

An Endoscopic View in Gastric Linitis. A Case Report

Lucian Negreanu^{1,3}, Philippe Assor¹, Franck Bumsel^{1,2}, Etienne-Henry Metman¹

1) Service d'Hépatogastroentérologie. 2) Service d'Anatomopathologie, CHU Trousseau, 37044 Chambray-les-Tours, France. 3) Gastroenterology Department, University Hospital, Bucharest, Romania

Abstract

A case of an 84 year old woman addressed for dysphagia is discussed. The endoscopic and histologic examination revealed a gastric linitis with cardial invasion.

Classically, there is no characteristic macroscopic description associated with gastric linitis. An endoscopic aspect considered highly suggestive for gastric linitis is presented.

Key words

Dysphagia - secondary achalasia - gastric linitis - endoscopic view - independent cells

Introduction

The most common cause of secondary achalasia is gastric adenocarcinoma with or without esophageal invasion. The diagnosis must be suspected in patients over 50 years, with recent dysphagia and severe weight loss (1).

Plastic linitis, a rare form of primary or secondary undifferentiated adenocarcinoma may rarely manifest as pseudoachalasia secondary to massive invasion of the gastric walls and of the cardial area (2).

Case report

An 84 year-old female, complaining of progressive dysphagia and severe weight loss was addressed to our department for further investigations and management on January 2006.

Her medical history started three months before when she was admitted as an emergency to another hospital for a perforated duodenal ulcer with generalized peritonitis and severe sepsis. A surgical suture of the ulcer was realized

and aggressive antibiotic therapy initiated. The patient had a favorable evolution and she was discharged two weeks after the surgery.

In the following months, she continued to have epigastric pain, dysphagia to both liquids and solids and severe weight loss. Initially, an achalasia was suspected, but the endoscopic examination raised the suspicion of an infiltrative gastric cancer with esogastric junction invasion. Although several gastric biopsies were realized, the histological examination failed to confirm the malignancy. The patient was then transferred to our department.

At admission the clinical examination was unremarkable and the biological tests were within normal limits.

An abdominal CT scan confirmed the thickening of the gastric walls affecting the gastro-esophageal junction, the gastric body (especially the lesser curvature) and the gastric antrum. There was no evidence of bowel obstruction or extrinsic compression.

An upper endoscopy with gastric biopsies was performed on the second hospital day. It showed a dilated esophagus, with moderate liquid stasis. The passage of the gastro esophageal junction was difficult with a feeling of resistance that necessitated gentle forward pressure. There was marked thickening of the gastric mucosal folds, which were difficult to distend with subsequent luminal narrowing and gastric wall rigidity. Some gastric folds were enlarged and some parts of the mucosa had a mosaic pattern and a "leopard skin" aspect (photos) with localized nodular carmine-red lesions (photos). In the antral region the rigidity of the gastric walls and the enlarged folds gave a stenotic aspect with difficult passage of the pylorus. On the anterior wall of the duodenal bulb a large superficial ulceration was visualized.

In retroflexed vision, a giant fold around the cardial region was evidenced. The covering mucosa had a nodular appearance with carmine red lesions giving a pseudo hemorrhagic aspect (Figs.1-3). The "palpation" with biopsy forceps showed an indurated mucosa even on the nodular purple red lesions.

The histopathological study (Figs.4,5) revealed an undifferentiated adenocarcinoma with independent cells,

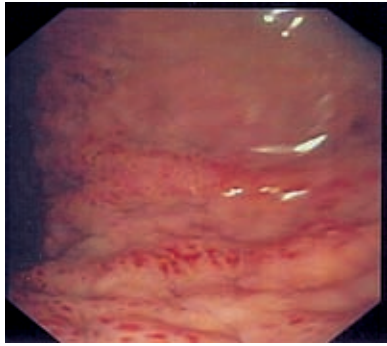


Fig.1 Endoscopy. Gastric corpus: folds with “leopard skin appearance” of the mucosa.

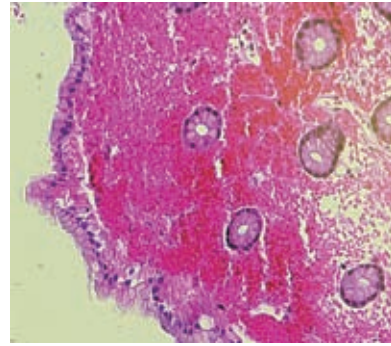


Fig.4 Superficial biopsy specimen: HE staining (carmine red lesion): subepithelial hemorrhagic suffusion.

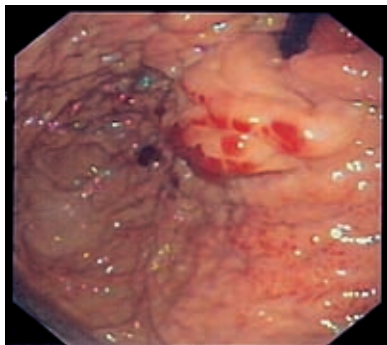


Fig.2 Endoscopy. Retrovisum: a pericardial pseudonodular fold is visualized.

