dependent on the anterior descending artery[2,3]. Ventricular aneurysm develops within two to ten days after AMI, becoming apparent in the first year after the infarction, with an incidence of 30–35% in patients who have experienced AMI[4–6]. As a secondary finding, intracavitary thrombus affects approximately 40–60% of patients[4] and results from the inflammatory process in the endocardial region affected by the AMI, being associated with the hypokinesia and hypercoagulability existing in the infarction, increasing the risk of a thromboembolic event after the third month in patients with ventricular aneurysm. There is a broad range of symptoms in LV aneurysms, ranging from none to dyspnea, heart failure, or angina, as well as severe manifestations such as acute pulmonary edema, thromboembolism, and ventricular rupture[5–7]. In the treatment of severe refractory cases, surgical procedures, such as plication, excision/suture, imbrication, and patch interposition, are indicated[8]. In the case presented here, despite the extensive area of left ventricular dyskinesia with aneurysm formation and adherent intracavitary thrombus, the patient remained asymptomatic, an uncommon presentation in large aneurysms, which was diagnosed only through CCTA, a noninvasive method that not only allows the diagnosis to be made but also provides accurate measurements and can be used in the postoperative follow-up[1,4–6,9–11]. Routine screening tests, such as echocardiography, often fail to assess the apex of the LV, even with a good access window[1,2,7]. In addition to allowing the diagnosis to be made, the CCTA findings promoted patient adherence to the treatment.

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Bouveret syndrome and its imaging diagnosis

Dear Editor,

An 84-year-old female patient with hypertension reported pain in the upper abdomen accompanied by immediate postprandial nausea and vomiting, without gas or stool elimination for three days. The physical examination showed that she was afebrile, with a distended abdomen, pain upon deep palpation of the upper abdomen, and no signs of peritoneal irritation. A conventional X-ray of the abdomen (Figure 1A) showed air in a branched configuration in the hepatic projection and an air-fluid level in the gastric chamber. Ultrasound (Figure 1B) showed intrahepatic and extrahepatic bile ducts of normal size and the presence of pneumobilia, with no gallbladder identified. There was distension of the gastric chamber, the distal segment appearing to be adhered to the hepatic hilum, as well as a calculus in the pyloric antrum, suggesting the diagnostic hypothesis of gastric obstruction by a gallstone. For better diagnostic elucidation and evaluation of possible complications, we performed computed tomography of the abdomen (Figures 1C and 1D), which demonstrated a correlation with the ultrasound findings, confirming the imaging diagnosis of Bouveret syndrome.

Bouveret syndrome is a rare cause of gastric outlet obstruction due to large-scale impaction of a large gallstone in the duodenal bulb/pylorus after migration through a cholecystoduodenal/cholecystogastric fistula[1]. Gallstone ileus is a disease that mainly affects women, and the pathophysiology is often explained by a previous episode of acute cholecystitis[2]. The incidence is highest in elderly individuals with comorbidities or biliary tract diseases. The following distribution of gallstone ileus sites has been described[1,3]: terminal ileum, 60%; proximal ileum, 24%; distal jejunum, 9%; colon and rectum, 2–4%; distal duodenum, 1–3%; and, less frequently, the proximal portion of the duodenum, where it causes immediate obstruction of emptying. Of all cases of gallstone ileus, 1–3% result from impacted stones in the pyloric or duodenal region, a condition known as Bouveret syndrome.

The diagnosis of Bouveret syndrome can be suspected on the basis of conventional X-ray findings, especially Rigler's triad (Rigler's sign), which is pathognomonic of gallstone ileus and appears in 40–50% of the cases in which conventional X-ray is employed. Rigler's triad is the combination of dilated loops with an air-fluid level, ectopic biliary lithiasis, and gas in the biliary tract[4]. Contrast-enhanced imaging of the upper digestive tract may be useful, with visualization of a filling defect, corresponding to the gallstone, and contrast enhancement of the orifice of the cholecystoduodenal/cholecystogastric fistula[3,5]. Ultrasound can show pneumobilia, gastric distension, and dilation of intestinal loops, as well as sometimes showing gallstones. Rigler's triad is most commonly seen on tomography scans, on which aerobic and gastric chamber dilatation are easily identified and the fistula can be diagnosed after administration of oral contrast, characterizing its leakage, or indirectly by the identification of contrast enhancement within the gallbladder[3]. Although prompt

