

Case Report

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ACUTE PANCREATITIS REVEALING POORLY COHESIVE GASTRIC CARCINOMA WITH SECONDARY RECTAL LOCATION: A CASE REPORT

Oussama Lakhdar¹; Rachid Ardif ¹, Loubna Elrharbaoui ¹, Ihsane El Jaâdi ¹, Houda Meyiz^{1,2}; Ihsane Mellouki ^{1,2}
1. Gastroenterology & Hepatology Department, University hospital in Tangier, Morocco.
2. Faculty of medicine and pharmacy of Tangier, Abdelmalek Essaadi University, Tangier, Morocco.

ARSTRACT

Poorly cohesive gastric carcinoma currently constitutes a full-fledged histological type, defined by a percentage of isolated cells more than 50% dispersed in a fibrous stroma. Its revealing clinical symptomatology is often non-specific. We report the exceptional case of a patient admitted to the emergency department for acute pancreatitis, whose etiological investigation revealed a poorly cohesive gastric carcinoma with secondary rectal location.

Keywords: Gastric adenocarcinoma; Gastrointestinal endoscopy; Pancreatitis; Poorly cohesive gastric carcinoma.

Corresponding Author:

Oussama Lakhdar, MD.

Affiliation: Gastroenterology and hepatology department, University hospital in Tangier.

E-mail: oussamalakhdar3@gmail.com

ORCID ID: https://orcid.org/0000-0001-9308-5135

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INTRODUCTION

Among the histological types of gastric cancer, we find the poorly cohesive gastric carcinoma whose frequency has increased in recent years reaching 35% to 45% according to recent studies. [1, 2] It is now a completely different histological and pathological entity from other types of gastric neoplasia. It is distinguished by its epidemiological, etiopathogenic, evolutionary and prognostic characteristics [3]. Its therapeutic management is controversial, studies and clinical trials are underway to determine the best therapeutic strategies against this type of cancer.

CASE REPORT

We report the case of a 51-year-old female patient, with a 6 months history of iron deficiency anemia never investigated by digestive endoscopy. The patient was admitted to the emergency room for pancreatic-like pain, without signs of clinical severity. The initial work-up showed lipemia at 3.5 times normal, hemoglobin at 11.6 g/dl, serum ferritin at 14µg/ml, leukocytes at 8500 elements/mm3, platelets at 369000 elements/mm3, CRP at 29, correct renal function and a blood ionogram without abnormalities. The initial treatment was based on digestive rest with fasting of the patient, intravenous rehydration and analgesic treatment. She was subsequently transferred to the gastroenterology department for monitoring and additional care. The patient

benefited from an abdominal ultrasonography which showed an undistended alithiasic gallbladder without dilatation of the intrahepatic bile ducts or of the main bile duct. After 72 hours of hospitalization, an abdominal-pelvic computed tomography (CT scan) was performed, showing Balthazar stage E pancreatitis (Figure 1) with moderate and non-specific parietal thickening of the rectosigmoid (Figure 2) associated with coeliomyenteric and retroperitoneal nodes along the primary and external iliac chains. A rectosigmoidoscopy was performed to characterize the recto-sigmoid thickening which showed an extensive circumferential rectal hypertrophy of 10 to 30 cm from the anal margin (Figures 3 & 4).





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Figure 1: Axial section CT image after contrast injection showing peripancreatic fat infiltration and necrotic cast along the right renal fascia

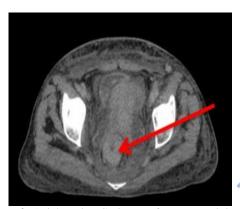


Figure 2: Axial section CT image after contrast injection showing a panrectal thickening measuring 19 mm in maximum thickness

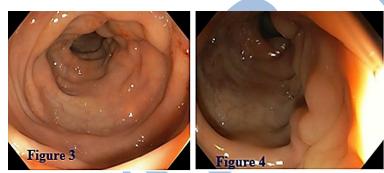


Figure 3 & 4: Endoscopic images (rectosigmoidoscopy) showing a circumferential hypertrophy of fundal

Biopsies were taken and the result of the pathological examination was in favour of an undifferentiated carcinomatous process with independent cells evoking a poorly cohesive cancer. Upper gastrointestinal endoscopy (UGIE) showed an aspect of hypertrophy of the fundal folds with local ulcerations and antral infiltration. At the level of the duodenal bulb and down to D3 there was a circumferential hypertrophied aspect with infiltration of the papilla (**Figures 5 & 6**) explaining the cause of

the pancreatitis presented by the patient: the infiltration of the duodenal papilla causing an obstruction to the passage of pancreatic juice to the



duodenum and therefore an activation of pancreatic enzymes within the gland leading to its inflammation. **Figures 5 & 6:** UGIE images showing a circumferential hypertrophied duodenum infiltrating the papilla.

Pathological study of the gastric and the duodenal biopsies also showed a poorly differentiated carcinomatous process with independent cells (poorly cohesive cancer) (**Figures 7 & 8**).



