**Cellular injury. Necrosis**

Patient A., 55 years old, who suffers from atherosclerosis, was urgently admitted with the following complaints: retrosternal pain radiating to the shoulder blade and left arm (nitroglycerin administration does not alleviate the pain), general weakness, and sweating. Presumptive diagnosis – myocardial infarction.

**Objective:** pallor, cold and moist skin;
**ECG:** ST-segment elevation; pathological Q wave.
**Blood:** elevated total CK (creatine kinase), CK-MB > 190 U/L (Normal < 24 U/L), troponin I, myoglobin, LDH, AST, lactate. Hypercholesterolemia. Hyperkalemia.

**Questions:**

1. What are the pathogenetic mechanisms of hypoxic injury? (Example of a pathogenetic chain)
2. What is the pathogenetic mechanism of hypoxic injury through the cessation of Na+/K+ pump activity?
3. What is the pathogenetic mechanism of hypoxic injury through the cessation of Ca2+ pump activity?
4. What is the pathogenetic mechanism of hypoxic injury through the activation of anaerobic glycolysis?
5. What is the role of oxidative stress in the pathogenesis of cellular hypoxic injury?
6. What indicators confirm cardiomyocyte injury in the patient?
7. What is the mechanism of hyperkalemia, and what electrophysiological changes are observed in the patient as a result?

**Apoptosis**

Patient D, 54 years old, complains of neurological symptoms: difficulty remembering recent events, disorientation in time and space, speech disturbances, slowing of cognitive processes, and rapidly progressing dementia. He was diagnosed with Alzheimer's disease, which is a neurodegenerative pathology (progressive loss of structure and function, as well as neuron death).

**Questions:**

1. What is apoptosis and what are the stages of apoptosis?
2. What are the differences between apoptosis and necrosis?
3. What do the extrinsic and intrinsic pathways of apoptosis represent?
4. What disorders of apoptosis do you know, and what pathologies can result from excessive or insufficient apoptosis?

**Dystrophies**

Patient G., 52 years old, was admitted to the hepatology department with a preliminary diagnosis of "hepatitis." From the medical history, for 20 years he has been under the care of a narcologist with the diagnosis of "chronic alcoholism."
Objective - enlarged liver with soft consistency.
Liver biopsy performed for diagnostic purposes: Hematoxylin and eosin staining revealed cytoplasmic vacuolization of hepatocytes, and Sudan staining showed fat droplets.
Biochemical analysis: Moderately elevated AST and ALT, elevated triglycerides.

**Questions:**

1. What causes contribute to fatty liver disease?
2. Through what mechanisms has alcohol contributed to the development of the dystrophy?
3. What metabolic processes have been affected and contributed to the accumulation of lipids in hepatocytes?
4. How does oxidative stress contribute to the damage of hepatocyte structure and to the intracellular accumulation of lipids?
5. What is the pathogenetic mechanism of dystrophies affecting the mitochondria?
6. What is the pathogenetic mechanism of dystrophies affecting the functioning of the cell membrane pumps?
7. What are the possible consequences of hepatic dystrophy?