International Journal of Advanced Science and Technology
Vol. 29, No. 11s, (2020), pp. 3202-3207

Effect Of Enerosorption On Morphological Changes Of The Barrier-Protective Structures Of The Gastrointestinal Tract In Chronic Kidney Diseases And Uremia

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The gastrointestinal tract is an active dynamic system that implements a number of important life support processes [30,2,57,87,203]. The effects of ES on structures performing barrier-protective functions remain virtually unstudied.

In recent years, the idea of the role of the epithelium of the digestive tract has expanded significantly and it has been established that it is significant in the loss of renal function [7,8,2]. In this regard, the gastrointestinal epithelium should be considered not only as a selective barrier to the penetration of toxic substances into the interstitium, but also a key link in the deactivation of endogenous intoxication.

The barrier-protective function of the digestive system is associated with borderline tissues that have a strict morphological organization. These phenomena are diverse in the same way that the nature of physical, chemical or biological aggression on the internal environment of an organism can be different. The data on the multi-level nature of the processes is important for the practical use of the "layered defense" phenomenon, when stress or damage intensifies secretion and secretion, increases the proliferation and desquamation of the epithelium, and also the migration of neutrophilic leukocytes from the bloodstream into the epithelial layer. [22] .

Under ordinary conditions, the gastrointestinal tract is considered as a balanced ecosystem in which there is a certain habitat - the physical space.

The condition of the small intestinal mucosa, the main participant in the processes of absorption and excretion of metabolic products, deserves the most attention. It was found that oral administration of enterosorbents (Karbovit and Polypefan) to intact rats does not cause alteration of the mucous membrane of the jejunum and ileum and does not affect the state of enterocytes. Nevertheless, in some cases, ES enhances the secretion of goblet cells and especially in the apical part of the intestinal villi. [22]. At the same time, a relatively large number of Pannet cells were recorded in crypts, and Lamina propria around them contains many immunocompetent plasmocytes and neutrophils. Electron microscopic studies of various parts of the small intestine found that when mercury chloride was administered to rats, the number of microvilli of enterocytes was shortened and reduced, and the number of mitotically dividing cells increased. At the same time, pronounced vacuolization of enterocytes is observed; vacuoles are electronically bright and contain flocculent material. The goblet cells are emptied, in the Pannet cells contains an insignificant number of secretory granules. A characteristic feature of the effect of mercury poisoning on the mucous membrane of the small intestine is the inhibition of the reaction of the cells of the connective base.

The protective barrier of the gastrointestinal mucosa consists of three levels: luminal, epithelial and connective tissue.

The luminal level is presented above the epithelial layer of mucous deposits, consisting of multicomponent mucus, digestive gland secrets, substances secreted by Pannet cells and other components. Intoxication with salts of heavy metals, pesticides and other toxic substances of exo- and endogenous nature can cause deep disturbances, up to the loss of the epithelial layer [63,64,72,91].

The connective tissue level has three features; firstly, by the extreme intensity of bilateral metabolic processes between the stroma and epithelial lining, secondly, by the content of a significantly larger number of connective tissue cells, and thirdly, by the formation of immune processes.

Studies have confirmed that uremic poisoning with chronic renal failure occurs with the constant penetration of UT into the bloodstream. This is also evidenced by chronic mercury intoxication of 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 of morphological rearrangement of the hemocirculatory bed and gastrointestinal tissue structures. Without clarifying this mechanism, it is impossible to imagine the morphological basis of damage and the renoprotective role of ES in uremic aggression.

In our study, the duration of monitoring the barrier-protective function of the gastrointestinal tract of experimental animals was 3 months.

The first data were obtained after 14 days of the experiment. Inflammatory-destructive changes in tissue structures were noted, especially in the mucous membrane of the small intestine, as indicated by intercellular swelling and, in some places, thinned enterocytes. The state of microvessels is heterogeneous - along with blood-filled, spasmatically narrowed precapillaries and capillaries were observed, as well as without vascular zones and extravasates. The diameter of precapillaries, capillaries, and postcapillaries of the mucous membrane in all sections of the small intestine exceeded the control by 26.5%, 43.7%, and 43.8%, respectively (Fig. 1).