Figure 7: duodenal infiltration by a tumoral proliferation with a signet ring cells; Asterixis*: Normal duodenal glands; Red arrow: tumor proliferation

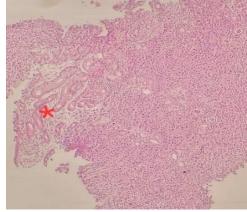


Figure 8: gastric infiltration by tumor proliferation in diffuse small cell sheets; Asterixis*: normal gastric glands.





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Cervical and thoracic CT scan showed cervical and sub-diaphragmatic nodes with a bilateral pleural effusion, more marked on the right. The tumour was classified as stage IV (T3N3M1) according to the TNM classification (presence of a secondary rectal location with multiple subdiaphragmatic cervical, coeliomyceteric and inguinal adenopathies), palliative chemotherapy based on fluoropyrimidine and platinum salts was scheduled. In the meantime the patient presented a respiratory distress with a bilateral pleural effusion leading to a rapid deterioration of her general condition and causing her death. A massive pulmonary embolism was suspected.

DISCUSSION

Poorly cohesive carcinoma (PCC) is a special type of gastric adenocarcinoma, which is a distinct entity according to the WHO classification, and is part of the diffuse type of adenocarcinoma according to the Lauren classification [3]. It is defined as a form of adenocarcinoma composed of at least 50% of isolated cells dispersed within a fibrous stroma. [1] Gastric linitis is part of, but not synonymous with PCC. Gastric linitis has an endoscopic and macroscopic definition with a rigid and thickened appearance of the gastric wall. It can be made up of signet ring cells (SRC) (most often) as it can be made up of other types of tumor cells [4]. There are currently 3 types of PCC: the first with more than 90% of the signet ring cells called PCC-SRC type, the second with a percentage of SRC between 10 and 90% called PCC with SRC component and the third PCC with < 10% of SRCs called PCC not otherwise specified (PCC-NOS) [5]. Over the past 30 years, the worldwide incidence of gastric adenocarcinoma has been decreasing, but the incidence of PCC has been increasing, especially in Western countries [6]. This histological type has an etiopathogeny directly linked to genetic mutations, in particular of the CDH1 gene coding for the Ecadherin protein involved in gastric cell adhesion [7]. Like other types of gastric cancer, its clinical symptoms are non-specific and are dominated by abdominal pain and altered general condition [8]. The occurrence of acute pancreatitis revealing a poorly cohesive gastric carcinoma is exceptional; no case has been described in the literature. Confirmation of the diagnosis is made by endoscopic examination with anatomical-pathological study of the biopsies taken from the lesions observed [9]. The endoscopic appearance is variable depending on the duration of evolution, in the early stages they present as a flat lesion, whereas advanced PCC often has an infiltrative-ulcerative appearance [9]. The primary gastric location is the most frequent, other primary

digestive locations are exceptional and require the absence of an associated gastric tumour lesion to be retained [10]. Moreover, the frequency of PCC and gastric linitis of duodenal location is rare, about twenty cases have been described in the literature so far, as well as primary tumours and neoplastic invasion of the ampulla of Vater by PCC (less than 10 cases have been described in the literature) [11,12]. Other complementary examinations, in particular thoracic-abdominal-pelvic CT scan and endoscopic ultrasonography for gastric linitis are necessary to assess the locoregional extension and search for secondary locations [13]. Lymph node involvement and peritoneal carcinosis are the main metastatic sites; secondary digestive locations are rare, as in our patient's case, who had both gastric and rectal involvement. [14, 15]. The treatment of PCC deemed to be resectable is essentially surgical by total gastrectomy associated with lymph node dissection to improve the prognosis of patients with non-metastatic PCC. The combination of surgery with radiotherapy or postoperative chemotherapy depends on the stage of the tumour, the resection margins and lymph node invasion. In fact, patients with a stage III, positive resection margins or regional lymph node invasion will benefit from adjuvant treatment [16]. The place of perioperative chemotherapy remains controversial according to the various studies evaluating its impact on overall survival in patients treated surgically. Indeed, perioperative chemotherapy was the rule in the treatment of gastric cancers, but some studies analyzing the contribution of chemotherapy according to the histological type of gastric cancer have shown possible chemoresistance of PCC. These results need to be confirmed by other studies [17]. The poor prognosis of PCC is still debated, and depends mainly on the stage of the tumor, indeed studies on superficial PCC have shown a good prognosis for this histological type compared to other types. However, the prognosis of deep PCC often remains pejorative with some differences between Eastern and Western studies which show a worse prognosis than Eastern studies. This poor prognosis could be explained by the late diagnosis, the rapid extension to the serosa and the lymph nodes and the peritoneal carcinomatosis most often found in this type of gastric cancer. Currently studies are underway to determine the prognostic nature of this type of cancer compared to other histological types [18,19].

CONCLUSION

This case reports the diagnosis of gastro-duodenal PCC, which remains a rare localization of this histological type, following the occurrence of pancreatitis. The presence of a second associated



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rectal location makes this case exceptional. In view of this unusual mode of revelation and the non-specific nature of the clinical symptoms of PCC, a more thorough etiological investigation with a multiplication of complementary examinations is necessary in the event of any suspicion of a cancer of this type.

CONFLICT OF INTERESTS: The authors report no conflicts of interest. They are responsible for the content and writing of this article.

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