

Infiltrative Gastric Carcinoma – Linitis Plastica

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ABSTRACT

Diffuse gastric carcinomas infiltrate gastric walls creating fibrous reactions and are frequently hard to diagnose. Biopsies of the mucosa may not contain the cancerous tissue. We present the case of a 51 year old male with dyspepsia. The upper GI endoscopy revealed thickening of the gastric mucosal folds, with no ulceration; the biopsy was normal. The endosonography confirmed the plastic linitis without invasion of the surrounding organs. A total gastrectomy was performed with partial ganglionic excision. The histopathology revealed a poorly differentiated diffuse adenocarcinoma. The patient passed away two months after surgery.

Key words Infiltrative, Gastric tumor, Linitis plastica.

INTRODUCTION:

The prognosis of diffuse gastric carcinoma remains extremely poor.^{1,2} This may be due to disregard of initial symptoms, the false negative results due to potential submucosal location of the lesion and the consequent late diagnosis.³ Increased survival with early diagnosis requires new therapeutic strategies. In some cases, diffuse carcinomas infiltrating the gastric wall, triggering a reaction associated, with a shrunken organ with thickened walls, results in the presentation of the stomach in "leather" designated plastic linitis.³ Often confined to mucosa, biopsies can omit tumor tissue confined to the submucosa tunics and deeper layers, and endoscopic ultrasound-guided fine needle aspiration (EUS-FNA) is an appropriate technique for establishing the diagnosis and guiding patient treatment.⁴ It is the objective to present a case report on the subject.

CASE REPORT:

A 51 year old Caucasian male, smoker, with moderate drinking habits, began with a symptomatic dyspepsia, which improved with antacids, but presenting a periodic recurrence of symptoms. Six months later, an abdominal ultrasound was performed, showing normal results. Nine months after the onset of symptoms, faced to the persistence of symptoms (upper abdominal pain, post-prandial bloating, regurgitation and indigestion), an upper gastrointestinal endoscopy (UGE) was performed,

revealing the presence of a gastric ulcer, for which the patient was prescribed anti-ulcer therapy. The symptoms persisted and four months later the patient attended a gastroenterology consult where a new UGE was performed. It showed the presence of ulcer scar, a small sessile polyp in the duodenal antrum and bulb in the form of clover-leaf from former ulcer formation. The biopsies showed adenomatous changes with high-grade dysplasia in some patches and the presence of isolated cells with nuclear atypia, without infiltrative aspects. Repetition of the biopsy was suggested.

About one year and five months after the onset of symptoms a new UGE was performed. Findings were prominent pseudopolypoid gastric folds and little distensible walls, scars and small sessile polypoid formations, which were biopsied, showing a histological diagnosis of gastric intestinal metaplasia, chronic active gastritis lesions with moderate dysplasia and infection by antral *Helicobacter pylori* (Hp), subcardiac and body chronic gastritis, without metaplasia, with no signs of severe dysplasia as revealed in the previous report. A new abdominal ultrasound showed, leaning against the left lobe of the liver, not seeming to be a part of it, a round structure with very thick walls, which, by the cuts in several incidences, was assumed to be a part of the stomach, without para-aortic or hepatic hilum adenopathies.

Patient was treated with triple therapy for Hp eradication. About one year and half after the beginning of symptoms, UGE was repeated with biopsies; endoscopic examination and histopathological results were similar to the former test results. The patient presented with malaise,

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pallor, anorexia and weight loss of about 8kg in 3 months, with hepatomegaly of 4 cm, painless, smooth, with no abdominal pain under palpation, with no other palpable swellings. The analysis revealed HB = 6.11 g/dl, MCV=57fl, MCH=17.8pg, protein = 69.2 and the remainder were normal. The patient was transfused and hospitalized with a diagnosis of anemia secondary to possible gastric cancer. New endoscopies were performed, with similar results to the previous, including total colonoscopy which was normal. A thoraco-abdominal CT scan showed thickened gastric walls, thickening of the gastro-hepatic ligament with lymphadenopathy in celiac trunk (Fig I). An echoendoscopy was performed, reporting that it was linitis plastica with invasion of all coats the of the stomach wall without invasion of the adjacent abdominal organs, with multiple lymphadenopathy. In all imaging the liver and spleen were normal (T2N2MI). Patient underwent total gastrectomy and excision of some lymph nodes (Fig II). The examination of surgical specimens revealed poorly differentiated adenocarcinoma, diffuse Lauren and Ming infiltrative-type lymph node metastasis (pT3N3Mx). The patient passed away about two months later.