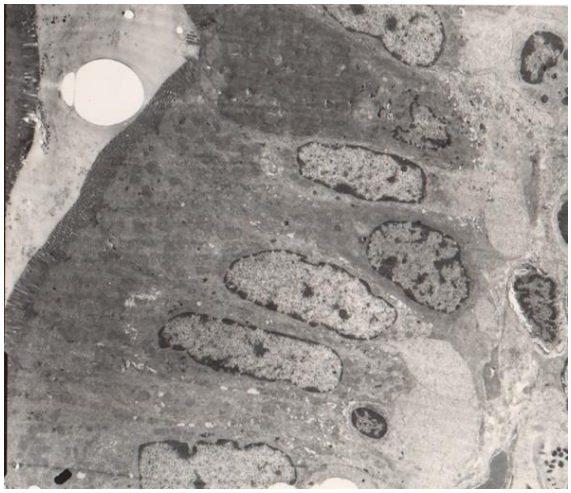

Fig. 1 CKD. Uremia 14 days. Inflammatory destructive changes, intercellular edema, stagnation and vasodilation. SEMx500

On the 30th day after poisoning with mercury salts, destructive-dystrophic changes in all small intestine shells intensified. This is evidenced by slept and thinned capillaries and eccentrically located villi. Various sizes of ulcers are found on the surface of the mucosa.

With 60 day uremia, the identified violations were exacerbated. All the membranes of the walls of the small intestine were thinner more distinctly, degeneration intensified in the mucous layer, and the number of short villi increased. The lumen of the crypts became sinuous, areas with desquamation of the epithelial layer were revealed, microerosion appeared in the stroma (Fig. 4.2). There was an increase in the number of avascular zones and a decrease in the density of the walls of blood vessels. These changes were also characteristic of structural disorders in the duodenum.

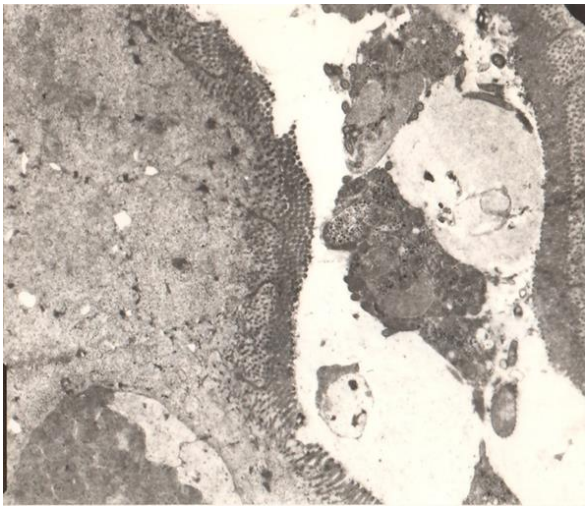

Fig. 2 CKD. Uremia 30 days. An increase in the number of avascular zones, a decrease in the density of the walls of blood vessels. Edema. SEMx500

On the 90th day of poisoning, the vascular density decreased to 63% of the control. In the capillaries, stagnation and varicose expansion were detected. In general, everything indicates a deep inhibition of the hemocirculatory bed and the morphological and functional state of the small intestine.

Features of the distribution of extra- and intraorgan vessels in various parts of the colon indicate a differentiated degree of severity of pathological processes that adversely affect the normal functioning of the large intestine. In the early period (5-14 days) after seeding rats with mercury nitrate, inflammatory-reactive changes appear accompanied by swelling of all layers of the colon and basal cells; diffuse infiltration; swelling of the cells and the eccentric arrangement of their nuclei or spasmodic narrowing of the vessels, their tortuosity and extravasation indicate a violation of the permeability of the vessel wall. Crypt stroma is edematous, with the presence of lymphoid and plasma elements (Fig.5.3). Throughout the colon, the epithelial layer of crypts is exfoliated in places.

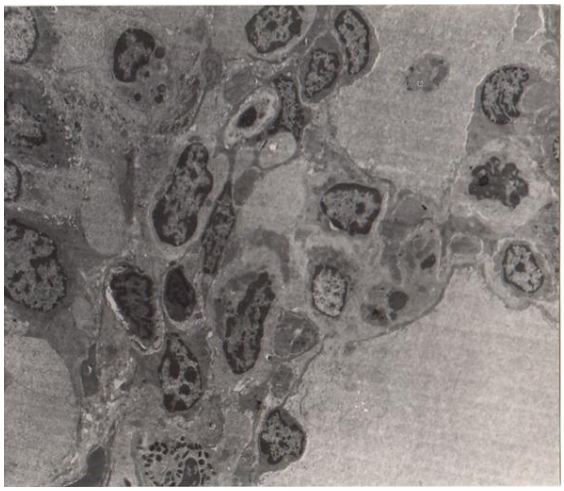

Fig. 3 CKD. Uremia 90 days. Large intestine. Vasoconstriction, diffuse infiltration and extravasates. Swelling of all layers and basal cells. SEMx500

Erosive or ulcerated areas are detected in the proximal and middle sections. Due to edema, the thickness of the intestinal membranes significantly exceeds the norm by 1.2 times. Crypts of a cylindrical shape, the lumen of which is filled with mucus, there is stromal edema, infiltration and expansion of intercellular clefts

In the subsequent periods of the experiment, deep atrophic processes develop, which are evidenced by the thinning of all layers of the colon wall, a decrease in epithelial, goblet and mitotically dividing cells. The microvessels are dilated or narrowed throughout, indicating continued vascular dystonia. The capillaries in all membranes are aneurysmically dilated, venous congestion persists, which contributes to the progression of tissue hypoxia (Fig. 5.4).

