of the above

10. The main pathogenetic factor in iron-deficient anemia is:

1. Impaired haemoglobin synthesis

2. Reduced stimulus for erythropoesis
3. Impaired maturation of the erythroblasts
4. Reduced protoporfirin levels
5. Genetic transferin deficiency

11. The mechanism of action of Vit B12 is:

1. Transforms folic acid into folinic; activates nucliec acids synthesis
2. Stimulates the M-phase during mitosis in erythrocytes
3. Stimulates transformation of protoporfirin into haemoglobin
4. Stimulates the accumulation of haemosiderin and development of haemosiderosis
5. Stabilizes the haemoglobin molecule

12. The pathogenesis of pernicious anaemia is due to:

1. Genetically determined insufficiency of the haemopoesis
2. Production of autoantibodies against gastric parietal cells and/or gastromucoprotein
3. Structural alterations in the erythrocytic membrane
4. Intestinal parasites (Diphylobotrium latum)
5. Insufficient secretion of Hydrochloricacid and pepsin

13. Pathognomonic sign of pernicious anaemia is:

1. Hunter's glossitis
2. Funicular myelosis
3. Shunt hyperbilirubinemia
4. Megaloblasts in the bone marrow and megalocytes in the peripheral blood
5. Nocturnal haemoglobinuria

14. Causes for haemolytic anaemias are:

1. Exogenous factors in primarily intact erythrocytes
2. Endogenous extra-erythrocyte factors in primarily intact erythrocytes
3. Intraerythrocyte factors
4. Combined effect of intra- and extraerythrocyte factors
5. 1,2,3
6. 1,2,3,4

15. Which of the following exogenous extraerythrocytic factors are causes for haemolysis:

1. Serpent and fungal poisons, drugs
2. Aniline-containing paints, x-ray radiation
3. Thermogenic and cryogenic antibodies
4. 1,2
5. 1,2,3

16. Haemolytic anaemias due to intraerythrocytic factors are:

1. Hereditary shperocytic anaemia of Minkowski-Chauffard
2. Hereditary non-spherocytic enzyme-deficient anaemias
3. Haemoglobinopathies
4. Haemolytic anaemias in malaria and kala-azar
5. 1,2,3
6. 1,2,3,4

17. Haemolytic disease of the newborn is a consequence of:

1. Increased HbF levels
2. Increased number of enzyme deficient erythrocytes
3. Increaed levels of maternal Rh antibodies in the blood of Rh-negative fetus
4. Incompatible (ABO) haemotransfusion to the mother during delivery
5. Spontaneous activation of cryogenic antibodies during delivery

18. Haemoglobinopathies are hereditary diseases, resulting from:

1. Enzyme defect in the glicolytic cycle
2. Glucose-6-phosphatedehydrogenase deficiency in erythrocytes
3. Reduced gluthatione deficiency in erythrocytes
4. Genetically determined defect in the globin synthesis of haemoglobin
5. Genetically determined defect in the protoporphyrin ring synthesis

19. Microshperocytic anaemia of Minkowski-Chauffard is a result of:

1. Defect in the synthesis of haemoglobin beta chains
2. Genetic defect in the protein structure of the erythrocyte membrane
3. Inactivated glucose-6-phosphate-dehydrogenase and presence of cryogenic autoantibodies
4. Hexokinase inactivation
5. Methaemoglobin reductase deficiency

20. Glucose-6-phosphate-dehydrogenase deficiency in erythrocytes leads to:

1. Glycolytic chain disturbance with consequent energetic deficiency
2. Impaired glutathione reduction with decreased erythrocyte resistance
3. Increased tendency to polymerisation of haemoglobin
4. Impaired synthesis of globin beta-chain
5. Disturbances in the tricarbonic acids cycle

21. Cooley's anemia (Thalassemia major) is a result of:

1. Genetic deffect in the synthesis of haemoglobin beta-chain
2. Genetic deffect in the synthesis of haemoglobin alpha-chain
3. Deficiency of glucose-6-phosphate-dehydrogenase and reduced gluthatione
4. Replacement of glutamine with valine at 6th place in the beta-chain
5. A complication of haemorrhagic disease of the newborn

