
MORPHOLOGY AND PATHOMORPHOLOGY

Quantitative Characterization of Enterochromaffin Cells of the Pyloric Part of the Stomach in Experimental Duodenal Stasis

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Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol. 123, No. 3, pp. 336-337, March, 1997
Original article submitted January 22, 1996

Morphological changes in the enterochromaffin cells of the pyloric part of the stomach are studied in rats with experimental duodenal stasis. Morphometrical and mathematical characteristics of endocrinocyte secretion and elements of the information theory were used. Depletion of the secretory activity of enterochromaffin cells is a mechanism responsible for reflux gastritis in duodenal stasis.

Key Words: *enterochromaffin cells; stomach; duodenal stasis*

Duodenogastral reflux is an important factor contributing to the development of postgastroresection and postvagotomic syndromes, reflux gastritis, and ulcer formation [1]. Reflux gastritis is found in 33.7% of gastroenterological patients [4] and is most often associated with duodenal stasis. Numerous effects exerted by the enterochromaffin (EC) cell serotonin on digestive organs [3] and insufficient data on the morphogenesis of gastric changes in duodenogastral reflux [2] prompted us to examine EC cells of the pyloric portion of the stomach in experimental duodenal stasis.

MATERIALS AND METHODS

Experiments were carried out on 82 adult male rats. Duodenal stasis was induced by an original method: partial standard narrowing of the distal portion of the duodenum with a polyvinyl chloride ring. Surgical interventions and sacrifice (on days 3, 14, 21, 30, and 60 of experiment) were carried out under ether narcosis at the same hours of the day (between 15:00

and 17:00). Histological preparations were stained with hematoxylin and eosin and basic brown, and PAS reaction was performed. Enterochromaffin cells were identified by impregnation by the methods of Grimelius, Lillie, and Pasqual and by Schmorl's ferricyanide method. The cells were counted in the standard field of view, and their number per mm² and relative number of cells with different density of granules were calculated. The cells were divided into 4 types with different numbers of granules: I) with the minimal, II and III) with medium, and IV) with the maximum content. The coefficient of granulation (CG) was calculated as the quotient of the sum of products of the cell counts and the number of their type and the total count (at least 100 cells). Informative values were as follows: absolute (*H*) and relative (*h*) entropy, excess coefficient (*R*), and informative index of the effect (IIE). The data were statistically processed using Student's *t* test.

RESULTS

In control rats, the structure of EC cells varied within a wide range, which was manifested as a high entropy

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TABLE 1. Count of Enterochromaffin Cells in the Pyloric Part of the Stomach in Health and Duodenal Stasis

Time of investigation, day	Parameters					
	cell count	CG	H	h	R	IIE
Control	145.2±4.6	2.0±0.1	1.8353	91.8	8.2	—
3	119.0±5.9	1.8±0.1*	1.6142	80.7	19.3	0.1205
14	47.3±3.5	1.5±0.1	1.2839	64.2	35.8	0.3004
21	101.8±3.9	1.9±0.1*	1.7837	89.2	10.2	0.2811
30	115.0±3.4	1.7±0.1*	1.6004	80.0	20.0	0.1280
60	35.6±2.7	1.3±0.1	1.1620	58.1	41.9	0.3669

Note. *The difference is statistically insignificant.

(Table 1). This reflects normal asynchronous pattern of the secretory cycle, in which cells in the phase of extrusion of the endocrine granules predominate (38% of type I cells and 33% of type II cells).

In experimental duodenal stasis the number of these cells decreased as early as on day 3 ($p < 0.001$), which is caused by increased degranulation. An 11.1% decrease in entropy indicates an increase in secretory activity of endocrinocytes and synchronization of secretory cycle. The CG, however, does not significantly differ from the control. Its decrease becomes statistically significant only by the end of the second week of experiment ($p < 0.002$). The count of detected endocrinocytes decreased by 67.4% at this period.

After 3 weeks, changes in EC cells somewhat stabilized. Their count was 29.9% lower than in the control, although the difference was still significant ($p < 0.05$). The CG better characterizes the secretory activity of EC cells. It is almost normal on day 21, when the count of cells in the phase of secretion and accumulation (types III and IV) is almost normal. The period of stable secretory activity lasts up to 30 days, when decreased mucus production by the integumentary epithelium and leukocytic infiltration of the gastric mucosal layer proper are observed.

After 2 months, duodenal stasis leads to decompensation of EC cells, which is seen from the 75.5% decreased count of endocrinocytes, a significant ($p < 0.001$) decrease in CG, and a 33.7% drop in their structural variations (Table 1). The wave-like

course of changes in IIE reflects the initial period of mobilization of EC cells (days 3-14), followed by stabilization of their secretory activity (days 21-30) and its depletion (day 60).

Thus, a stable increase in intraduodenal pressure in duodenal stasis leads to a gradual depletion of the secretory activity of EC cells in the pyloric part of the stomach, which is compensated for a short time after the initial period of degranulation. This may be caused by increased pH of the gastric contents under conditions of congestion [4], which results in insufficiency of the natural stimulating effect of the hydrochloric acid. Reduced serotonin secretion by EC cells leads to a decrease in the mucus production, lowers protective activity of the epithelium, and induces gastric changes [3]. Inadequate level of this bioamine secretion may augment the motor activity of the intestinal wall and result in the formation of the vicious circle with duodenal stasis and bioamine insufficiency of the duodenum.

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