Structural Changes of Internal Organs in Experimental Purulent Peritonitis

Suleymanov R.I.	Assistant of the Department of Human Anatomy Samarkand State Medical University
Kiyamov B.E.	Assistant at the Department of Human Anatomy Samarkand State Medical University
Kamalova M. I.	Associate Professor at the Department of Human Anatomy Samarkand State Medical University

ABSTRACT

The structure of the intestine's wall. liver and heart were studied in acute peritonitis and postoperative period. The experiment was performed on 25 male rabbits. Morphological changes were investigated using light microscopy with haematoxylin-eosin and Van Gison staining.

It was shown that the development of acute peritonitis led to significant changes in the intestine. liver and heart. Inflammatory reaction with predominance of exudation was combined with dystrophic alteration in described organs. The changes arose towards the first day and lowered by the fifth day of postoperative period when regeneration processes developed.

Keywords: acute peritonitis, intestine, liver, heart, structural changes.

Introduction. The problem of treatment of widespread purulent peritonitis is still topical. First of all, it is associated with high mortality rate, which is estimated by different authors to be from 12% to 84%. The leading cause of the progression of peritonitis with subsequent adverse outcomes is enteral insufficiency - a violation of the motor, absorptive and excretory functions of the small intestine. Lack of peristalsis leads to loss of colonization resistance of intestine. translocation of pathogenic and opportunistic microflora to unusual habitats. bacteremia. development of abdominal sepsis. multiple organ failure.

Over the past decades significant progress has been made in the study of widespread purulent peritonitis. Nevertheless, many questions of pathogenesis and efficacy of various approaches to the treatment of widespread peritonitis remain unsolved.

Objective of the work. To study the dynamics of structural changes of internal organs at widespread purulent peritonitis in the experiment.

Material and methods: The experiment was carried out on 25 male rabbits of the chinchilla breed (weight 2500-3000 g). The animals were divided into the following groups: Group I - 6-hour widespread purulent peritonitis (n=5); Group II - 6-hour widespread purulent peritonitis in 1. 3. 5 days after the operation (n=15). 5 healthy rabbits were used as a control. To simulate widespread purulent peritonitis we used a microbial mixture. consisting of equal amounts of aerobes). The microbial mixture was injected into the animals' abdominal cavity with a sterile syringe at the rate of 6 billion microbial bodies per 1 kg of rabbit weight.

Laparotomy was performed in the 1st group after 6 hour peritonitis under intravenous nembutal anesthetic (30 mg/kg) plus local anesthesia with 50 ml 0.25% novocaine.

Purulent hemorrhagic effusion was removed. The abdominal cavity was flushed with 0.02% chlorhexidine bigluconate solution and 3% H202 solution in the ratio 10:1. After that, two "acidic" sutures were placed on the wall of the cecum close to the inflow of the small intestine into it. Cecotomy was performed. A perforated polychlorvinyl tube 3 mm in diameter was inserted 30-40 cm into the small intestine. intestinal contents were removed. decompression and washing of the intestine with physiological solution until clear water was obtained. Intestinal sutures were tightened tightly around the tube and tied. Through a puncture in the anterior abdominal wall 4-5 cm to the right of the laparotomy wound, the tube was led outside. The intestine was sutured hermetically to the parietal peritoneum with sutures. The laparotomy wound was sutured layer by layer and the drainage tube was fixed to the skin with a knotted caprone suture-holder. Immediately after the operation and every 8 hours during the first day, the small intestine was irrigated with physiological solution in the amount of 40-60 ml per procedure. The drainage tube was removed from the small intestine lumen on the 2nd day after surgery.

Material for morphological study was sections of small intestine wall, liver and heart. Group I animals with widespread purulent peritonitis were removed from the experiment 6 hours after infection. Group II - in 24 hours (1 day). 72 hours (3 days). 120 hours (5 day) after the operation.

For light microscopy the material was fixed in a 10% solution of neutral formalin. After standard wiring, paraffin sections were prepared, which were stained with hematoxylin-eosin and Van Gizon method. Evaluation of morphological changes was performed on light optical level at x100. x200 and x400 magnification.

Results and discussion. Experimental modeling of widespread purulent peritonitis in animals resulted in the development of marked changes both in the intestinal wall and in the liver parenchyma and heart wall. Examination of intestinal wall in 6 hours after peritonitis development showed sharp disorder of blood and lymph circulation in the form of stasis in capillaries and venules. edema. full blood vessels. hemorrhages. which were combined with the appearance of exudative purulent inflammation in all membranes. and also pronounced dystrophic, necrobiotic and necrotic (with total necrosis of villi) changes. The material for morphological study was sections of small intestine wall, liver and heart. Group I animals with widespread purulent peritonitis were removed from the experiment 6 hours after infection. Group II - in 24 hours (1 day). 72 hours (3 days). 120 hours (5 day) after the operation.

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Dystrophic, necrobiotic and necrotic changes were most pronounced in the muscular layer of small intestine. In the mesentery there were also full blood vessels, hemorrhages, edema and loosening.

Edema and loosening of the capsule were determined in the liver of the animals of this group. Hemodynamic disturbances also occurred in the parenchyma. which was expressed in the fullness of central veins and sinusoid capillaries. rupture of the vascular wall and the appearance of small hemorrhages. The

wall of the central veins and portal tracts was edematous in most fields of view.

Large number of neutrophils with marginal standing in most of them was detected in the lumen of vessels of various links. In addition. there was diffuse pronounced polymorphic-cellular. with predominance of neutrophils. inflammatory infiltration of parenchyma.

In most part of hepatocytes dystrophic (from granular to small drop fatty dystrophy). necrobiotic. necrotic changes with presence of foci of cell necrosis mainly at periphery of lobules and in subcapsular zone were determined.