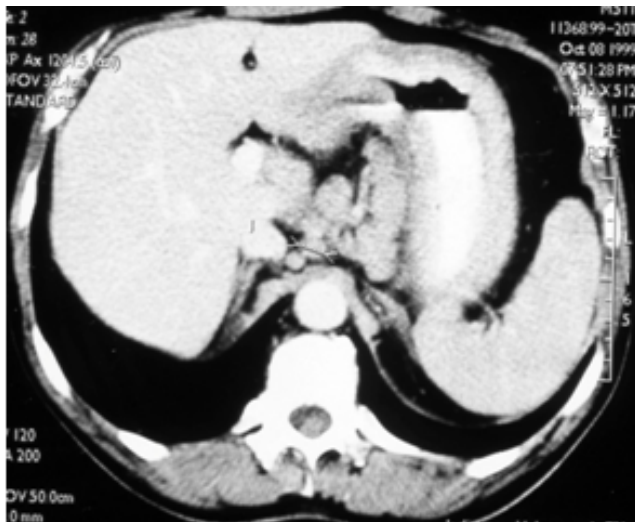


Fig I: An image of thoraco-abdominal CT scan, showing a thickened gastric walls and lymphadenopathy in celiac trunk.

DISCUSSION:

In the described case it was found, and as the literature revealed, that the initial symptoms of the patient were not properly taken in account. Indeed, it is noted that the initial symptoms can be vague and controllable with anti-ulcer or anti acid treatment.⁵ However, there was no proper appreciation of the clinical outcome of the initial biopsies that already revealed the presence of lesions of high-grade

dysplasia. The fact that, on the initial biopsies, no infiltrative aspects were observed, and having recurred, to a reevaluation that did not confirm initial data, lead to disregard of that data, which resulted in a loss of more than one year before the correct diagnosis was made.

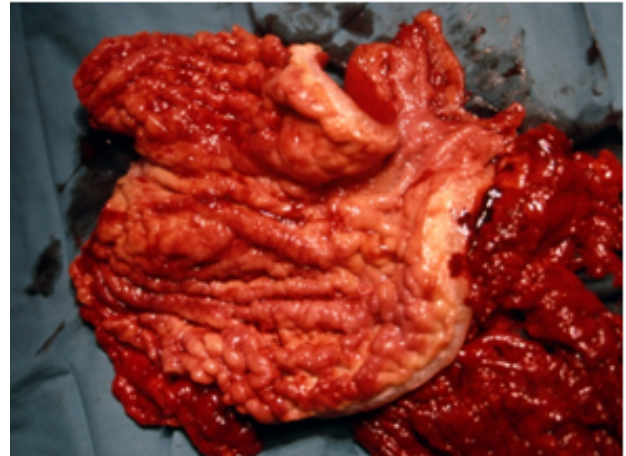


Fig II: The stomach after the gastrectomy, showing a prominent pseudopolypoid and thickened gastric folds.

In several series, at the time of diagnosis, the majority of patients had distant metastases.^{1,6} In turn, the surgical decision, very questionable, was of little help on the patient's survival. Some authors believe that an extended surgery associated with aggressive chemotherapy can sometimes be of benefit.^{7,8} Others take the opposite position, considering that chemotherapy should be the primary treatment.⁹ The truth is that in most studies survival remained short, so it requires a new approach to early diagnosis and therapeutic strategies.² For some authors, in cases with nodal metastases, the surgery alone is one of the potentially healing interventions.^{3,6}

In this case, where it was observed a very important lymphatic invasion, the utilization of chemotherapy protocols based on oral derivatives of 5-FU (TS-1) and of paclitaxel,⁸ or others, that for some authors appear a promising treatment, was not tried. It is important to recognize the importance of a multidisciplinary team in the decision workup that involves the surgeon, the gastroenterologist and the oncologist.

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