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Morphological Aspects of Intestinal Insufficiency in Experimental Peritonitis

Dilorom Bakhtiyarovna Adilbekova[1,*], Sayfiddin Risbaevich Baymakov[2], Khusain Salimatdinovich Abdurazzakov[3]

[1]Department of Anatomy and Clinical Anatomy, Tashkent Medical Academy, Tashkent, Republic of Uzbekistan
[2]Department of Surgery, Tashkent State Dental Institute, Tashkent, Republic of Uzbekistan
[3]Department of Surgery, Tashkent Medical Academy, Tashkent, Republic of Uzbekistan

Abstract Our studies show that pathomorphological changes in the small intestine in acute experimental peritonitis are characterized by dystrophic, inflammatory-destructive and vascular disorders in the vascular-tissue structures of the small intestine wall. In the pathogenesis of damage to the small intestine, deep disorders in the vessels of the microhemocirculatory course from hyperemia to inflammatory and destructive changes are of great importance, subsequently leading to a violation of the trophic cells, tissue hypoxia, damage to cellular elements, violation of cellular metabolism, deficiency in energy and plastic materials, accumulation of perverted metabolic products in cells and tissues.

Keywords Acute experimental peritonitis, Small intestine, Vessels, Tissue structures

1. Introduction

Intestinal insufficiency, occupies a leading place among all complications of acute pathology of the abdominal cavity, the mortality rate is high (from 15% to 40%) and has no tendency to decrease [1,6,11]. Taking into account the fact that the intestines are the main source of intoxication, the intestinal insufficiency syndrome is currently considered as a disease of the entire digestive tract with the appearance of multiple organ changes [3,8,10]. The severity, prevalence, severity and phases of pathological processes in the abdominal cavity determine the degree of morphofunctional changes in the intestinal wall. In the literature available to us, we noted the lack of data on the degree of pathomorphological changes in the intestine in acute diseases of the abdominal cavity. In this regard, the study of this issue is an urgent problem for both clinical and fundamental medicine.

The aim of the study was to study and evaluate morphological changes in the intestinal wall in acute experimental peritonitis.

2. Materials and Methods of Research

The object of the study is the materials of the small intestine of rats removed on the 1st, 2nd, 3rd and 4th days of the experimental model of peritonitis (the experimental group was 50 rats). Peritonitis was caused by the introduction of a filtered 2% autofecal suspension in the amount of 350 mg/kg into the abdominal cavity, no later than 20 minutes after preparation under ether anesthesia. The control group consisted of 40 rats, normal (physiological) saline in an equivalent volume was injected into the abdominal cavity them. In order to avoid damage to internal organs when fecal suspension was introduced into the abdominal cavity, the animals were placed vertically, with the caudal end up. To achieve the goal and set tasks, general morphological, electron microscopic research methods were used. All studies were conducted in accordance with the "International Recommendations for conducting biomedical research using Animals" (1985, 1989). The morphological data obtained during the study were subjected to statistical processing on a Pentium-IV personal computer using the Microsoft Office Excel-2012 software package, including the use of built-in statistical processing functions. The differences satisfying $P<0.05$ were considered reliable.

3. Results

1-2 hours after the simulation, the first clinical signs of peritonitis were observed in the experimental rats: restless animals, refusal of food, tension in the abdominal area. After 6-12 hours, the animals are sedentary, inhibited, grouped in the corner of the cage, sluggish, apathetic to food, there is frequent shallow breathing, ruffled hair. After a day, the abdomen is sharply swollen, with laparotomy, the accumulation of 1.5-2 ml of serous-hemorrhagic fluid in all

* Corresponding author:
dilorom.adilbekova65@gmail.com (Dilorom Bakhtiyarovna Adilbekova)
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parts of the abdomen was determined. The leaves of the peritoneum and mesentery of the small intestine were dull, rough, there was vasodilation, small-point hemorrhages and inhibition of intestinal motility. Morphological examination of the wall of the small intestine revealed that the intestinal wall is edematous, loosened, infiltrated. There is edema in the mucous membrane, infiltration by mononuclear cells. Moderate dystrophic changes were detected in the epithelial cells of the mucous membrane. The stroma of the villi and crypts is edematous, infiltrated, loosened. In the tops of the villi (strands), desquamation of the epithelial layer is determined, which led to the formation of microerosion. The height of the villi and crypts, the total number of epithelial cells and the number of mitotically dividing cells are reduced compared to the indicators of control animals. There is an increase in the proportion of swollen goblet cells (see Table 1). Electron microscopically, fine-grained granules and transparent vacuoles are detected in the cytoplasm of enterocytes, some cells are enlarged in size, are in a state of pronounced cytoplasmic edema with a shift of the nuclei to the apical edge of the cell.

