An uncommon cause of portal vein thrombosis

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CASE REPORT

A 36-year-old male presented with a two-week history of abdominal pain and chills. His medical history was unremarkable. His vital signs were normal except for mild pyrexia of 37.6 °C. Examination of the abdomen revealed some tenderness in the mid-abdominal region. His liver enzymes were elevated with a total bilirubin 200 μ mol/l (normal 0-17 μ mol/l), aspartate aminotransferase 92 IU/l (normal 0-40), and gamma glutamyl transpeptidase 100 U/l (normal 0-60 U/l). Ultrasound and computed tomography (CT) scan of the abdomen revealed thrombosis of the mesenteric and portal veins. Blood cultures were

Figure 1. CT scan: Thrombus of the superior mesenteric vein and its branches



Figure 2. CT scan: Appendiceal wall thickening and free abdominal fluid



positive for *Escherichia coli, Streptococcus millerus* and *Staphylococcus epidermidus*. Because of progressive sepsis with circulatory and respiratory failure the patient was admitted to the intensive care for intubation and resuscitation. Two days later a CT scan of the abdomen was repeated, which showed multiple hypodense lesions in the liver consistent with abscesses.

WHAT IS YOUR DIAGNOSIS?

See page 435 for the answer to this photo quiz.

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ANSWER TO PHOTO QUIZ (PAGE 431) AN UNCOMMON CAUSE OF PORTAL VEIN THROMBOSIS

The patient was diagnosed with a pylephlebitis, portal-mesenteric thrombosis, and multiple liver abscesses. Pylephlebitis, or infective suppurative thrombosis of the portal vein, is a serious condition with significant morbidity and mortality, which can complicate intra-abdominal sepsis of any aetiology. Pylephlebitis is caused by a thrombophlebitis of small veins draining an area of infection. Extension of the thrombophlebitis into larger veins leads to septic thrombophlebitis of the portal vein resulting in septic emboli to the liver.

Since an abdominal infection was very likely in this case, all CT scans were revised. In retrospect these also revealed appendiceal wall thickening with periappendiceal fat straining consistent with acute appendicitis.

In 1898, Dieulafoy described the association between appendicitis and liver abscesses as le foie appendiculaire.¹ Pyogenic hepatic abscesses are rare with less than 10% of cases caused by appendicitis.² Pylephlebitis can complicate any intra-abdominal infection that occurs in the region drained by the portal venous system, such as diverticulitis, inflammatory bowel disease, pancreatitis and cholangitis.

The patient was treated with broad-spectrum antibiotics (ceftriaxone, metronidazole and vancomycin) and anticoagulation. Yet he showed no clinical improvement, but progression of the liver abscesses and an abdominal compartment syndrome due to major abdominal bleeding. An appendectomy was performed and five litres of old blood were removed. Postoperatively, the patient recovered well and was treated with antibiotics for another six weeks. Anticoagulation was continued for another three months after the laparotomy. No hypercoagulable status was found.

The conservative management of complicated appendicitis is associated with a decrease in the complication and reoperation rate compared with acute appendectomy and it has a similar duration of hospital stay.³ But as this case shows, some patients whose symptoms fail to resolve still need surgical intervention.

There are neither prospective randomised controlled studies nor consensus on the use of anticoagulation in pylephlebitis. The rationale for anticoagulation in acute pylephlebitis is the prevention of thrombus extension and its sequelae. If there is mesenteric vein involvement (as in this case), patients may benefit from anticoagulation, since the risk of bowel ischaemia is higher.⁴

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