

Laser-Induced Clasmatosis of Gastric and Intestinal Epitheliocytes

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Clasmatosis, a possible cell response to laser treatment, can be induced in gastric mucosa by copper-vapor laser and nitrogen laser radiation administered in a dose of 20 and 4 J/cm², respectively. Clasmatosis is most pronounced in parietal cells. In the intestine, clasmatosis is observed in *lamina propria* connective tissue cells. Treatment with copper-vapor and nitrogen lasers in doses higher than 34 and 6 J/cm², respectively, less frequently causes clasmatosis in the intestine and gastric mucosa.

Key Words: *clasmatosis; laser; epitheliocytes*

A limited set of reactions to a variety of external stimuli is one of the most remarkable features of living organism. This universal property is intrinsic to the whole organism and down to subcellular structures [6].

Cells and subcellular structures are characterized by limited number of standard reactions to various stimuli, for instance, clasmatosis.

Clasmatosis (clasma in Greek means fragment) was first described by Ranvier in 1880 as separation of cytoplasmic fragments from cell surface. Electron microscopic features of clasmatosis were described by A. Policard and M. Bessis [7]. Clasmatosis and microclasmatosis are considered as phenomena of cell pathology [1].

Clasmatosis induced by low intensity laser irradiation (LILI) [4,5] has not been studied.

Our more than 15-year experience in investigating the effect of various LILI on cells [2-5] allows us on the basis of electron microscopic observation of the gastric, duodenal, jejunal, and ileal mucosa to consider LILI as inductor of clasmatosis.

MATERIALS AND METHODS

Gastric, duodenal, jejunal and ileal mucosa of Wistar rats (120-140 g) was treated with a copper-vapor

(CVL) or UV nitrogen laser (UVL) either through the serosa (laparotomy) or directly (endoscopically) using a light guide connected to ILGI-101 or LGI-21 devices, respectively. Treatment with the CVL was performed for 1, 3, and 5 min and the corresponding doses were 7, 20, and 34 J/cm², while the duration of treatment for UVL was 1, 2, and 3 min and the doses were 2, 4, and 6 J/cm², respectively. Gastric and intestinal mucosa of control animals were treated with white light or noncoherent light with analogous wavelength and intensity. Mucosa samples from 210 animals were analyzed.

All painful procedures were carried out under ether anesthesia. The animals were decapitated 5 min, and 1, 8, 24, 48, and 96 days after single irradiation. Samples of gastric and intestinal mucosa were fixed in 2.5% glutaraldehyde on phosphate buffer, dehydrated, and embedded in Epon-Araldite. Ultrathin (double contrast) and semithin sections were examined in a Hitachi-H600 electron microscope.

RESULTS

Our previous studies [4,5] showed that doses 7 and 20 J/cm² for CVL and 2 J/cm² for UVL are optimal and induce pronounced changes attesting to stimulation of proliferation and metabolic processes. Higher

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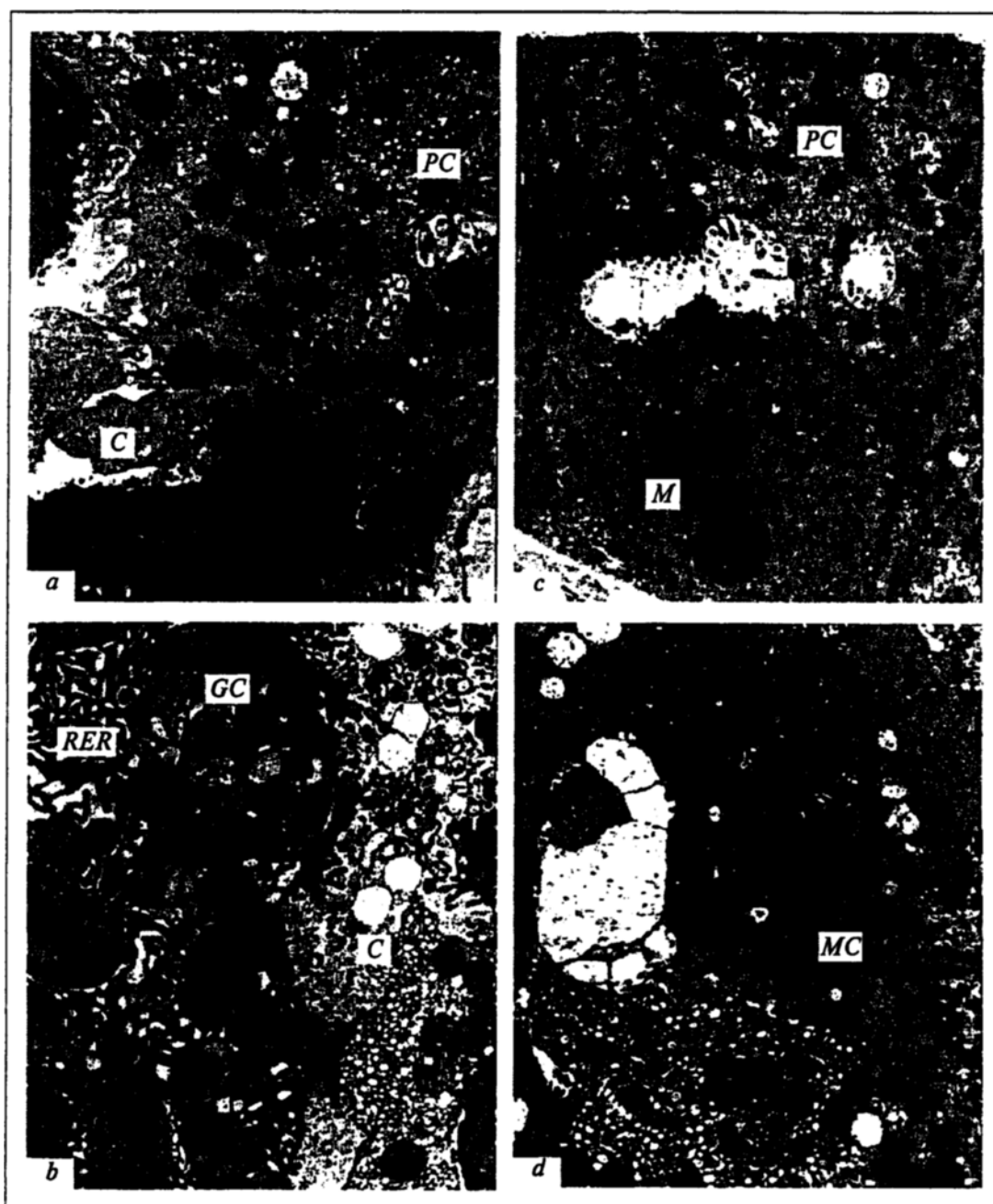