In the submucosal layer, there is edema, loosening, infiltration by mononuclear cells. There is pronounced edema in the muscle membrane, infiltration of connective tissue layers by mononuclear cells. Subserous edema, swelling, loosening and subtotal desquamation of mesothelial cells are also detected in the serous membrane of the intestinal wall (Fig. 1).

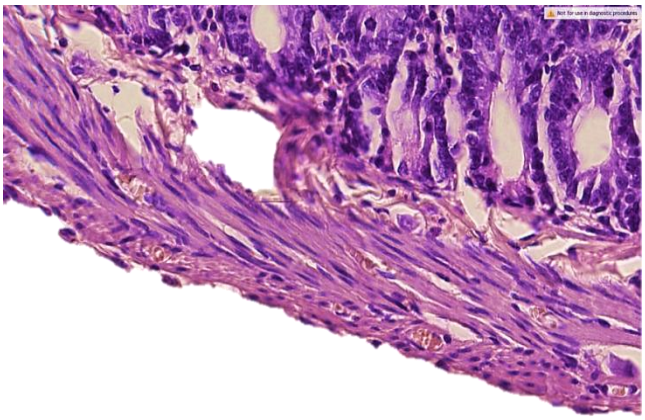

Figure 1. The wall of the small intestine on the 1st day of the development of peritonitis. Desquamation of mesothelial cells, fullness of venous vessels. Infiltration by mononuclear cells and destructive changes in the muscle membrane. Staining with hematoxylin-eosin. 40x10

Macroscopically, there is full blood in the vessels of the omentum and mesentery of the small intestine. Infiltrates are noted in the perivascular zones. Expansion and stasis are noted in the intra-organ vessels of the microcirculatory bed, microextrovasates and plasmoragia are detected in places. In the vessels of the venous part, there is swelling of the vascular wall, swelling and stretching. Stagnant phenomena are detected inside the vessels. The processes of expansion, fullness and diapedesis of shaped blood elements through the vascular wall indicated the development of angiitis and phlebitis. The capillaries are narrowed, tortuous, as a result, the capillary network is intermittent. In some places, there are "few" and "vascular-free" zones. Arterioles are spasmodic, the density of the distribution of blood vessels is significantly less, compared with the indicators of the control group of animals (see Table 2). Most animals died on the 1-2 day of the disease against the background of the development of deep pathomorphological disorders in the organ. On the 3rd day of the experiment, the preservation of edema, swelling and infiltration by mononuclear cells was noted in all layers of the intestinal wall. Often the villi are bare, without an epithelial layer. Destructive processes are detected in the apical parts of the villi and in the epithelium of the crypts (Fig. 2).

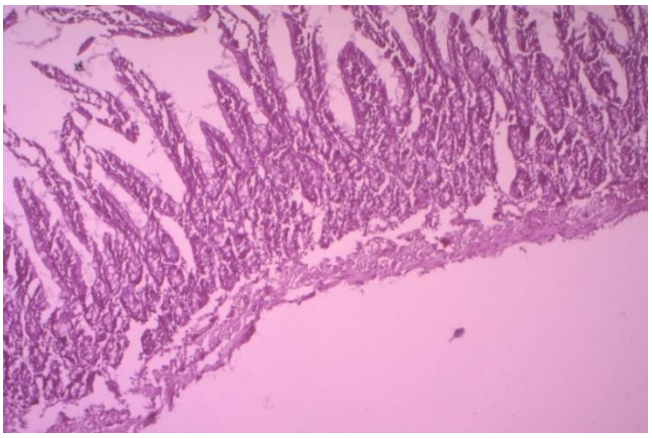

Figure 2. Morphology of the small intestine wall on day 3 in peritonitis. Destructive changes in all layers of the intestinal wall. Staining with hematoxylin-eosin. 10x10

Morphometric parameters of all layers of the small intestine wall significantly differ from those of control animals (see Table 1).

