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Caso Clínico / Radiological Case Report

Pilephlebitis Mimicking Malignant Hepatic Lesions: A Rare Diagnostic Pitfall

Pileflebite Simulando Lesões Hepáticas Malignas: Um Pitfall Diagnóstico Raro

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Abstract

Pylephlebitis results from the venous spread of an intra-abdominal infection through the mesenteric and portal systems. The most common cause in adults is acute diverticulitis, while in young people it is acute appendicitis. Patients usually arrive at the emergency department with fever and abdominal pain. Once hemodynamic stability has been ensured, blood cultures should be collected to identify the pathogen(s). Laboratory tests reveal increased inflammatory parameters and liver cytolysis and cholestasis enzymes. Abdominal ultrasound or computed tomography highlights the presence of thrombi in the mesenteric-portal circulation and can also identify the site of infection, as well as any complications, such as pyogenic liver abscesses or intestinal ischemia. Treatment consists of antibiotic therapy (initially broad-spectrum and then targeted) and, frequently, anticoagulants. Greater awareness of this condition and early medical intervention have reduced mortality to less than 10%.

Keywords

Portal vein thrombosis; Liver abscess; Computed tomography; Diverticulitis; Pylephlebitis; Sepsis.

Resumo

A pileflebite resulta da disseminação venosa de um foco infeccioso intra-abdominal através da circulação mesentérica e portal. A causa mais comum nos adultos é a diverticulite aguda, enquanto nos jovens é a apendicite aguda. Os doentes habitualmente chegam ao serviço de urgência com febre e dor abdominal. Garantida a estabilidade hemodinâmica, devem ser colhidas hemoculturas para a identificação do(s) agente(s) patogénico(s). O estudo analítico revela aumento dos parâmetros inflamatórios e das enzimas de citólise hepática e de colestase. Na ecografia ou tomografia computorizada abdominal destaca-se a presença de trombos na circulação mesentérico-portal, podendo também identificar-se o ponto de partida infecioso, bem como eventuais complicações, nomeadamente abcessos hepáticos piogénicos ou isquemia intestinal. O tratamento é feito com antibioterapia (inicialmente de largo espectro e depois dirigida) e, frequentemente, com anticoagulantes. O maior reconhecimento atual desta patologia e a intervenção médica precoce permitiram reduzir a mortalidade para valores <10%.

Palavras-chave

Trombose venosa portal; Abcesso hepático; Tomografia computorizada; Diverticulite; Pileflebite; Sépsis.

Case Presentation

A 56-year-old man was admitted to the emergency department via the sepsis referral pathway, presenting with fever, nausea, and generalized abdominal pain for the past 5 days. He was a Black man born in Angola. Regarding his medical history, he only reported a previous diagnosis of liver cirrhosis. He denied any surgical history and had no family doctor assigned to him. On physical examination, he presented with tachycardia and hypertension, with defense against superficial and deep abdominal palpation, especially in the lower quadrants. Analytically, what stood out was leukocytosis (15732 leukocytes/µL), increased C-reactive protein (CRP) [129 mg/dL], increased hepatic cytolysis parameters (alanine aminotransferase (ALT) [76 U/L] and aspartate aminotransferase (AST) [118 U/L], increased cholestasis parameters (alkaline phosphatase (ALP) [463 U/L], gamma-glutamyl transferase (GGT) [210 U/L]), with total bilirubin and creatinine only slightly increased.

An urgent abdominal and pelvic computed tomography scan with intravenous contrast (in venous phase) was performed with the purpose to identify the infectious focus. Colonic diverticulosis was documented in the descending colon and sigmoid colon, particularly pronounced in the colonic curvature of transition between the two segments. In the sigmoid colon, a cluster of prominent diverticula with thickened and hyperenhancing walls stood out, coexisting with slight densification of the surrounding fat, aspects that together were highly suggestive of acute diverticulitis (Figure 1).

In the upper abdomen, a marked hepatomegaly was identified (longitudinal diameter of the right lobe measured 26 cm), with relative hypertrophy of the caudate lobe and slightly lobulated contours, consistent with known chronic inflammatory liver disease. Multiple hypovascular liver lesions were evident, predominantly in the right lobe, and thrombosis of the main branch of the portal vein and its right main branch (Figure 2), placing the differential diagnosis between an infectious and a malignant etiology.

The presence of lymphadenopathies was also noted in the hepatic hilum, the largest measuring 13 mm in the short axis, and in the aortocaval region, with a short axis of 11 mm.

