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Cerebral-Vascular Diseases (CVD)

Ischemic Heart Diseases (IHD)

Lecture on pathomorphology for the 3-rd year students

CVD - it is a cerebral variant of atherosclerosis
IHD - this is a cardiac variant of atherosclerosis

Common pathological changes of vessels at CVD and IHD

- atheromatosis and athero-caltsinosis of arteries with stenosis
- circular hyalinosis with the critical narrowing of heart or brain vessels at the patients with hypertension
  disease

VASCULAR DISEASE OF THE NERVOUS SYSTEM

1. Vascular-discirculation encephalopathy:
   Ischemic
   Hypertensive
2. Cerebral haemorrhage:
   - Intracerebral
   - subarachnoidal
3. Brain stroke (ischemic, hemorrhagic, ischemic infarction with haemorrhages)

The ischemic encephalopathy (IE)

- It is a diffuse defeat of brain neurons with diffuse small-part character necrosis of neurons and hyalinosis of vessels.
- IE develops at the decrease of cerebral blood-volume less then 25-10 ml on 100gr of tissue.
- At the decreasing of cerebral blood-volume in 2 times the ischemic damage of neurons is observed.

Reasons of the decreasing of cerebral blood-volume

- stenosis of cerebral arteries
- thrombosis of the atherosclerotic plaque
- protracted spasm of vessels

The ischemic encephalopathy (IE)

- Laminar necrosis - ischemic changers of pyramidal cell layers of the cerebral cortex.
- Adaptive (around-neuronal) satellitosis - glial cells are gathered round neurons.
- Zones of gliofibrosis are observed in the place of necrotic changers.

The ischemic encephalopathy (IE)

- Acute
- Sub acute
- Chronic with relapses (at seniors with the expressed atherosclerosis)

Outcomes of IE

- violations of sensitiveness
- violations of motions
- violations of memory

The hypertensive encephalopathy

- It is hypertensive hyaline arteriolar sclerosis. At the moment of crisis a fibrinoid necrosis of the arteriole walls of brain is observed, it leads to vascular-genic edema of brain (acute swelling).
- The dislocation (herniation) of brain begins into the natural opening (foramen magnum);
- The cortex layer of brain stake is hurt in the process of dislocation;

The hypertensive encephalopathy

- Haemorrhage begins in the upper 1/3 of Pons (in the zone of cardio-respiratory centers).
- Displacement of cerebellum in foramen magnum leads to compression of basal artery and ischemia of cardio-respiratory center.
- The diapedesis haemorrhage arises up round vessels, so the cavities with haemosiderophages are formed. They are named - lacunar infarcts.

The hypertensive encephalopathy

- Lacunar infarcts ("lacunae") are little infarcts, a few mm across, typically in the deep structures of the brain
- In fatal cases, necrotic changers of blood vessels are seen, much like in the kidney at "malignant hypertension".

Outcomes of HE

- death in the acute period
- the progressive disorders of memory, sensitiveness, motions and etc

“Brain Stroke“ -

- it is a sudden onset of a permanent, localized neurologic deficit, may result either from hemorrhage (1) or infarction (2), and has a multitude of specific causes.

The infarction of brain

- ischemic infarction (75%) - develops at the obstructive thrombosis or thrombi-emboli
- ischemic infarction with hemorrhages (5-10%) - at embolism of vessels
- hemorrhagic infarction - "anemic infarcts" complicated by dissolution of an embolus or backflow of blood from the margins.

Clinic: hemiplegia and disorders of sensitiveness on the other part of defeat, and disorders of speech at the involving of cortex of brain.

Reasons of brain infarcts

- Thrombotic infarcts
- Embolic infarcts
- Subclavian steal syndrome (Robin Hood syndrome), in which a patient with occlusive atherosclerosis of a proximal subclavian artery suffers brainstem syndromes upon exercising the arm on the involved
- Granulomatous angiitis of the CNS
- Moyamoya disease - the process in which the vessels of the Willis circle and nearby become narrowed (fibrosis of the intima) and may bleed.

The evolution of brain infarction (stages)

- Ischemia (1–2 days) – there is an area of [illegible]
- [illegible] very soft [illegible] necrosis [illegible]
- After 2–3 weeks – resorption of the necrosis, formation of cyst

Brain hemorrhage

- Sudden arising up of the volume in one hemisphere of brain brings to the rapid dislocation of brain & death.
- The haemorrhage mass can break through into the ventricles of brain on any stage that leads to coma. The second trunk syndrome develops (defeat of reticular structure).

Brain hemorrhage. Reasons.

- "Hypertension" - arterial pressure higher then 180mmHg item
- the break of artery, or aneurism, or vascular malformations ("angiomas")
- bleeding disorders
- hemorrhage into brain tumors (primary, metastatic)
- Congo-philic (amyloid) angiopathy (hereditary, idiopathic; "Alzheimer's amyloid angiopathy")

Brain hemorrhage. Classification.

- Intra-brain - in area of under-cortex ganglier and visual hillock, rarely in the cerebellum and trunk of the brain
- Sub-arachnoidal hemorrhage.

- **According to morphology features**
- hematoma - massive bleading
- hemorrhagic infiltration.