Table 1. Morphological parameters of tissue structures of the small intestine wall in experimental peritonitis, M±m

Indicators	Control group	Experimental group
Thickness of the mucous membrane, mc (micron)	548,8±3,60	568,2±4,20
The height of the villi, mc	480,0±5,01	410,5±5,05
Crypt depth, mc	284,0±2,6	245,2± 3,73
The number of epithelial cells on one side of the longitudinal section of the villi	90,2±2,58	75,4±0,57
Number of mitoses (per 1000 cells)	15,2±0,20	11,4±0,16
The relative number of goblet-shaped cells on the villi (per 100 cells)	18,5±0,50	27,5±0,35
The thickness of the serous-muscular membrane, mc	148,2±3,40	168,3±4,8

Note: * - the reliability of differences compared to the control, P≤0.05

During these periods, the above inflammatory and stagnant processes continue to persist in the microvessels of the intestinal wall: the vessels are dilated, full-blooded, swollen, their walls are edematous. There are stagnant phenomena, microthrombosis in the vessels of the venous

department. In some places, zones with the phenomena of microextravasates and plasmorrhagia are detected.

Loose cellular infiltrates are detected everywhere in the perivascular zones.

The study of the surviving animals on the 4th day of the experiment showed that there is 3-4 ml of cloudy liquid with a sharp unpleasant smell in the abdominal cavity of the animals. The parietal and visceral leaves of the peritoneum are dull, rough, sticky. There were multiple loose, purulent-fibrinous overlays on the surface of the intestine, with areas of spot hemorrhages. Necrosis foci were detected in the area of ulcers. The large omentum is swollen, infiltrated. Along with pathomorphological changes, areas with regressions of acute inflammatory processes were found in places, as evidenced by areas with granulations surrounded by thin-walled vessels. Histological examination of micro-preparations of the small intestine wall in these terms showed that all layers of the small intestine wall are swollen. There is edema in the mucous membrane, infiltration by mononuclear cells. The epithelium of the villi and crypts is flattened, with dystrophic changes, the stroma is edematous. Many villi are short and deformed. Most epithelial cells showed signs of dystrophic changes: karyopycnosis, karyolysis and vacuolization of the cytoplasm. Tissue edema was accompanied by local epithelial cell exfoliation. In some places, necrotic changes in tissue structures reached the submucosal base and the serous-muscular layers of the intestinal wall. On the apical part of many villi, desquamation of the epithelial layer is detected, with exposed, microerosion areas. The exposed areas were covered with fibrinous overlays, as a result, ulcerative defects were formed. At the bottom of the crypts, multiple goblet-shaped cells filled with secret are revealed. The submucosal base is edematous, loosened and infiltrated by mononuclear cells. Hyperemia and edema were noted in the serous membrane of the small intestine. swelling of the mesothelium, local desquamations, coldness and spot hemorrhages in the stroma. Electron Microscopically, enterocytes are flattened, their cytoplasm contains multiple vacuoles and hypochromic nuclei. Their cytoplasm is light, contains relatively less swollen mitochondria with a light matrix. A large number of vesicles are detected. Microvilli are thin, short, their number is reduced. The vacuolized Golgi apparatus is revealed. However, despite the above-mentioned pathomorphological disorders against the background of these disorders, local activation of reparative processes was noted in some places. The study of intra-organ microvessels of the small intestine showed that during these periods, some narrowing of the vessels of the arterial link and the expansion of the vessels of the venous link were observed in all the vessels of the layers of the small intestine wall, which indicates the processes of imbalance between these parts of the vessels (Fig. 3).

In the lumen of the sinuous capillaries, aggregated shaped blood elements are detected, indicating a violation of the permeability and dystonia of the vessel walls. In the vessels of the venous department, stagnant phenomena, microthrombosis are noted. The opening of arterial-venous shunts was noted. The contours of the vessel wall are fuzzy, erased. Morphometric parameters of the intestinal wall microvessels significantly differed from those of the control group animals (see Table 2). Ultrastructural study of microvessels showed that the capillary wall is three-layered: endothelium with a thin basement membrane, a layer of pericytes and an outer one consisting of adventitial cells. The basement membrane is of moderate density, somewhat thinned, loosened.