Considering the possibility of malignancy, a percutaneous ultrasound-guided biopsy of the infiltrative hepatic nodules

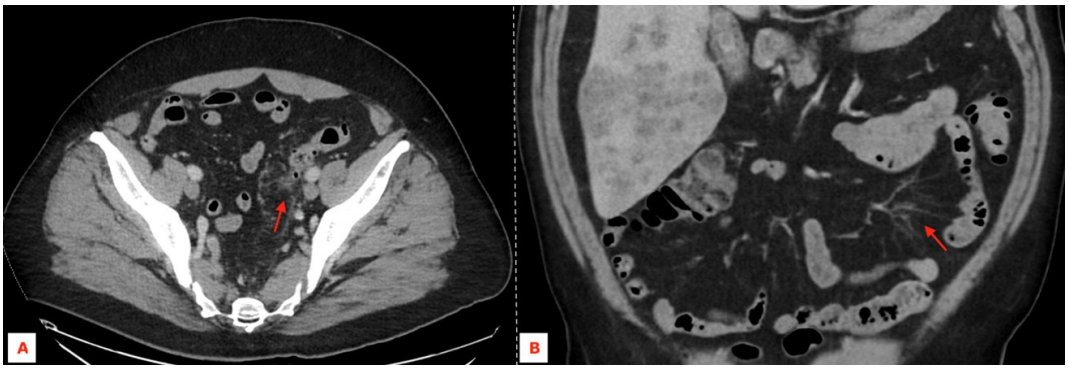

Figure 1 – Computed tomography after intravenous contrast, in the venous phase, in the axial plane (A), has demonstrated colonic diverticulosis at the transition from the descending colon to the sigmoid colon, coexisting with slight fat densification around some diverticula, which presented evident parietal thickening, aspects suggestive of acute diverticulitis. No extraluminal gas or locoregional or intra-abdominal abscessed collections (Hinchey classification grade Ia) were documented. In the coronal plane (B), vascular engorgement was also identified, reflecting the greater need for vascular supply in inflammatory processes.

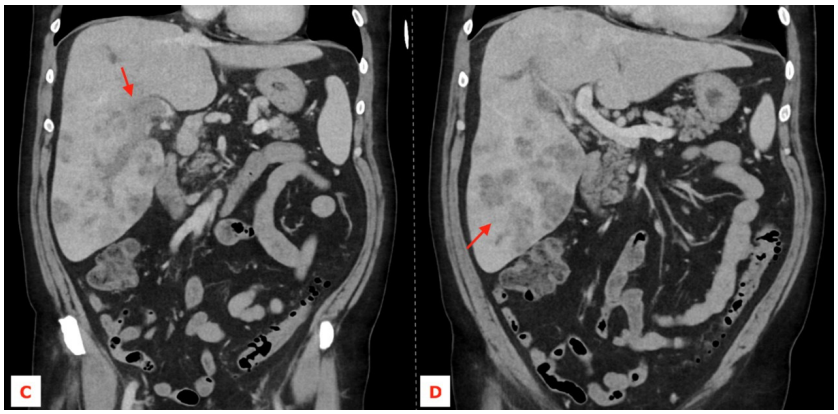

Figure 2 – In the computed tomography scan after intravenous contrast, in the venous phase, in the coronal plane, thrombosis of the portal vein was highlighted, starting at its bifurcation, with involvement of the entire right tree (C). Hepatomegaly was also noted (craniocaudal axis of the right lobe measures 26 cm), identifying multiple hypovascular lesions with predominance in the right lobe (D), raising the differential diagnosis between an infectious and a malignant etiology. Lymphadenopathy was also described in the hepatic hilum, the largest with a short axis of 13 mm.

was performed, with the collection of one 18G fragment for histological evaluation, which proved negative. It was decided to repeat the ultrasound-guided biopsy and ultrasound showed a reduction in the volume of the hepatic lesions and in the extent of the portal vein thrombosis, so the procedure was cancelled. The patient was re-evaluated 1 month later by abdominal and pelvic CT scan, which now highlighted a slightly heterogeneous enhancement of the hepatic parenchyma in segments 6 and 7, with practically complete resolution of the previously described hypovascular nodules. Resolution of the portal vein thrombosis was also observed, with only chronic/sequelae changes persisting, with a reduction in the caliber of the anterior and posterior segmental branches of the right main portal branch (Figure 3).