Fig. 1. Changes in epitheliocytes of fundal glands in gastric mucosa treated with copper-vapor laser. *a*) clasmotosis (C) of a parietal cells (PC) 5 min after 3-min endogastral irradiation, $\times 12,500$; *b*) enlarged profiles of rough endoplasmic reticulum (RER) and Golgi complex (GC) 1 h after 3-min endogastral irradiation, $\times 7500$; *c*) mitosis (M) in the upper third of fundal gland 1 h after 3-min irradiation, $\times 6000$; *d*) swelling and clarification of mitochondrial matrix in a mucous cells (MC) 5 min after 5-min irradiation, $\times 7500$.

doses induced cell damage and did not stimulate cell proliferation in comparison with minimal doses, whereas lower doses induced minor morphological changes and only slightly stimulated cell proliferation.

Electron microscopic examination revealed no signs of clasmotosis in samples from intact and control animals. Minimal doses did not induce clasma-

tosis in the mucosa over the entire observation period. When gastric mucosa was laparoscopically and endoscopically treated with CVL for 3 min, clasmotosis was noted primarily in parietal cells. At the initial stages, the apical cytoplasmic zones were free from cytoplasmic structures (mitochondria, tubulovesicles, etc.). These zones protruded and then separated into the lumen of parietal glands (Fig. 1, *a*,