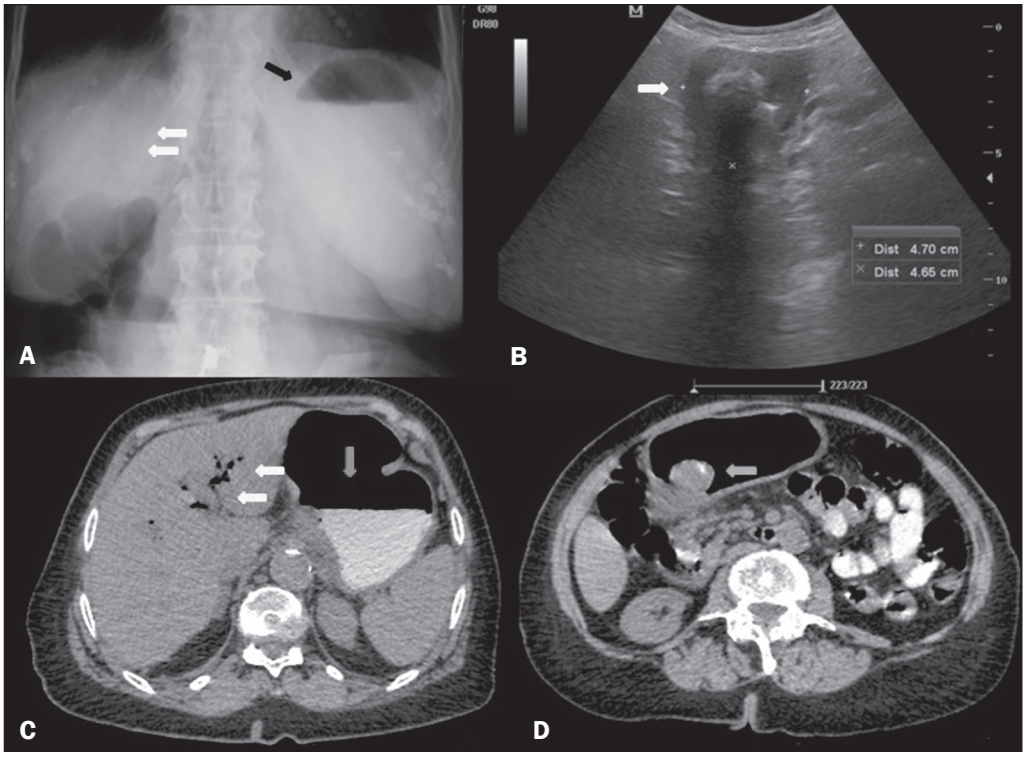

Figure 1. A: X-ray of the abdomen, with the patient standing, showing an air-fluid level in the stomach (black arrow) and intrahepatic pneumobilia (white arrows). **B:** Abdominal ultrasound showing the pyloric-duodenal region with a calculus impacted in its interior (arrow). **C:** Oral contrast-enhanced computed tomography of the abdomen, showing an air-fluid level in the stomach (vertical arrow) and gaseous content in intrahepatic biliary tracts/pneumobilia (horizontal arrows). D: Oral contrast-enhanced computed tomography of the abdomen, showing a mixed-density calculus in the pyloric region (arrow), causing upstream obstruction and dilation (i.e., of the stomach).

diagnosis can promote the rapid extraction of a gallstone, mortality remains relatively high, especially among elderly patients and patients with comorbidities, because the extraction requires surgical intervention[6,7].

Although Bouveret syndrome is a relatively rare disease, the diagnosis can be made on the basis of the imaging findings, thus allowing early endoscopic and surgical intervention[1–8].

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Stress fracture and osteomyelitis in a patient with systemic lupus erythematosus

Dear Editor,

A 38-year-old woman who had been diagnosed with severe systemic lupus erythematosus (SLE) 15 years prior, had refractory nephritis, and had been treated with high-dose corticosteroids and immunosuppressive drugs (cyclophosphamide, mycophenolate mofetil, and rituximab), presented with a several-week history of pain and edema on the dorsum of the left foot after wearing tight shoes. She had extremely low bone density, which had been treated with bisphosphonate and teriparatide. Magnetic resonance imaging (MRI) of the left foot showed a diaphyseal fracture of the second metatarsal with extensive fluid collection and peripheral contrast enhancement of the surrounding tissue, indicating an abscess (Figure 1). In addition, bone marrow edema of the second metatarsal with gadolinium enhancement suggested osteomyelitis.

Stress fractures may occur in SLE patients treated with corticosteroids, most commonly in the femoral head but also in the foot[1]. Atraumatic metatarsal stress fractures typically occur in association with antiphospholipid syndrome. Although the pathogenesis remains uncertain, it likely involves high bone strain and repetitive submaximal stress, causing microfractures and microinfarcts[2–5]. Other possible contributory factors include vasculopathy of the vessels supplying the bones and osteoporosis. Osteoporosis is usually observed in SLE patients, increasing the risk of fractures, and its pathogenesis is multifactorial. High disease activity and immobility are also common factors that substantially increase fracture risk in these patients, as do other factors such as age, body mass index, and gender.