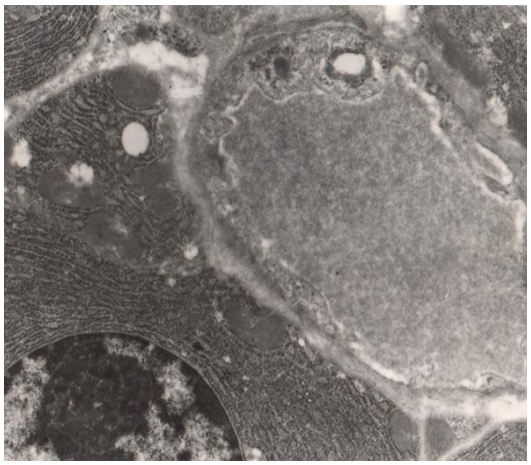

Fig. 4 CKD. Uremia 90 days. A decrease in the number of epithelial, side-shaped and mitotically dividing cells. Infiltration and swelling of the stroma.

Further, a picture of microangiosclerosis develops. The number of epithelial and mitotically dividing cells decreased by 35-39%. compared to control. These phenomena are more pronounced in the proximal intestine.

Consequently, one of the main factors in the development of atrophic processes in chronic renal failure and CSS is hypoxia associated with capillary stasis and venous congestion (Fig. 5.5). These disorders lead to metabolic damage to the tissue structures of the small and large intestines, causing the severity of chronic renal failure and thereby significantly determine the outcome of uremic aggression.

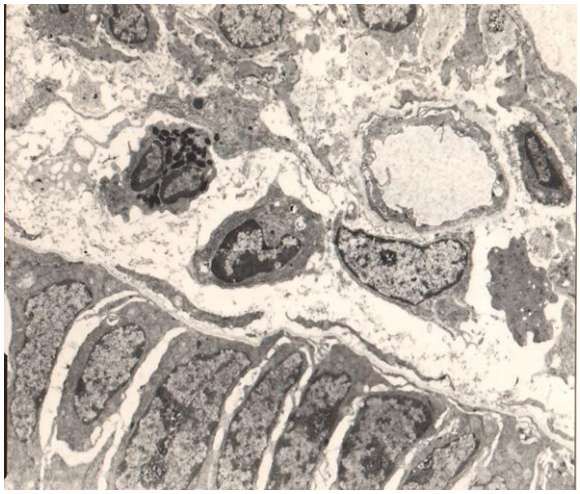

Fig. 5 CKD. Uremia 90 days. Capillarostasis and venous congestion. SEMx500

Analysis of the results of gastrointestinal mucosal lesions during mercury intoxication allows the development of measures of nephroprotection, taking into account the ability of various sections of the gastrointestinal tract to excrete Ut. It has been established that the role of the stomach in the early stages of chronic renal failure, which is vicarious in terms of elimination of Ut, appears to be inhibited as it progresses. The concept of morphological features of the barrier-protective function of the digestive tract in chronic renal failure and uremic intoxication allows us to establish the ratio of aggression factors (Ut) and protective factors (enterosorbents), as well as to develop the optimal treatment tactics for chronic renal failure.

It should be noted that Ut released in the digestive tract is absorbed already in the initial section of the jejunum. Further, with blood and lymph, these substances are transferred to the tissues that form the intestinal barrier and to the glands, the secrets of which enter the digestive tract. Through the intestinal barrier in the digestive juices, endogenous toxins again enter the intestines. Further, the recirculation coefficient characterizing the ratio of the amount of UT entering the gastrointestinal tract to its mass absorbed in the initial section of the jejunum exceeds 100%. This means that part of the metabolites that are absorbed by the lower intestines is involved in recycling. The equilibrium of the internal environment in these cases is maintained by creating a correspondence between the absorption rate of Ut and the speed of their utilization and deposition. The processes of physiological recycling of metabolites in the intestine are a key factor for inclusion in the complex of therapeutic measures Es [30].

Oral administration to the laboratory animals of the enterosorbents Karbovit and Polyphepan showed their effectiveness in the absence of any negative changes in the tissues of organs. Entering the intestines, the sorbents bound Ut and increased the luminal level of protection and regeneration processes, as indicated by the appearance of full-blooded sections of the mucous membrane with a relatively normal structure (Fig. 5.4; 5.6; 5.7).