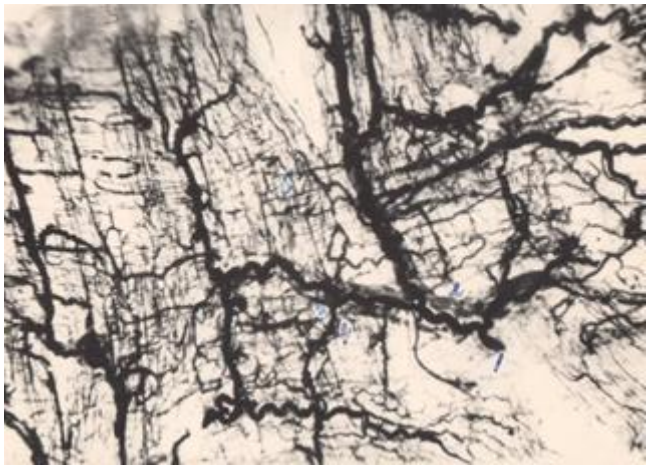

Figure 3. Blood vessels of the submucosal base of the small intestine. The capillaries are sinuous, the vessels of the venous department are dilated, full-blooded. Filling of vessels with a mass of Gerot. 10x10

Table 2. Morphometric parameters of microvessels of the mucous membrane of the small intestine wall in experimental peritonitis, M±m

Indicators	Control group	Experimental group
Diameter of the inner lumen of arterioles, mc	22,2±0,31	19,2±0,20
Capillary lumen diameter, mc	6,2±0,25	5,1±0,15
Diameter of the lumen of the postcapillaries, mc	13,1±0,15	19,5±0,24
Diameter of the venule lumen, mc	26,0±0,18	28,0±0,21
The density of the distribution of the vessels of the mucous membrane conl. units.	410,2±5,1	305,0±5,3

Note: * - significant differences were noted compared to the control $P \leq 0.05$.

4. Discussion

Pathomorphological changes in the small intestine in acute diffuse peritonitis confirmed the development of intestinal insufficiency syndrome and impaired intestinal function [2,4,5,7,9]. Our scientific studies showed a violation of microcirculation, which subsequently led to an aggravation of dystrophic and necrobiotic disorders in the tissue structures of the small intestine wall. At the beginning of the pathological process, dystrophic changes of various degrees of severity were observed, and then – necrobiotic.

More pronounced pathomorphological changes in the vessels were detected at the end of the first day: there was a decrease in the internal diameter of the vessels of the arterial link, swelling and thickening of their walls. These processes gradually progressed on the 2, 3, 4 day of the experiment. In the vessels of the venous department, pronounced fullness, expansion of the internal lumen, microthrombosis, extravasates, a decrease in the density of the distribution of vessels were observed, which led to a violation of the metabolic processes between the blood and the intestinal wall tissue. All these pathomorphological processes in vascular structures negatively affected the state of cellular metabolism and caused dystrophic and destructive changes in the tissue structures of the intestinal wall.

5. Conclusions

1. Intestinal insufficiency in experimental peritonitis is characterized by vascular, inflammatory-destructive and dystrophic disorders in the vascular-tissue structures of the small intestine.
2. The pathomorphological disorders in peritonitis are based on deep vascular disorders, as evidenced by edema and swelling and high variability of the walls, expansion, fullness of venous vessels, microthrombosis, violation of the permeability of the vessel walls, multiple extravasates, a decrease in the density of the distribution of vessels in the microhemocirculatory flow.
3. All these processes were accompanied by a violation of trophic activity in the intestinal wall, tissue hypoxia, damage to cellular elements, violation of cellular metabolism, deficiency in energy and plastic materials, accumulation of perverted metabolic products in cells and tissues.
4. A deep understanding of the pathogenesis of morphological disorders of this condition determines in the future the implementation of targeted, scientifically based, highly effective complex therapy.

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