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CHANGES IN THE GASTRIC MUCOSA IN THE EARLY STAGES OF CHRONIC RENAL FAILURE IN RATS

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KEY WORDS: renal failure; gastric mucosa; rats.

The nature and mechanism of formation of pathology of the gastric mucosa during chronic renal failure (CRF) (uremic gastritis) have not yet been explained [3, 6], so that a study of this problem from the experimental aspect is essential.

To determine the precise mechanisms of action of CRF in the early stages of its formation on the gastric mucosa, in the investigation described below a series of chronic experiments were carried out on rats. Special attention was paid to changes in the stomach during initial disturbances of kidney function, which have hardly been studied at all under clinical conditions.

EXPERIMENTAL METHOD

Altogether 30 mature rats of both sexes were used; under ether anesthesia a two-stage subtotal nephrectomy was performed through an extraperitoneal approach. Material for examination, consisting of pieces of the mucosa from the fundal and antral portions of the stomach, was taken 1, 4, and 6 months after the operation. Sections 5 μ thick, stained with hematoxylin and eosin, and by the Dominici–Kedrovsky method in Samsonov's modification [4], were used for histological investigation. For histochemical investigation pieces of stomach tissue frozen in liquid nitrogen were used. The following oxidoreductases — succinate dehydrogenase (SDH), lactate dehydrogenase (LDH), glucose-6-phosphate dehydrogenase (G6PDH), and monoamine oxidase (MAO), and the hydrolases acid phosphatase (AP) and ATPase — were studied in frozen sections 10 μ thick. To determine the distribution of neutral mucopolysaccharides the PAS reaction was used. Quantitative assessment of activity of these enzymes was carried out with the MUF-5 cytospectrophotometer. Activity of the enzymes was calculated from mean values obtained by the study of 100-150 cells. The results were subjected to statistical analysis. The degree of development of CRF was estimated by the increase in the blood serum urea level from 4.4 ± 0.2 mM in the control to 7.0 ± 0.5 mM after 1 month, to 10.0 ± 0.5 mM after 4 months, and to 8.5 ± 0.4 mM 6 months after the operation.

EXPERIMENTAL RESULTS

In all nephrectomized animals hyperplasia of the mucosa of the fundal portion of the stomach developed by the sixth month on account of an increase in the number of parietal and chief cells (in the normal gastric gland there are 42.0 ± 4.0 chief and 19.5 ± 3.0 parietal cells, but by the end of the experiment there were 80.5 ± 10.0 and 34.5 ± 4.0, respectively). This phenomenon is accompanied, as the data showed indirectly, by an increase in gastric secretion, as shown by an increase in SDH activity and a decrease in LDH activity (parietal

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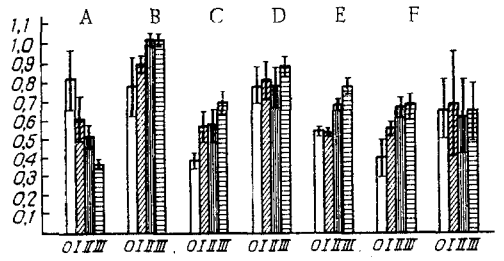

Fig. 1. Activity of enzymes in cells of principal gastric glands. 0) Control, I) 1 month, II) 4 months, III) 6 months after operation. A) LDH, B) SDH, C) MAO-1, cell population with low enzyme activity, D) MAO-2, cell population with high enzyme activity, E) ATPase, F) AP, G) α-glycerophosphate dehydrogenase (αGPDH). Ordinate, optical density (in relative units).

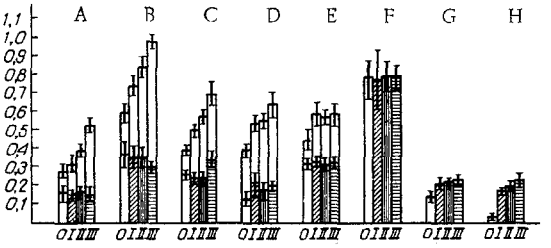

Fig. 2. Enzyme activity in cells of antral glands of gastric mucosa. A) G6PDH, B) LDH, C) SDH, D) GPDH, E) MAO, F) ATPase-1 (in capillary endothelium), G) ATPase-2 (in cytoplasm of cells of lower portions of glands), H) AP. Shaded part of columns denotes enzyme activity in cells of upper part of antral glands. Remainder of legend as to Fig. 1.

cells), an increase in AP activity (chief and parietal cells), and also an increase in the content of PAS-positive material in the cytoplasm of the cells and in the lumen of the glands (Fig. 1).