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Changes in the heart of animals in 6 hours after peritonitis development were characterized by the development of sharply pronounced (more under epicardium) circulatory disorders in the form of vascular hyperemia. stasis in the vessels of microcirculatory channel. diapedesis hemorrhages and sharply pronounced interstitial edema of myocardium. Cardiomyocytes stained irregularly, some of them did not have transverse striation. In 24 hours after surgery in all animals there was an increase in the severity of morphological changes in the studied organs, but to a greater extent in the wall of the small intestine. At the same time in all parts of the intestine there was sharply pronounced diffuse polymorphous cellular infiltration with predominance of neutrophils in all membranes, destruction of part of villi, diffuse vascular hyperemia, focal diapedesis hemorrhages.

The stroma of preserved villi was edematous, their epithelial surface showed a rather pronounced fibrin plaque. Dystrophic up to hydropic dystrophy was observed in villous epithelium. Dystrophic and necrobiotic changes were also noted in epithelial cells of glands, in elements of intermuscular nerve plexus with phenomena of cytolysis and places of neuron loss (necrosis and autolysis) in ganglia, as well as in myocytes, to a greater extent in the outer layer of the muscularis. In the intestinal mesentery there was also an increase of exudative purulent inflammation.

In the liver along with pronounced hemodynamic changes in the form of portal tracts expansion, sinusoidal capillaries and central veins, as well as hyperemia of some capillaries the destruction of the central vein wall and massive necrosis fields were observed, which determined the violation of the classical lobule structure. To a greater extent in the periphery of lobules in a significant part of capillaries there was a marginal standing of neutrophils.

To a greater extent in the periphery of lobules inflammatory polymorphocellular with predominance of neutrophils inflammatory infiltration of parenchyma and expressed dystrophic (swelling of hepatocytes, hydro-epic dystrophy), necrobiotic and necrotic changes of hepatocytes were observed.

Circulatory disorders in the cardiac wall in the form of moderate hyperemia of vessels. their dystonia with alternating areas of contraction and dilation. stasis in the microcirculatory vessels. diapedesis (mostly in pericardium) hemorrhages combined with moderate to severely pronounced interstitial and perivascular edema

Cardiomyocytes stained irregularly, in some of them there were no determined transverse striation, there were signs of necrobiosis and necrosis in some fields.

On the 3rd day in all layers of the small intestine there were slightly to moderately pronounced hemodynamic changes and diffuse moderately pronounced polymorphocellular inflammatory infiltration, and in the direction from proximal to distal parts the infiltrate composition varied from predominantly lymph-macrophage to neutrophil-cellular.

Reactive hyperplasia of lymphoid follicular tissue was observed in the distal sections of the small intestine. In the mesentery, neutrophils also dominated the inflammatory infiltrate.

Stromal edema and necrosis of the apical part were detected in some villi. In the preserved sections the epithelium of villi and crypts was indistinct, the brush border was not pronounced throughout. Swelling of myocytes was detected in muscular mucosa against edema and diffuse inflammatory infiltration).

Dystrophic and partially necrobiotic changes were preserved in cellular elements of mucosa, muscular membranes and elements of nerve plexus.

Thickening and edema of capsule and moderately pronounced diffuse polymorphocellular with large number of neutrophils inflammatory infiltration of parenchyma with presence of purulent inflammation foci and predominantly lymphomacrophage infiltrates around portal tracts in some areas were detected in the liver

Diffuse from slightly to sharply pronounced interstitial edema and pronounced perivascular edema and vascular hyperemia persisted in the heart. These changes were combined with diffuse dystrophic changes in a significant part of cardiomyocytes and predominantly polymorphocytic interstitial infiltration with the presence of single microabscesses. By the end of the 5th day the character of changes in the internal organs was the same, but less pronounced. Besides, along with inflammatory, dystrophic and necrotic processes, there was an increase in compensatory-adaptive changes.

Throughout the intestine the intestinal structure was preserved. However, in all layers (but mostly in mucosa, including epithelium) there was diffuse from slightly to moderately expressed polymorphocellular (mainly lymph-macrophage in proximal sections and neutrophil-cellular with presence of single abscesses in submucosa - in distal sections) inflammatory infiltration. The submucosa edema was expressed to a great extent, while the mucosa edema was predominantly focal and weak. There were focal epithelial desquamation in villi as well as dystrophic changes in some epithelial cells. Dystrophic changes in the epithelium of villi and glands were more pronounced in the distal parts of the intestine.

In the liver, there was a decrease in the number of cells in a state of dystrophy and necrosis and an increase in mitotic activity of hepatocytes. A consequence of this was restoration of the structure of liver beams and liver lobules as a whole. At the same time there was uniform dilation of most veins and sinusoidal capillaries and predominantly large focal polymorphocellular (with significant amount of neutrophils) inflammatory infiltration of parenchyma.

In the heart there was marked diffuse polymorphocellular (mainly lymphomacrophage) inflammatory infiltration with the presence of sufficiently large focal. mainly perivascular infiltrates. combined with hyperemia of a significant part of vessels. sufficiently pronounced perivascular and diffuse interstitial edema and degenerative changes of a significant part of cardiomyocytes.

Conclusion.

Peritonitis is an abdominal sepsis. Widespread purulent peritonitis already in 6 hours leads to generalization of intra-abdominal infection with severe structural changes not only in the small intestine, but also in the liver and heart. The increase of changes in the specified organs 24 hours after surgical intervention indicates that sanation of the abdominal cavity and decompression of the small intestine are insufficient to interrupt "cascade" pathological process, eliminate enteral insufficiency and endogenous intoxication. The results of investigation testify to the necessity of including preparations promoting increase of small intestine resistance and possessing detoxifying properties in complex treatment of widespread purulent peritonitis.

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