22. The basic mechanisms for quantitative changes in leukocytes are:

1. Stimulated or suppressed leukopoesis
2. Recruitment of reserve leukocytes from haemopoetic organs
3. Re-distribution of leukocytes in the vascular system
4. ncreased leukocyte destruction in peripheral tissues
5. 1,2,3
6. 1,2,3,4

23. Distributive (peripheral) leukocytosis is observed in:

1. Strenuous physical work
2. Infectious diseases
3. Inflammatory and necrotic processes
4. Stress conditions
5. 1,4
6. 1,2,3,4

24. Causes for leukopenia are all of the stated, except for:

1. Severe and continuous intoxication
2. Treatment with cytostatic drugs
3. Hypersplenism
4. Increased sympathetic tone
5. Decreased vagal tone

25. The clinical manifestation of leukopenia is related to:

1. Impaired defensive reactions of the organism
2. Development of oedematous syndrome
3. Disturbed control of anabolic processes
4. Postponed regenerative ability of the bone marrow
5. Thromboembolic diatheses

26. Leukosis is characterized by:

1. Isolated bone marrow hypoplasia
2. Inflammatory-toxic leukoproliferation
3. Hyperplasia, metaplasia and dedifferentiation of haemopoetic cells
4. Metaplasia of hemopoeticcells into non-hemopoetic
5. 1,2,4
6. 3,4

27. Leukemoid reactions are considered as:

1. Premorbid conditions, potentially progressing into leukosis
2. Reactive alterations in haemopoesis, similar to leukoses
3. Post-leukotic pathological conditions
4. Antigene-provoked response of the haemopoesis
5. A form of leukosis remission

28. The ethiologyof leukoses is related to:

1. Ionizing radiation
2. RNA-viruses
3. Chromozome anomalies
4. Exogenous and endogenouscancerogenic substances
5. All of the above

29. Haemorrhagic diatheses are characterized by:

1. Increased propensity tohaemorrhages
2. Increased propensity to haemoconcentration
3. Decreased fibrinolytic activity
4. Potential for embolism development
5. 1,3,4

30. Disturbances in haemostasis are classified as:

1. Coagulopathies

2. Thrombocytopathies
3. Vasopathies
4. Fibrilopathies
5. 1,2,3
6. 1,2,3,4

31. Coagulopathies develop as a result of:

1. Quantitative and qualitative alterations in thrombocytes
2. Alterations in the permeability of the vessel wall
3. Quantitative and qualitative alterations in plasma factors
4. Quantitative and qualitative alterations in erythrocytes
5. Spontaneous haemodilution

32. Which are the alterations in plasma factors thatparticipate in coagulation?:

1. Congenital, related to genetic defect
2. Acquired, resulting from liver and renal disease
3. Manifestation of disturbed Vit K metabolism
4. Consequences of uncontrolled anticoagulant application
5. All of the above

33. The genetically caused deficiency of factor VIII is a main pathogeneticunit in:

1. Haemolytic disease of the newborn
2. Haemophilia A
3. Haemolytic anemia of Minkowski-Chauffard
4. Haemoglobinopathies
5. Thrombasthenias

34. Which phase of coagulationis affected in haemophilias?:

1. Fibrinolysis

2. Fibrinogenesis
3. Blood clot retraction
4. Thromboplastin synthesis
5. Transformation of prothrombin into thrombin

35. Which are the pathogenetic mechanisms in the development of thrombocytopenia?:

1. Suppressed thrombogenesis
2. Increased platelets destruction in the periphery
3. Suppressed thrombin synthesis
4. 1,2
5. 1,3

36. Haemorrhagic syndrome in thrombasthenia is a result of:

1. Significant thrombocytopenia
2. Acquired or genetically determined functional insufficiency of platelets
3. Disturbed thrombin synthesis
4. Disturbed thromboplastinsynthesis
5. Increased blood clot retraction

37. The main pathogeneticunit in capillaropathies is:

1. Insufficient platelets adhesion
2. Procoagulant deficit
3. Affected endothelial barrier and capillary permeability
4. Anticoagulant surplus
5. 2,3