Abdominopelvic lymphadenopathies were no longer evident. These findings, as a whole, are interpreted as inflammatory/infectious liver changes (liver abscesses) due to pylephlebitis (infectious thrombosis of the portal venous complex), with an infectious starting point in acute diverticulitis of the sigmoid colon.

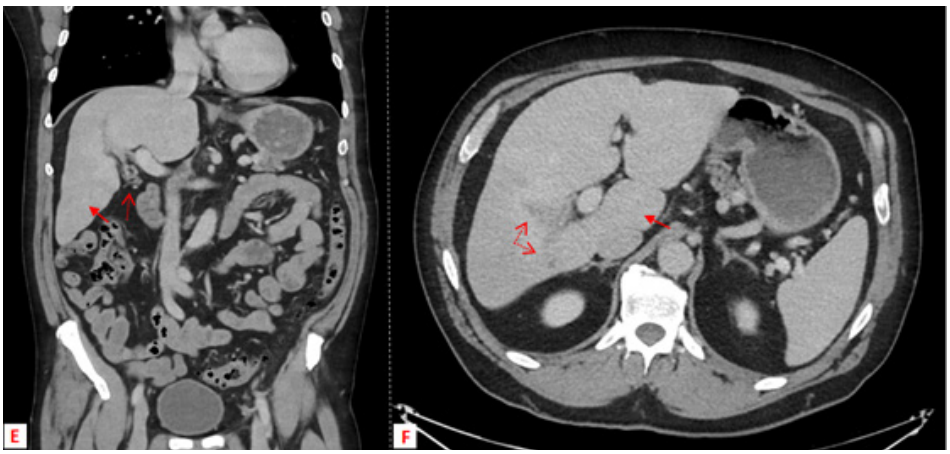

Figure 3 – In the contrast-enhanced computed tomography scan, in the venous phase, in the coronal plane, performed 1 month later, slightly heterogeneous parenchymal enhancement was highlighted in segments 6 and 7, with practically complete resolution of the previously described hypovascular nodules (filled arrow in E). Partial resolution of the portal vein thrombosis also occurred, and in this follow-up CT study, only chronic changes were identified, with a reduction in the caliber of the anterior and posterior branches of the right main portal branch (dashed arrows in F) and the appearance of collateralization (dashed arrow in E). Relative hypertrophy of the caudate lobe was also observed (filled arrow in F), consistent with chronic liver disease.

Discussion

Pylephlebitis results from the venous spread of an intra-abdominal infectious focus through the mesenteric circulation (superior or inferior), subsequently entering the main branch of the portal vein, and it may extend to the main or left and right segmental branches.[2,3,4] Impaired portal blood flow can result in transient alterations in hepatic perfusion (due to flow compensation by the hepatic artery) and the formation of pyogenic liver abscesses.[1,3,4] Total occlusion of mesenteric venous drainage can ultimately lead to intestinal ischemia.[1,2]

Acute diverticulitis is the most frequent cause of pylephlebitis.[1,2,3,4] It corresponds to one of the most frequently associated intra-abdominal infectious foci with this condition, and may manifest on computed tomography by increased thickening and enhancement of the diverticular wall, densification of the surrounding fat, vascular engorgement, and segmental thickening of the affected intestinal wall.[1,2] Another common cause of pylephlebitis is acute appendicitis, which is most common in young individuals.[1,4] Less common causes include exacerbation of inflammatory bowel disease, acute pancreatitis, acute cholangitis, urinary tract infections, and pelvic infections.[3,4] In about 11.5% of cases, the infectious starting point is not identified.[4]

The first thing to do when faced with patients admitted to the emergency department in septic shock is to stabilize them hemodynamically. Next, we look for the source of the infection. The patient presented with abdominal pain, so the initial investigation focused on this area. Imaging plays a fundamental role in this process.[1,2,3,4] An abdominal-pelvic CT scan with intravenous contrast (in the venous phase) was chosen as the first-line examination, given its greater diagnostic accuracy in identifying the intra-abdominal septic focus and its complications, as ultrasound may be limited in this clinical context.[3,4] An abdominal infectious focus was identified in this patient, namely acute diverticular inflammation. However, additional findings were found, notably thrombosis of the portal venous complex and the presence of multiple hypodense liver lesions (with and without intravenous injection of iodinated contrast, in the venous phase).[1,3] The differential diagnosis of liver lesions with hypoenhancement in the venous phase is vast and should include infectious and malignant neoplastic causes (primary and secondary).[4] Although it is not possible to assess the behavior of the lesions in an earlier phase (arterial phase), multifocal HCC should be considered a hypothesis, because the patient has a diagnosis of liver cirrhosis, and there is always the possibility that the lesions are hypervascular in the arterial phase and hypovascular in the venous phase due to washout.[2,4] On the other hand, if the lesions were hypovascular in the arterial phase, other diagnoses would become more likely, such as multifocal cholangiocarcinoma.[2] There is also the possibility that they may correspond to metastases, although this is, in principle, a less likely hypothesis, considering the history of liver cirrhosis and the absence of known neoplasia.[1,2] The possibility of pylephlebitis, a rare complication of acute diverticulitis, should also be considered, since this is a patient with sepsis without known neoplasia, and the hypodense liver lesions may correspond to pyogenic abscesses.[1,2,3,4]

