Effect Of Energy Absorption On Morphological Changes In The Barrierprotective Structures Of The Gastrointestinal Tract In Chronic Kidney Diseases And Uremia

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ABSTRACT

The gastrointestinal tract is an active dynamic system that implements a number of important life support processes [30,2,57,87,203]. Impact of emergency situations on structures performing barrier-protective functions remain largely unexplored. In recent years, ideas about the role of the epithelium of the digestive tract have expanded, significant, and it has been found to be significant in loss of renal function [7,8,2]. Therefore, the epithelium of the gastrointestinal tract should be considered not only as a selective barrier to penetration of toxic substances into the interstitium, but also a key link in decontamination endogenous intoxication. The barrier-protective function of the digestive system is associated with border tissues, which have a strict morphological organization. These phenomena are as diverse as nature physical, chemical or biological aggression on the internal environment of the body can be various. Process layering data is important for practical use of the phenomenon of "layered defense", when stress or damage increases secretion and secretion, increases proliferation and desquamation of the epithelium, as well as migration of neutrophilic leukocytes from the bloodstream to the epithelial layer. [22].

Under normal conditions, the gastrointestinal tract is considered a balanced ecosystem where there is a certain habitat - physical space. The state of the mucous membrane of the small intestine, the main participant in the absorption processes and excretion of metabolic products deserves the most attention. It has been found that oral administration enterosorbents (Karbovit and Polypefan) in intact rats does not cause mucosal changes membrane of the jejunum and ileum and does not affect the state of enterocytes. However, in some in some cases, ES enhances the secretion of goblet cells, especially in the apical part of the intestinal villi [22]. At the same time, a relatively large number of Pannet cells were registered in crypts, and Lamina the propria around them contains many immune-competent plasmocytes and neutrophils. Electron microscopic examination of various parts of the small intestine showed that when mercury chloride injected into rats, the number of microvilli of enterocytes was shortened and decreased, and the number mitotically dividing cells increased. In this case, there is a pronounced vacuolization of enterocytes observable; vacuoles glow with electronics and contain flocculent material. Side cells emptied, Pannett cells contain a small amount of secretory...
granules. Characteristic feature of the effect of mercury poisoning on the mucous membrane of the small intestine is inhibition of the reaction of cells of the connective base. The protective barrier of the mucous membrane of the gastrointestinal tract consists of three levels: luminal, epithelial and connective tissue.

1. INTRODUCTION:

The luminal level is presented above the epithelial layer of mucous deposits, consisting of multicomponent mucus, secretions of the digestive glands, substances secreted by Paneth cells, etc. components. Intoxication with salts of heavy metals, pesticides and other toxic substances exogenous and endogenous nature can cause profound disturbances, up to the loss of the epithelial layer [63, 64, 72, 91]. The connective tissue level has three characteristics; first, the extraordinary intensity of bilateral metabolic processes between the stroma and the epithelial lining, secondly, due to the content of significantly greater number of connective tissue cells, and thirdly, due to the formation of immune processes. Studies have confirmed that uremia poisoning in chronic renal failure occurs when constant penetration of UT into the bloodstream. This is also evidenced by chronic mercury intoxication experimental animals in which hemodynamic, dystrophic, focal, necrotic, inflammatory and sclerotic changes are observed [87]. However, we did not observe a clear idea of the sequence morphological restructuring of the hematocirculatory bed and tissue structures of the gastrointestinal tract. Without clarifying this mechanism, it is impossible to imagine the morphological basis of damage and Renoprotective role of ES in uremic aggression.

In our study, the duration of monitoring of the barrier-protective function of the gastrointestinal tract the path of the experimental animals was 3 months. The first data were obtained after 14 days of the experiment. Inflammatory and destructive changes in tissue structures, especially in the lining of the small intestine, as indicated intercellular swelling and in places thinning of enterocytes. Condition of microvessels: heterogeneous - together with blood, precapillaries and capillaries were spasmodically narrowed observed, as well as without vascular zones and extravasates. Diameter of precapillaries, capillaries, and postcapillaries of the mucous membrane in all parts of the small intestine exceeded the control by 26.5%, 43.7% and 43.8%, respectively (Fig. 1).
Figure: 1 CKD. Uremia 14 days. Inflammatory destructive changes, intercellular edema, stagnation and vasodilation. SEMx500

On the 30th day after poisoning with mercury salts, destructive-dystrophic changes in all small intestinal membranes are strengthened. This is evidenced by dormant and thinned capillaries and eccentrically located villi. Ulcers of various sizes are found on the surface of the mucous membrane. With 60-day uremia, the revealed disorders were aggravated. All wall membranes small intestine more distinctly thinned, degeneration intensified in the mucous layer, and the number of short villi increased. The lumen of the crypts became tortuous, areas with peeling the epithelial layer, microerosion appeared in the stroma (Fig. 4.2). It was an increase in the number of avascular zones and a decrease in the density of the vessel walls. These changes were characteristic of structural disorders of the duodenum.