The most marked trend in enzyme activity in the antral portion of the stomach (Fig. 2) was observed in the cell population located in the middle third of the mucosa, where endocrine cells of the G, EcL, and Ee types [1, 5] are mainly concentrated. The increase observed in activity of the oxidoreductases (G6PDH, SDH, LDH, αGPDH) and hydrolases (AP, ATPase) undoubtedly indicates intensification of synthesis and secretion processes in these cells in the initial stages of experimental CRF, but which would be very difficult to analyze under clinical conditions.

The scheme in Fig. 3 shows hypothetical pathways of interconnection between the various pathological disturbances. In the early stages of CRF the dominant role is evidently played by the vicarious function of the stomach, which plays a more active part than in the healthy individual in the excretion of nitrogenous waste products into the lumen of the organ. Excess ammonia formation sharply increases the pH of the gastric contents, and this is accompanied by activation of acid formation and gastrin production by G cells. Later a working hyperplasia of the gastric cells regularly develops. Meanwhile, to protect the mucosa from injury by urea and its hydrolysis products, there is a considerable increase in mucus production, as is confirmed by changes in the content of PAS-positive substances in the cytoplasm of the cells, and also by increased MAO activity (Figs. 2 and 3) in the mucosa — this enzyme hydrolyzes serotonin, one of the chief regulators of mucus formation in the stomach [2].

With progression of CRF, these adaptation mechanisms probably eventually fail, and the profound pathology of the stomach develops which, under clinical conditions, can be diagnosed in patients in the terminal uremic stage of renal failure [3]. The formation of atrophic

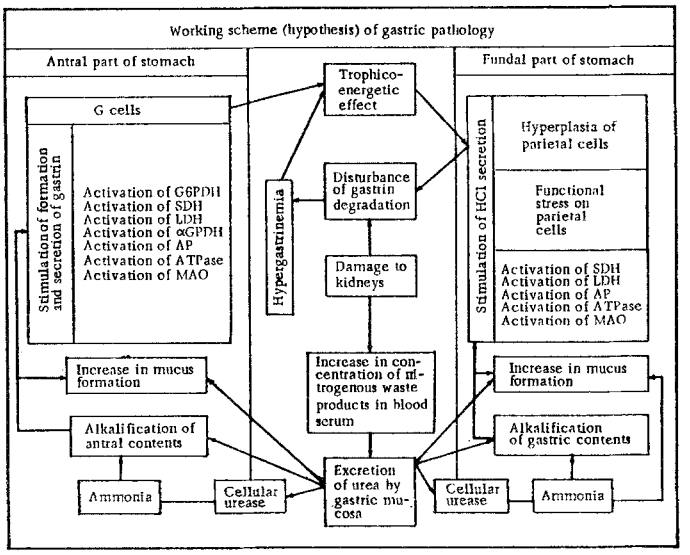

Fig. 3

gastritis against the background of previous hyperplasia of the glandular structures can evidently explain the unique "mosaic" character of the structural changes and the "paucity" of the functional parameters in patients with CRF.

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FUNCTIONAL AND MORPHOLOGICAL FEATURES OF THE ADRENAL CORTEX OF ALBINO RATS AFTER LONG EXPOSURE TO STYRENE

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KEY WORDS: aromatic hydrocarbons; metapyrone; quantitative functional-morphological analysis; adrenocortex of albino rats.

According to some observations [5, 7] aromatic hydrocarbons (AH) have a toxic action on cells of the adrenal cortex. There have been few histochemical investigations of the chronic effect of AH on the adrenal cortex, and in none of them was a quantitative analysis made of the character of changes in the adrenal cortex in response to exposure to AH.

Considering the wide use of AH in modern industry and probability of injury to the endocrine system arising in persons working in processes associated with AH [1, 4], it was decided to study the possible mechanism of action of these compounds on the adrenal cortex, with particular reference to styrene.

The object of this investigation was to determine the mechanism of long-term action of styrene on the albino rat adrenal cortex.

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