

**STRUCTURE OF THE SMALL INTESTINE MUCOSA AFTER ACUTE
HEMORRHAGIC SHOCK AND REPERFUSION OF THE ISCHEMIC LIMB**

Salomov Shokhabbos Nozimjon ugli

Student of Andijan State Medical Institute

E mail: salomovshoxabbosigro@gamil.com

Phone number: +998978374147

Abstract: The functional system of digestion and absorption, integrated with the immune system of the small intestinal mucosa, regulating the homeostasis of the internal environment of the body in the norm, is disrupted 1 hour after acute hemorrhagic shock, reperfusion of the ischemic limb of rabbits. The structures of enterocytes and microvilli, glycocalyx and supraepithelial layer of mucus are damaged. Their disintegration and inclusion of sIgA in their composition causes the translocation of microorganisms into the small intestinal mucosa. Correction with the antihypoxant succinylcholine effectively prevents ischemic damage to the intestine and the translocation of microorganisms and their toxins.

Keywords: small intestine, structure, enterocytes, epithelium, hemorrhagic shock, reperfusion.

INTRODUCTION

It is known that the mucous membrane of the small intestine, located on the border of the external and internal environments of the body, due to the presence of a huge number of microorganisms and their antigens, the intake of qualitatively and quantitatively unpredictable nutrients, has formed a perfect and adaptable functional system that ensures homeostasis. Thanks to it, under physiological conditions, digestion and absorption of nutrients are optimally carried out and regulated, the interaction and penetration of microorganisms and their antigens are prevented *1-4,14+. However, with some somatic diseases, shock, traumatic surgical interventions, the barrier-protective and homeostatic properties of the mucous membrane of the small intestine, the integration of its immune and digestive-absorption functions are disrupted *1,2,10,12,14+.

MATERIALS AND METHODS

Acute hemorrhagic shock was produced in 76 chinchilla rabbits weighing 2.6 ± 0.2 kg that had been fasted for 15 hours using a modified Wigger method [2]. Experimental procedure: animals were fixed to a machine, and the inguinal region was anesthetized locally with 0.5% novocaine solution. After surgical isolation of the right femoral artery, it was cannulated with a system of siliconized tubes filled with saline. Blood was released ($2.4 \pm 0.1\%$ of the animal's body weight) in fractions, every 15 minutes, according to the following schedule: 0.4; 0.3; 0.2; 0.1 parts of the total volume. Hemorrhagic shock is calculated from the moment the arterial pressure drops to 40 mm Hg. After reaching this level below the cannulation site of the femoral artery, a clamp was applied to it.

RESULTS AND DISCUSSION

One hour after hemorrhagic shock and restoration of blood flow in the ischemic limb, the mesentery and intestine look paler than in animals of group 1. The vessels of the mesentery, going from its root to the intestine, are collapsed. The small intestine is slightly swollen, in some areas it contains gases and chyme with mucus. Light-optically, no visible damage to the villi and crypts is noted in the jejunum and ileum. Only a large part of the goblet cells and Paneth cells are almost devoid of secretion, the supraepithelial layer of mucus is unevenly thickened, the capillaries of the stroma of the villi are spasmodic. Electron microscopically, the supraepithelial layer is polymorphic, has a fibrillar structure and includes, in addition to extruded cells, microorganisms. The water-electrolyte layer, glycocalyx and microvilli on the surface of the limbic enterocytes of

the villi do not have visible changes. The cytoplasm of the absorptive cells is heterogeneous: some are compacted, others are clear. In the former, the microvilli have a typical structure and have many endocytic vesicles between the bases. In their supranuclear cytoplasm, the Golgi complex is hypertrophied, the mitochondria are extended lengthwise with a moderately dense matrix and a number of cristae. In cleared enterocytes, the microvilli are in a state of partial or complete vesiculation, the number of ribosomes and polysomes is reduced, the mitochondrial matrix is clear, and the cristae are reduced. At their base or between enterocytes with a similar ultrastructure above the level of the basal membrane, as a rule, lymphocytes are determined, less often other leukocytes. If in rabbits of the 1st group interepithelial lymphocytes are single, then after 1 hour of shock and reperfusion of the ischemic limb, their number increases between enterocytes; neutrophils, eosinophils, macrophages, and rarely mast cells are also determined.

High rates of enterocyte renewal in the crypt-villus system require a constant blood supply, and with it, nutrients and energy. Oxygen is required for optimal utilization and provision of intensive processes in the small intestinal mucosa. If the nutrient and oxygen supply is disrupted, for example, due to acute hemorrhagic shock and reperfusion of the ischemic limb, the digestive-absorptive and immune functions of the small intestinal mucosa disintegrate. Despite the absence of a factor that has a direct effect on the small intestinal mucosa, shock and reperfusion, as can be seen from our data, damage the supraepithelial layer of mucus and other barriers integrating with it. As a result, there is a direct interaction of intestinal microorganisms with the glycocalyx and plasma membrane of the microvilli of enterocytes, their translocation into the cytoplasm and stroma of the villi of the small intestine.

CONCLUSION

1. Structural and functional damage to the supraepithelial layer of mucus, the mucous membrane of the small intestine in acute hemorrhagic shock and reperfusion causes direct interaction of microorganisms with the microvilli of enterocytes, their translocation into the absorptive cells and stroma of the villi, phagocytosis and digestion by macrophages, activation of immunocytes and other connective tissue cells.
2. Succinylated chitosan effectively prevents structural and functional damage to the mucous membrane of the small intestine and, as a consequence, the interaction of intestinal microorganisms with the glycocalyx and microvilli, their translocation into the internal environment.

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