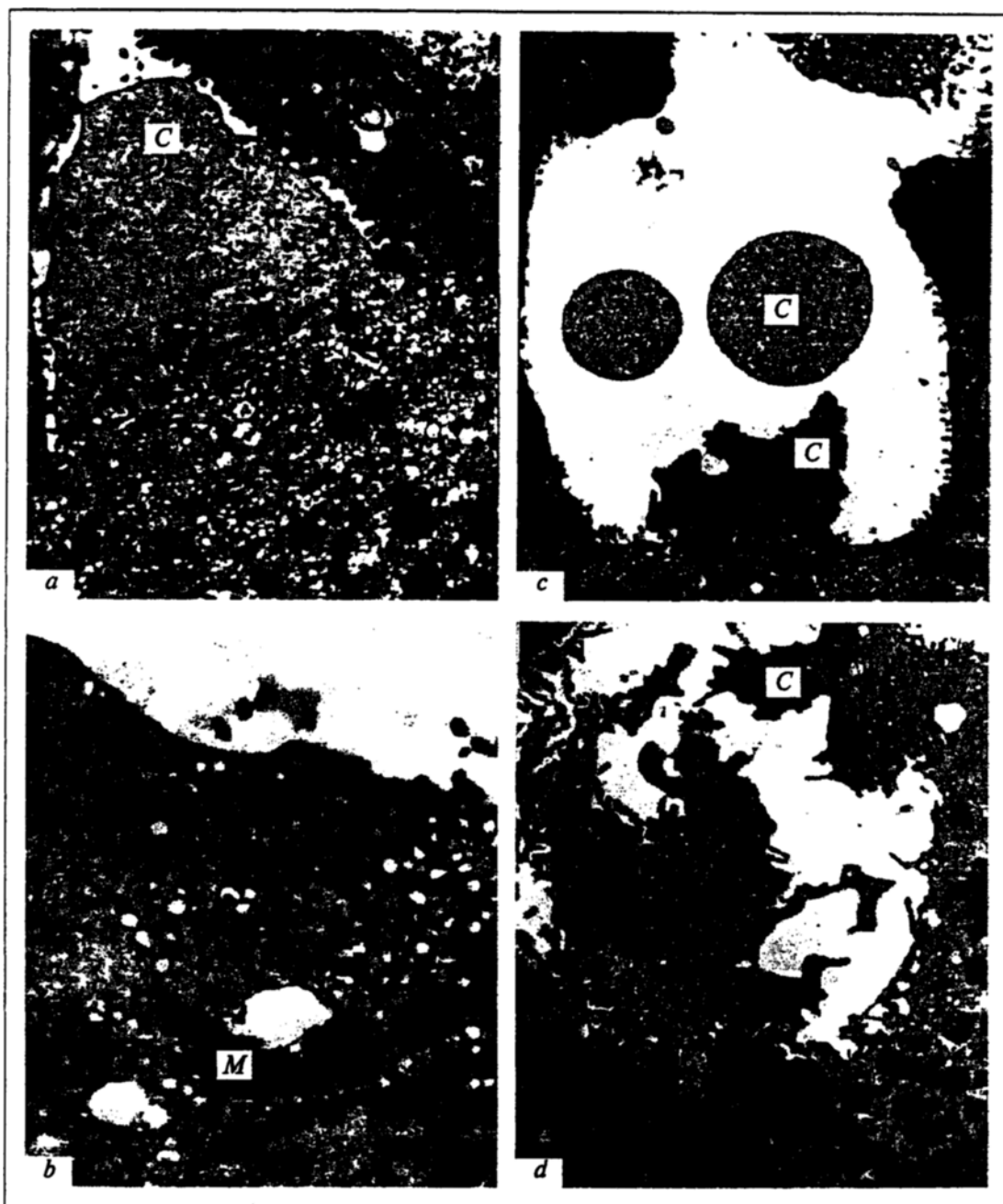


Fig. 2. Changes in epitheliocytes of fundal glands in gastric mucosa treated with UV (nitrogen) laser. *a*) clasmatosis (C) of parietal cells 1 h after 1-min laparoscopic irradiation, $\times 12,500$; *b*) swelling and partial clarification of mitochondrial matrix in parietal cells 5 min after 1-min irradiation, $\times 20,000$; *c*) clasmata (C) on the surface of a parietal cell and in the lumen 1 h after 1-min irradiation, $\times 7500$; *d*) pronounced clasmatosis of parietal cells 5 min after 2-min irradiation, $\times 7500$.

b). In the chief cells, structural alterations are less pronounced; however, multiple fragments of sub-cellular structures (profiles of the rough endoplasmic reticulum) are seen in the glandular lumen (Fig. 1, *b*). Great number of tubulovesicles and electron dense mitochondria in parietal cells and enlarged profiles of rough endoplasmic reticulum and Golgi complex indicate high secretory potential of these

cells. Numerous mitoses in the upper third of the fundal glands also attest to stimulating effect of LILI (Fig. 1, *c*).

In gastric mucosa subjected to 5-min irradiation, signs of clasmatosis are less pronounced: 5 min after treatment we observed swelling and clarification of mitochondrial matrix, especially in accessory mucous cells (Fig. 1, *d*), which disappeared 1-h after treatment.

Treatment with UVL for 1 min induced clasmato-sis in mucosa samples, these alterations increased with the dose and were similar to those induced by CVL (Fig. 2). The most pronounced changes were seen 5 min after laparoscopic irradiation with UVL in a dose of 4 J/cm², at the later stages no signs of clasmato-sis were observed. Laparoscopic and endoscopic treatment produced similar effects.

The same dose-effect relationship characterized clasmato-sis induced by helium-neon laser and CVL in cells of *lamina propria* in the intestine. Irradiation for 5 min and longer induced hydropic alterations. Clarified cytoplasmic fragments formed peculiar vacuoles, which penetrated through the basal membrane into *lamina propria*. This phenomenon differs from clasmato-sis: it is caused by hydropic disturbances and cell fragments contain no secretory material [3,5]. Secretory material (specific granules of eosinophils and mast cells and protein flocculate of plasma cells) usually presents in clasmato-sis of these cells.

Our findings suggest that clasmato-sis, a phenomenon observed in cell hyperfunction, can be induced by local and regional treatment on the stomach and intestine with CVL and UVL. In the stomach, clasmato-sis primarily develops in chief and especially in parietal cells. The most pronounced signs of clasmato-sis are induced by doses exceeding the optimal.

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