Unusual Causes of Abdominal Pain

by James McNicholas, Robert Lawson

CASE

A 24-year-old man with native mitral valve Streptococcus mitis endocarditis presented to the emergency department with acute burning epigastric pain following three weeks of intravenous penicillin therapy. The patient described four days of near-constant pain of acute onset, unrelated to meals and unrelieved by H2-receptor blockers, proton pump inhibitors, and over the counter antacids. Exacerbation was described with leaning forward and deep inspiration, and partial remission was achieved only by lying still. The patient admitted to nausea without vomiting and review of systems was otherwise unrevealing. On examination, vital signs were within normal limits and his abdomen was soft and nondistended but tender to palpation in the epigastrium. There was no rebound tenderness, guarding or rigidity. A GI cocktail administered in the emergency department was ineffective, and intravenous hydromorphone resulted in modest pain reduction. The patient’s complete blood count, liver panel, electrolytes, renal function and serum lipase were all within normal limits, and following an unremarkable acute abdominal plain film series he was sent for computed tomography (CT) scan of the abdomen with intravenous contrast.

See the answer and discussion on page 58
UNUSUAL CAUSES OF ABDOMINAL PAIN, #7

(continued from page 52)

ANSWER AND DISCUSSION

Hepatic Infarction
The relative rarity of hepatic infarction is commonly attributed to the dual blood supply and extensive collateral circulation of the liver. There are multiple causes of hepatic infarction: iatrogenic ligation (e.g., following laparoscopic cholecystectomy), thrombosis, toxemia of pregnancy, polyarteritis nodosa, and emboli which may be bland, iatrogenic (e.g., following angiography or transarterial chemoembolization), or septic, which are almost always associated with infective endocarditis, as in this case. On presentation, hepatic infarction may result in epigastric or right upper quadrant pain in addition to fever, nausea and vomiting. Alternatively, hepatic infarction may be asymptomatic, detected only by biochemical tests and imaging studies. In the case of our patient, the initial CT with contrast demonstrated occlusion of the left hepatic artery with a small area of indistinct hypoattenuation in the posterior aspect of the left lobe of the liver, segment three (Figure 1, arrow), as well as several chronic-appearing renal infarcts. As there was no clear evidence of infarction on presentation and no prior abdominal CT for comparison, the timing of arterial occlusion and its relationship to the presenting complaints were uncertain, and the patient was admitted. Esophagogastroduodenoscopy performed on the day of admission was normal. On the second day of hospitalization, the patient’s abdominal pain intensified and routine laboratory tests were repeated with noted elevation in aspartate aminotransferase (145 U/L) and alanine aminotransferase (179 U/L). Repeat CT imaging showed occlusive thrombus in the left hepatic artery now with a large, wedge-shaped area of hypoattenuation consistent with infarction in the territory of the left hepatic artery (Figure 2, arrow). Recurrent emboli despite appropriate medical therapy represent a class IIa indication for surgical repair of a native heart valve. The risk of embolization with serious consequences in the setting of infective endocarditis is thought to be small and declines rapidly following antibiotic therapy, and though there are currently no data clearly showing a threshold for embolic events above which surgical intervention must be pursued, more than one instance of embolization while a patient is on appropriate therapy generally warrants surgical intervention. Our patient’s mitral valve replacement surgery was expedited on this basis and performed without complications.

Abdominal pain attributable to liver disease is encountered with malignancy, congestive hepatopathy, cystic disease and acute hepatitis, and is thought to be mediated primarily by the generation of visceral afferent signals transmitted within sympathetic nerves as a result of mechanical stretch of Glisson’s capsule. In symptomatic cases of hepatic infarction, pain is presumably secondary to irritation of these...
fibers. Additionally, presumptive nociceptive fibers within the hepatic parenchyma have been described by some authors, but these have not been extensively documented. Given the relatively low incidence of hepatic infarction in the general population and potential for vague or even asymptomatic presentation, a high clinical suspicion must be maintained when abdominal pain is encountered in the appropriate clinical scenario in order to promptly diagnose this condition.

References