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Case report

Abnormal Levels of Pepsinogen I and Gastrin 17 in a case of Ménétrier Disease

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Abstract. *Background and aim:* We describe a case of Ménétrier disease, occurred in female patient. *Methods:* We decide to assess by non-invasive way (serum pepsinogens and gastrin 17) the secretory status of gastric mucosa, to confirm previous data of the literature, claiming high levels of both acid secretion and hypergastrinemia in this rare pathological condition. *Results and Conclusion:* We find in the subject the highest values of pepsinogen 1 – a marker of acid secretion – never described in the literature to our knowledge: 1940 mcg/L, being normal values ranging from 30-160 mcg/L. Similarly, gastrin 17, produced 90% in the antrum and responsible for negative acid feedback, was very high: 139 pg/L, ranging normal values between 1-10 pg/L. (www.actabiomedica.it)

Key words: Ménétrier Disease, pepsinogen I, Gastrin 17

Hypertrophic protein-losing gastropathy represents a rare clinical entity of unknown etiology (1). In a survey of 40 patients collected in Netherlands in 1992 (2), mean age was 44.3 years and more frequent symptoms were epigastric pain, asthenia, anorexia, weight loss, edema and vomiting. Hypoalbuminemia was found in 81% of patient and an enteric protein loss in 85%. Until now, less than 1,000 cases are reported with a prevalence of male sex (1). A percentage of 8.9% of patient developed gastric cancer after 10 years of follow-up (3).

The pathophysiology of Ménétrier disease is not completely understood, but it's claimed that and increased production of TGF-alpha leads to the proliferation of epithelial cells, in turn producing abundant mucous (4). Under the effect of TGF-alpha there is decreased acid production and elevated levels of serum gastrin (5, 6).

The overexpression of TGF-alpha is able to induce the typical histological feature of Ménétrier disease as proved in an experimental model in mice (7). We reported a case of a female aged 42 years who develop a protein loss and typical feature of Ménétrier gastropathy characterized of foveolar hyperplasia, of mucous cells, reduction of parietal cells, gland dilatation and edema (fig. 1) by immunohistochemical stain Ki-67 shows an increase of proliferating cells (fig 2).

At endoscopy, a picture of multiple friable polypoid formations with superficial erosion was detected, the stomach showed reduction of distensibility and granular hyperemic mucosa; Helicobacter pylori was negative. To assess the function of stomach, by means of ELISA method (Biohit, Helsinki, Finland) Pepsinogen I (PG I) levels and serum Gastrin 17 (G17) isoform of the hormone secreted 90% in the antrum. In our patient we found the highest value of PG I reported in literature to our knowledge: 1940 mcg/L (normal values 30-160 mcg/L). Usually, when we measured PG I levels in a patient under PPIs, with reduced levels of acid, we found a similar increase of serum PG I values (due to a renversement in the polarity of flow inside of chief cells), but the values are ranging from 200 to 700 mcg/L at maximum (8). The enormous levels find in this patient – three-fold the maximum increase reported in subjects under PPIs – could reflect the deep acid hyposecretion, typical of

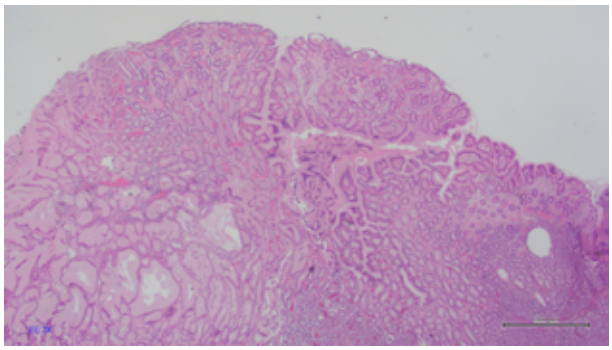

Fig. 1: Hematoxylin-eosin, 20x. Hyperplastic gastric plica with hyperplasia of mucous cells delineating tortuous and cistically dilated foveolar glands in the gastric body. Loss of parietal cells and edema of the lamina propria.

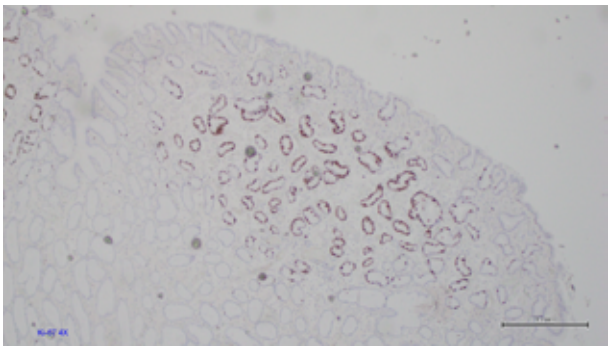

Fig. 2: Immunoistochemical staining for Ki-67, 4x: Increased proliferative activity in the adenoma portion, with minimal activity in the adjacent foveolar epithelium.

Ménétrier disease as consequence of mucous abundance and disruption of glands. Mirror of that, is the feature of very important hypergastrinemia: 139 pg/L (normal values: 1-10 pg/L), reflecting both the loss of acid secretion (negative feedback between gastrin and acid) and the high affinity bounding sites for Gastrin releasing peptide, typical of Ménétrier mucosa (9). In conclusion, in this female patient affected by Ménétrier Disease, we documented by non-invasive way (serology), a disruption of gastric physiology represented by a very huge increase of PG I and a parallel elevation of serum G17 levels.

Conflict of Interest: Each author declares no conflict of interest and that he or she has no commercial associations (e.g. consultancies, stock ownership, equity interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article

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