The sub-arahnoidal hemorrhage - reasons of development

- Break off innate or acquired aneurism.
- Vascular malformations - may bleed into the subarachnoid space, the brain substance, or both. Arteriovenous malformations (masses of large blood vessels) tend to be located in the hemispheres
- Germinal plate hemorrhages in premature babies - bleeds into the ventricles, rather than the subarachnoid space.
- Atherosclerotic aneurysms in the head are typically fusiform [illegible] tations of the basilar

Classification of Ischemic heart disease

- Acute IHD: angina pectoris, acute coronal insufficiensy, acute myocardial infarction, repeated myocardial infarction, Sudden cardiac death
- Chronic IHD: stenosis and occlusion of coronary arteries, postinfarction cardiosclerosis, chronic aneurism of heart wall.

Ischemic heart disease

- It is disease that is conditioned by the relative or absolute insufficiency of coronal blood supplying that is secondary leads to irreversible changers of myocardium.

CAUSES

- Atherosclerosis of coronal arteries
- Concentric hyalinosis and circulation stenosis

Angina pectoris

- It is disparity between necessities of oxygen and its supplying to myocardium.

Reasons of development:

1. Prolonged spasm of coronal arteries at hypertension disease. Spasm that is longer than 60 minutes leads to myocardial infarction.
2. Coronal stenosis at atherosclerosis
3. Circular hypoxia at: cardiomyopathies, arrhythmias, heart vices, heart de-compensation

Angina pectoris

- Stable ("classic") angina - results from increased work in a patient with coronary atherosclerosis, and relieved by rest.
- Unstable ("acute coronary insufficiency") angina - due to a thrombus developing, by fits and starts, over a ruptured plaque. In duration less than 60 minutes.

Prinzmetal's angina - primarily attributable to vasospasm. Sudden cardiac death can be observed at this patients.

Cardiac syndrome X ("microvascular angina") classical clinical angina and wide-open coronary arteries

Acute coronal insufficiency

- It is inability to satisfy metabolic necessities of myocardium by coronal blood supplying.

Reasons of development:

- Brief spasm of coronal arteries (less than 60 minutes)
- Brief increasing of concentration of catecholamine at stress
- Physical overload at stenosis of one artery (haemodynamic disturbances)

Acute coronal insufficiency.

Complications and outcomes:

- Reperfusion post-ischemic damage of myocardium by free radicals, ions, ets.
- Damage by mediators of platelets, toxins leucocytes and lymphocytes
- Local necrosis and apoptosis of cardiomyocytes
- Damage of endothelium that leads to thrombi formation

Myocardial infarction

- It is ischemic partial necrosis of myocardium wall due to sudden loss of the blood supplying.

Myocardial infarction. Reasons.

- Atherosclerosis: ruptured plaque - often with an overlying thrombus ("coronary thrombosis"); massive haemorrhage into a plaque, ballooning its cap against the opposite wall.
- Prolonged spasm of coronal arteries - more than 60 mines in duration
- Physical overloading of patient with critical stenosis of coronal arteries (more than 75%)
- Thrombosis of coronal arteries
- Cocaine use, Prinzmetal's coronary spasm, Vasculitis, Embolization, Syphilis ,other

Myocardial infarction. Classification.

- According to localization and spreading: sub-epicardial, sub-endocardial, intramural, transmural
- According to time of development: acute primary - 4 weeks from the beginning, recidivating (relapsed) - the formation of the new necrosis during 4 weeks on the background of primary infarction, repeated - the formation of the new necrosis after 4-th week from the beginning of 1-st one.
- According to the stage of development: Ischemic stage - 12-18 hours

  Stage of necrosis - 18-24 hours up to 5 days

  Stage of organization - 5 days - 7 weeks

Morphological characteristics:

- ischemic - through 60 seconds, after stopping of blood-circulation, the abbreviation of myocytes is halted, but during the 1-st days a nuclear is stored, and membranes of organell's gradually collapse (picnosis and eosinophylia of cytoplasm)
- necrosis - in a 24 hour from the beginning of ischemia (kariolysis, kariopiknosis) of about 5-7 days, grows myomalyatsia of heart walls (wall is yellow-green), on periphery - hemorragic halo.
- organization - into the area of necrosis vessels grow up and migrate fibroblasts - zone of cardiosclerosis. A scar is formed by the end of 2th month.

Diagnose of ischemic stage of infarction during autopsy

- The nitro-blue tetrazolium technique can demonstrate early myocardial infarcts.

  Drop a slice of heart in the solution, and viable heart, containing an oxidizing enzyme, will stain brown, and dead heart remain pale.

Complications of myocardial infarcts

- Ischemic stage: rhythm disturbances with stopping heart work, Left-sided congestive heart failure, Cardiogenic shock, Acute coronal insufficiency
- Stage of necrosis: Rupture of the heart - occur, when the damaged heart is most soft (days 3-5),

  Formation of acute aneurysm,

  Mural thrombus formation in aneurism and embolization,

  Rupture of the wall of acute aneurism,

  Dressler's pericarditis (fibrin pericarditis)

Complications of myocardial infarcts

Stage of organization

- Formation of chronic aneurysm.
- Near-wall mural thrombus formation in chronic aneurism and embolization
- Formation of recidivating (relapsed) infarction
- Progression of myocardial insufficiency