PYLEPHLEBITIS INCIDENTALLY FOUND ON A PATIENT WITH GASTRIC CANCER

PILEFLEBITE INCIDENTAL NUM PACIENTE COM CARCINOMA GÁSTRICO

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ABSTRACT

Pylephlebitis is a suppurative infection of the portal vein. This rare condition is most often related to intra-abdominal infections like appendicitis or acute diverticulitis. We present an incidentally found case of pylephlebitis in a patient who presented to the emergency department with unspecific symptoms and a recent diagnosis of gastric cancer.

Keywords: Pylephlebitis, gastric cancer, portal vein.

RESUMO

A pileflebite é uma infecção supurativa da veia porta. É uma patologia rara e habitualmente associada a infecções intra-abdominais como a diverticulite e a apendicite aguda. Descrevemos um caso incidental de pileflebite diagnosticada num doente que recorreu ao serviço de urgência com sintomas inespecíficos e com o diagnóstico recente de carcinoma gástrico.

Palavras-chave: Pileflebite, carcinoma gástrico, veia porta.

INTRODUCTION

Pylephlebitis is defined as a septic thrombophlebitis of the portal vein and its branches and usually occurs secondary to infection in the region drained by the portal venous system.¹

Since its first description in an autopsy specimen in 1846, the widespread use of abdominal computed tomography (CT) has led to an increase in reported cases.² Reports most commonly associate it with

diverticulitis, but it has also often been described in appendicitis, necrotizing pancreatitis and inflammatory bowel disease.¹⁻⁴

Pylephlebitis is a life-threatening infection that can further complicate with hepatic abscesses and less often with thrombus progression and mesenteric ischemia. The reported mortality rate of this condition varies between 11 and 32%.²⁻⁴



CASE REPORT

A 79-year-old Caucasian male presented to the emergency department with a one month history of food intolerance, sporadic epigastric pain, weight loss and an episode of lipothymia that day. His past medical history included moderated chronic obstructive pulmonary disease (GOLD stage II) with lung emphysema, hypertension, and hyperlipidemia. The patient was a former smoker (stopped smoking 10 years ago). He had no family history of hypercoagulable diseases or thromboembolism. An upper gastrointestinal endoscopy performed two days before identified an extensive and ulcerated tumor of the gastric antrum. He denied fever or chills. On physical examination he presented with a normal pulse rate and body temperature, a slight hypotension and no abdominal pain. Acute cardiac disease was excluded. Blood tests revealed an elevated C-reactive protein (CRP) level of 99.7 mg/L with a normal leucocyte count. He also presented hydroelectrolytic abnormalities compatible with the history of persistent vomiting (K⁺ 2.7 mmol/L; Cl⁻ 90 mmol/L). Hepatic and renal function were normal as well as the coagulation tests performed [prothrombin time (PT); partial thromboplastin time (PTT) and international normalized ratio (INR)].

The patient was admitted to the surgical ward for correction of the hydroelectrolytic disorders and tumor staging. Staging computed tomography (CT) scan revealed thrombosis of the portal vein and its branches and aeroportia, findings compatible with pylephlebitis (Figures 1 and 2).

The patient underwent broad spectrum antibiotic therapy with piperacillin/tazobactam and was anticoagulated with low molecular weight heparin. He remained asymptomatic throughout the course of admission. Blood culture tests were negative. CRP level before antibiotic withdrawal was 9.6 mg/L.

The pathological evaluation of the biopsy specimen and pre-operative clinical staging indicated a cT4aN0M0 gastric adenocarcinoma. The clinical case

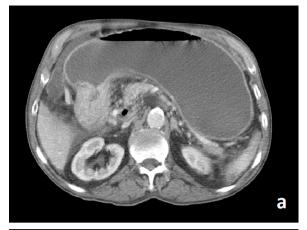




FIGURE 1: (a) Axial CT scan showing gastric distention and increased gastric wall thickness in the pyloric region; aeroportia can also be seen in this picture. (b) Left arrow shows thrombus and air in the portal vein, right arrow shows thrombosis in the splenic vein; arrowhead shows contrast in the splenic vein before the thrombus.

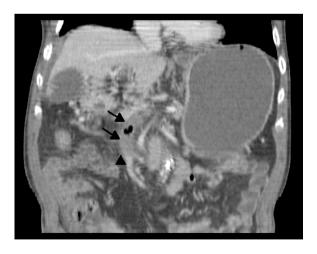


FIGURE 2: Coronal reconstruction showing superior mesenteric vein thrombosis and air; arrowhead shows contrast in the superior mesenteric vein before the thrombus.



was presented at the tumor board which decided for primary surgery based on the patient's medical history. Exploratory laparoscopy revealed gastric cancer invasion of the mesocolon and ascites. The patient was submitted to a palliative gastrojejunostomy. No post-operative complications were documented. Palliative chemotherapy was proposed but the patient refused it. Survival after surgery was 24 weeks.

DISCUSSION

We present a case of a patient with a pylephlebitis associated with a gastric cancer. Case reports of infected portal vein thrombosis have also been presented in association with other gastroduodenal diseases, namely gastric and duodenal ulcers, intragastric migration of an adjustable gastric band and emphysematous gastritis.^{2, 3}

The stomach has a widespread venous drainage into the portal system with the left and right gastric veins converging into the main portal vein, the short gastric and left gastroepiploic veins into the splenic vein and the right gastroepiploic vein into the superior mesenteric vein.

Although blood cultures were negative in this case, bacterial translocation through the distended and ulcerated gastric wall, associated with the thrombogenic potential of this tumor, may have been the main factors contributing to the occurrence of this condition.

Hypercoagulable states are known risk factors for pylephlebitis and these can be determined either by host conditions like clotting factor deficiencies and malignancy or pathogen associated characteristics like the thrombogenic potential of *Bacteroides species* and other bacteria.^{2,5} According to the literature, blood cultures may be negative in 12 to 77% of the cases of phylephebitis.²

There is currently no consensus on the use of anticoagulation in the management of pylephlebitis as it may respond to antibiotic therapy alone. ^{1, 4-6} The rational for its use is to prevent thrombus progression, promote recanalization, decrease embolization to the liver from infected portal thrombi and eventually increase survival. ⁴⁻⁶ Despite the absence of good quality evidence, current studies suggest that patients with pylephlebitis and a hypercoagulable state due to neoplasms or clotting factor deficiencies should be anticoagulated. ^{4, 5}

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Data de recepção do artigo: 14-06-2020

Data de aceitação do artigo: 08-12-2020