Early confirmation of the etiological nature of the lesions is fundamental as this allows the timely initiation of the most appropriate treatment, which is crucial for the success of the therapeutic intervention and, consequently, for the patient's prognosis.[3,4]

Clinically, patients typically present to the emergency department with fever and abdominal pain, which may be accompanied by nausea/vomiting and/or diarrhea.[3,4]

Blood samples may show several biochemical changes, the most common being elevated inflammatory parameters (leukocytosis and increased CRP and ESR), (leukocytosis and increased CRP and ESR) elevated liver cytolysis enzymes (ALT and AST), and elevated cholestasis enzymes (ALP and GGT).[2,4]

From an imaging perspective, we may not always be able to identify the infectious focus, and the occurrence of complications from hematogenous dissemination of the infection may not be evident as well, but there is one finding that is central to the diagnosis of pylephlebitis, which is the presence of thrombosis of the veno-portal system.[1,2,3] Both upper abdominal ultrasound and abdominal computed tomography can demonstrate this finding, the former by the presence of intraluminal content, associated with the absence or decrease of downstream vascular flow, and the latter by the venous filling deficit with contrast due to the presence of a (sub)occlusive thrombus.[1,3,4]

Collecting blood cultures before initiating broad-spectrum intravenous antibiotic therapy is part of the initial diagnostic process for sepsis. This allows the identification of the pathogenic agent(s) involved and, consequently, directs treatment after obtaining the results of the antimicrobial susceptibility test. The most frequently identified bacteria, in descending order, are Escherichia coli, Bacteroides spp., and Streptococcus spp. In a minority of cases, the identified etiological agents are fungi of the genus Candida spp. or intestinal parasites (helminths or amoebas). It is important to emphasize that blood cultures do not always yield positive results and may be non-informative in 30% of cases, although this is not sufficient to refute the diagnosis of pylephlebitis. [4]

The duration of treatment should be at least four to six weeks. Oral administration should only be considered after a significant improvement in the clinical, analytical, and imaging picture. There is no consensus regarding the need for anticoagulant therapy, nor regarding its approach. Its prescription is mainly recommended to patients with progression of venous thrombosis, those with more extensive thrombosis ab initio beyond the main portal branches, or those with a post-thrombotic state (hereditary or acquired).[4]

Pylephlebitis can be complicated by the formation of pyogenic liver abscesses (and less frequently, brain or lung abscesses) or intestinal infarction due to acute mesenteric venous ischemia. Septic shock is the cause of death in the vast majority of fatal cases.[3,4]

It follows, therefore, that pylephlebitis involves a high risk of mortality and morbidity if not diagnosed and treated promptly. The role of the radiologist is therefore fundamental. A better understanding of this entity has allowed not only increased recognition but also earlier intervention and, consequently, a reduction in current mortality to values <10%.[1,3,4]

Ethical Disclosures / Divulgações Éticas

Conflicts of interest: The authors have no conflicts of interest to declare.

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Confidentiality of data: The authors declare that they have followed the protocols of their work center on the publication of data from patients.

Confidencialidade dos dados: Os autores declaram ter seguido os protocolos do seu centro de trabalho acerca da publicação dos dados de doentes.

Protection of human and animal subjects: The authors declare that the procedures followed were in accordance with the regulations of the relevant clinical research ethics committee and with those of the Code of Ethics of the World Medical Association (Declaration of Helsinki).

Protecção de pessoas e animais: Os autores declaram que os procedimentos seguidos estavam de acordo com os regulamentos estabelecidos pelos responsáveis da Comissão de Investigação Clínica e Ética e de acordo com a Declaração de Helsínquia da Associação Médica Mundial.

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