Figure: 2 CKD. Uremia 30 days. Increase in the number of avascular zones, decrease the density of the walls of blood vessels. Edema. SEMx500

On the 90th day of poisoning, the density of blood vessels decreased to 63% of the control. In capillaries, congestion and varicose veins. In general, everything points to a deep oppression of the hemocirculatory bed and morpho functional state of the small intestine. Distribution features of extra- and intraorganic vessels in various parts of the colon indicate differentiated severity of pathological processes that negatively affect normal functioning colon. In the early period (5-14 days) after sowing rats with mercury nitrate, inflammatory-reactive changes appear, accompanied by edema of all layers of the colon and basal intestines cells; diffuse infiltration; swelling of cells and eccentric arrangement of their nuclei or spasmodic vasoconstriction, their tortuosity and extravasation indicate a violation of vascular permeability vessel wall. The crypt stroma is edematous, with the presence of lymphoid and plasma elements.(Fig. 5.3). Throughout the colon, the epithelial layer of the crypts is stratified in places.
Erosive or ulcerated areas are found in the proximal and middle sections. Due to edema the thickness of the intestinal membranes significantly exceeds the norm by 1.2 times. Crypts cylindrical, the lumen of which is filled with mucus, there is swelling of the stroma, infiltration and expansion of intercellular gaps. In the subsequent periods of the experiment, deep atrophic processes develop, which as evidenced by the thinning of all layers of the colon wall, a decrease in the epithelial, goblet and mitotic dividing cells. Microvessels are completely dilated or narrowed, which indicates the continuation of the vascular dystonia. Capillaries in all membranes are aneurysmically dilated, venous stasis persists, which contributes to the progression of tissue hypoxia (Fig. 4).
Further, the picture of micro angiosclerosis develops. The number of epithelial and mitotic dividing cells decreased by 35-39% compared to control. These phenomena are more pronounced in proximal intestine. Consequently, one of the main factors in the development of atrophic processes in chronic renal failure, insufficiency, and CSS is hypoxia associated with capillary congestion and venous congestion (Fig. 5). These disorders lead to metabolic damage to the tissue structures of the small and large intestine, causing the severity of chronic renal failure and thus largely determine the outcome of uremic aggression.

Figure: 5 CKD. Uremia 90 days. Capillarostasis and venous congestion. SEMx500

Analysis of the results of lesions of the gastrointestinal tract mucosa with mercury intoxication allows development of nephroprotection measures, taking into account the possibility of different areas gastro intestinal tract to remove Ut. It has been established that the role of the stomach in early stages of chronic renal failure replacing Ut elimination are apparently suppressed as you progress. The concept of the morphological features of the barrier-protective function of the digestive system tract in chronic renal failure and uremic intoxication allows you to establish the ratio of factors of aggression(Ut) and protective factors (enterosorbents), as well as to develop optimal treatment tactics chronic renal failure. It should be noted that Ut secreted in the digestive tract is absorbed already in the initial section, jejunum. Further, with blood and lymph, these substances are transferred to tissues that form intestinal barrier and glands, the secretions of which enter the digestive tract. Through the intestinal barrier, digestive juices, endogenous toxins again enter the intestines. Further recirculation coefficient characterizing the ratio of the amount of UT entering the gastrointestinal tract to its mass, absorbed in the initial part of the jejunum, exceeds 100%. This means that part metabolites that are absorbed by the lower intestine are involved in processing. Equilibrium the internal environment in these cases is maintained by creating a correspondence between absorption the rate Ut and the rate of their utilization and deposition. Physiological disposal processes intestinal metabolites are a key factor for inclusion in the treatment packages [30].Oral administration of enterosorbents Karbovit and Polyhepan to laboratory animals, showed their effectiveness in the absence of any negative changes in organ tissues. Login to the intestine, sorbents bind Ut and increase the luminal level of protection and regeneration processes, as evidenced by the appearance of full-blooded areas
of the mucous membrane with relatively normal structure (Fig. 4; 6; 7).

![Figure 6](image)

Figure: 6 CKD. Uremia. Enterosorption. Increased infiltration by macrophages and leukocytes SEMx500.

Carbovite actively absorbed toxic metabolites and more effectively blocked Ut than polypepam. Excretion of Ut increases the intensity of the reaction of connective tissue elements tissue, increases the number of lymphocytes, macrophages and eosinophils, as well as foci with edematous changes. In general, there is a tendency towards normalization of structures that create protective barrier (5.7) At the same time, the three-level principle of the organization's functioning persists throughout the gastrointestinal tract.

![Figure 7](image)

Fig. 7. CKD. Uremia. Enterosorption. Polypefan. An increase in the number of lymphocytes and mitotic cells. SEMx500.
Thus, oral administration of mercury chloride to experimental rats creates an adequate model of CKD and uremic syndrome, and also manifests itself in the corresponding disorders barrier-protective structure of the organs of the gastrointestinal tract. The use of enterosorbent "Karbovit" significantly blocks the changes caused by uremic poisoning, while the morphological picture epithelial cells are close to normal, and the structures of the connective tissue base are activated. Nevertheless, further study of the processes of structural protection of the mucous membrane of the gastrointestinal tract requires further study.is necessary for the development of new approaches to ET of progressive kidney disease.
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