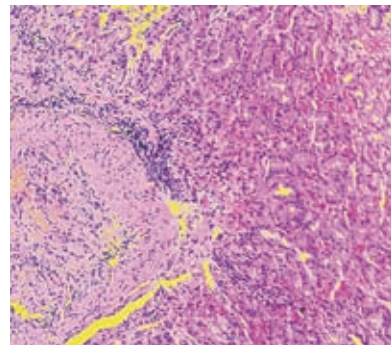


Fig.5 Deeper biopsy from the same nodule confirming the presence of independent cells in the submucosa.



Fig.3 Endoscopy. Gastric fundus: carmine red lesion giving a pseudo-hemorrhagic aspect.

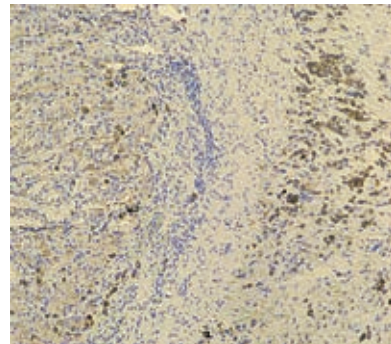


Fig.6 Immunohistochemistry: positive membrane staining for anti-cytokeratin (anti-KL1) antibodies confirming independent cell infiltration of the mucosal and submucosa layers (antral region).

which stained positive in immunochemistry for anti-cytokeratin (anti-KL1) (Fig.6) and anti-epithelial membrane antibodies (EMA). The biopsies were positive at three sites: antrum (2 of 5 specimens with “forage” at the same site), pericardial fold (2 of 2) and carmine red nodule (1 of 1).

The clinical evolution was rapid with further aggravation of the symptoms. Viewing the surgical risks, a conservative management was proposed to the patient. A naso-jejunal feeding tube was placed under endoscopic and radiological guidance and symptomatic treatment was initiated.

Discussion

Linitis plastica is a diffuse infiltrative gastric adenocarcinoma. This condition is marked by thickening and

fibrosis of the gastric wall, the malignant cells being scarcely distributed in the fibrous stroma. Frequently the gastric mucosa is spared of malignant invasion, making an endoscopic diagnosis very difficult.

The most common site of gastric linitis is the antral and pyloric regions (with variable spread proximally towards the gastric body). The fundus is least often involved.

Our patient's main complaint was progressive dysphagia. The pericardial gastric folds viewed in endoscopy were probably associated with local extension of the adenocarcinoma and responsible for the clinical manifestations.

The biopsies taken at this level confirmed the malignant infiltration.

The differential diagnosis of large gastric folds represents a challenge for the endoscopist and it includes malignancies (adenocarcinoma, lymphoma) as well as benign conditions (Menetrier's gastritis, lymphoid hyperplasia and amyloidosis).

Linitis plastica may show very little mucosal lesions on gross appearance. As a rule, since macroscopic features do not often permit the distinction between benign and malignant lesions, multiple endoscopic biopsies are required. However, standard endoscopic biopsy specimens which usually contain only mucosa offer frequently negative results for malignancy (3). In order to increase the diagnostic yield, the use of a diathermic snare which permits the obtaining of larger and deeper histologic samples is advised (4). This technique has a substantial risk of complications, particularly hemorrhage and perforation. Another possibility is to take multiple forceps biopsies from the same site: endoscopic "forage". We employed this technique taking an average of 5-6 biopsy specimens from the same site. Guided forceps biopsy on macroscopic lesions such as the pseudonodular carmine red lesions can also increase the diagnostic yield.

The CT scan and the endoscopic ultrasound may be useful for the diagnosis of gastric linitis and also for the evaluation of the local extension (5). The endoscopic ultrasound aspect of thickened deep layers is considered to be an independent predictive factor of malignancy (6). In patients with negative biopsy results on endoscopy, a fine needle biopsy can be performed under echographic, echoendoscopic or CT guidance with a good sensitivity and specificity for the diagnosis of malignancy (7-9).

Due to the severe altered status of the patient and the cardiac invasion, an echoendoscopic examination was considered aggressive and not beneficial. Also, we did not consider it useful to carry out an esophageal manometry. The CT scan confirmed the extensive infiltration of the gastric walls. The reexamination of the first (non contrast) CT scan realized at the time of the perforation revealed thickening of the gastric walls especially in the antrum. We believe that the previous duodenal ulcer perforation was not directly related to the gastric linitis, but probably favored by the cancer associated immune suppression. The erosions seen in the duodenal bulb were not suspect for malignancy and the biopsies were negative.

Gastric linitis has a very poor prognosis. The distant node metastasis and peritoneal seeding (carcinomatosis) make surgical excision rarely beneficial (10).

Conclusion

Gastric linitis with invasion of the esogastric junction may induce, although rarely, a clinical picture dominated by dysphagia mimicking primary achalasia. An endoscopic aspect of large gastric folds that fail to distend on insufflation covered by a nodular mucosa with carmine red lesions or a leopard skin appearance might be suggestive for gastric linitis. Deep biopsies realized by endoscopic forage or guided by the macroscopic lesions can spare the necessity of snare biopsies to confirm diagnosis.

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