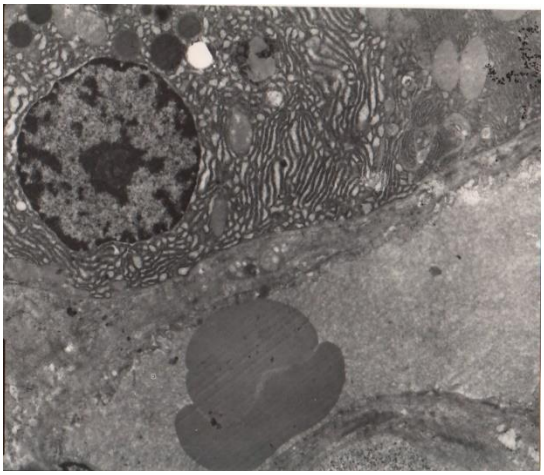

Fig. 6 CKD. Uremia. Enterosorption. Strengthening macrophage and leukocyte infiltration SEMx500.

Karbovit actively absorbed toxic metabolites and was more effective in blocking Ut than polypepam. Excretion of Ut increases the intensity of the reaction of the elements of the connective tissue, increases the number of lymphocytes, macrophages and eosinophils, as well as foci with hydropic changes. In general, there is a tendency toward normalization of structures that create a protective barrier (5.7). At the same time, the three-level principle of the functioning of the organization is maintained throughout the gastrointestinal tract.

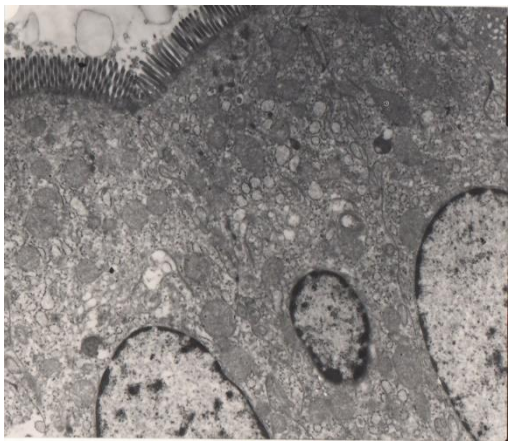

Fig.7. CKD. Uremia. Enterosorption. Polypefan. An increase in the number of lymphocytes and mitotic cells. SEMx500.

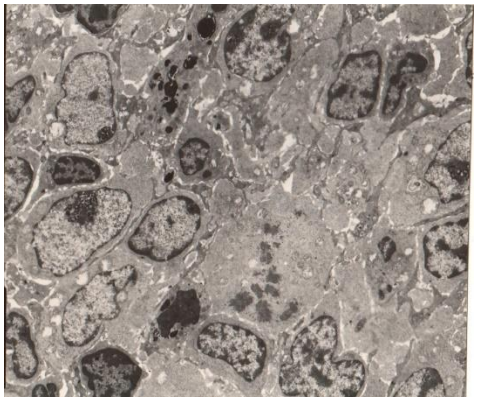

Fig. 8. Chronic renal failure. Uremia. Enterosorption. Carbovit. Thinning and fragmentation of the basement membrane. Expansion of blood vessels. SEMx500.

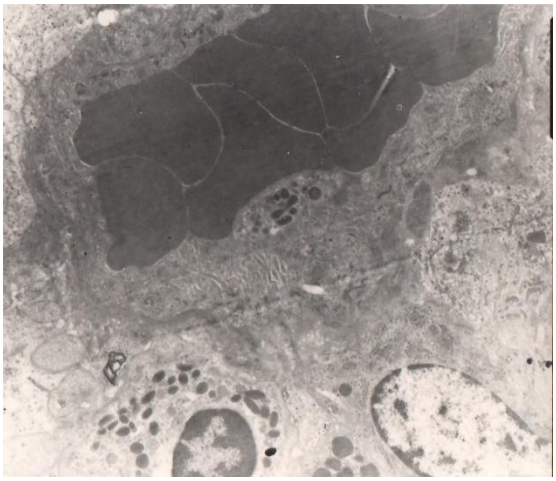

Fig. 9 CKD. Uremia. Enterosorption. Carbovit. Restoration of the tissue structure of the intestine. Thickening of the walls of blood vessels and plethora of the mucous membrane. SEMx500.

Thus, the oral administration of mercury chloride to experimental rats creates an adequate model of CKD and uremic syndrome, as well as manifests itself in corresponding violations of the barrier-protective structure of the gastrointestinal tract organs. The use of enterosorbent “Karbovit” significantly blocks the changes caused by uremic poisoning, while the morphological picture of epithelial cells approaches normal, and the structures of the connective tissue base are activated. Nevertheless, further study of the processes of structural protection of the gastrointestinal mucosa is necessary for the development of new approaches to the ET of progressive kidney diseases.