

## Images in EUS

## Gastric Ulcer Penetration into the Liver Mimicking Malignancy on Endoscopic Ultrasound

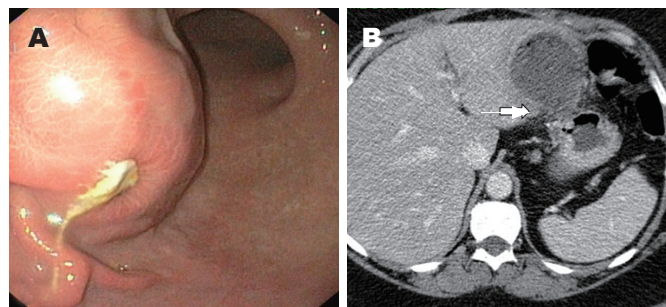
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A 55-year-old woman with no prior medical history was admitted due to worsening abdominal pain. Symptoms were aggravated by food but no radiation of pain, weight loss, or fever was described. Laboratory tests, including liver biochemistry, serum amylase and tumor markers, were only remarkable for elevated white blood cells ( $19 \times 10^3/\mu\text{L}$ ) and inflammation markers (erythrocyte sedimentation rate: 97 mm/h, C-reactive protein: 32 mg/L). Endoscopy revealed a submucosal mass with central ulceration on the anterior gastric wall (Fig. 1A) and computed tomography confirmed the presence of a bulky hypodense tumor between the stomach and the liver (Fig. 1B). Endoscopic ultrasound (EUS) demonstrated hypoechoic thickening of the gastric wall (25 mm), disruption of normal layer configuration and breaching of the serosa with extraluminal growth into the liver (Fig. 2A-B, arrows). Contrast-harmonic EUS (CH-EUS) was performed with a radial echoendoscope (GF-UE160, Olympus Corp, Tokyo, Japan) in conjunction with an Aloka alfa-10 ultrasound processor (Aloka, Tokyo, Japan). After a full vial of Sonovue (4.8 mL) was injected, contrast uptake, distribution and washout were evaluated for 120 s using a mechanical index (0.26). While an inflammatory mass would be expected to be hyperenhanced, CH-EUS displayed a hypoenhanced gastric lesion with inhomogeneous pattern, suggestive of lymphoma or cancer (Fig. 2C). However, biopsies from the ulcer's borders and EUS-fine needle aspiration yielded chronic inflammatory cells with no signs of malignancy.

Due to deterioration of the pain and lack of a definite diagnosis, the patient was referred to surgery in which a voluminous tumor originating from the stomach and



**Figure 1.** A: Upper endoscopy revealed a round submucosal lesion with central ulceration on the anterior wall of the gastric body; B: Abdominal computed tomography showed a hypodense mass involving the stomach and the liver (arrow).

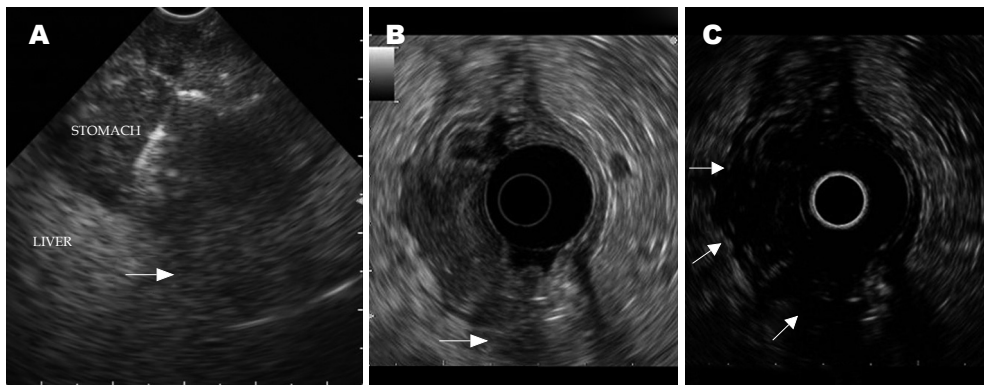
infiltrating the liver and the omentum was resected. Histology revealed an active benign gastric ulcer with excessive inflammatory-fibrosing reaction penetrating the gastric wall and the liver (Fig. 3). Immunohistochemistry for epithelial, hepatocytic, mesenchymal tumors and lymphoma was negative.

Penetration of benign gastric ulcers into adjacent organs (liver, pancreas, heart) has previously been described,<sup>1,2</sup> the first case of liver penetration being an autopsy finding reported in 1880.<sup>3</sup> In recent reports, hepatic involvement was readily demonstrated by the presence of hepatic tissue on endoscopic biopsies or presented as a subcapsular liver abscess.<sup>4,5</sup> A pattern of extensive inflammatory-fibrotic tissue formation mimicking a pseudotumor, as in the present case, is rare. Its EUS features closely resemble malignancy and clinicians' awareness is warranted not to misinterpret them in the absence of histology. Discrimination between benign and malignant gastric ulcers, based solely on EUS findings, may not be straightforward since thickening of the deep wall layers and disruption of layer architecture can be seen in

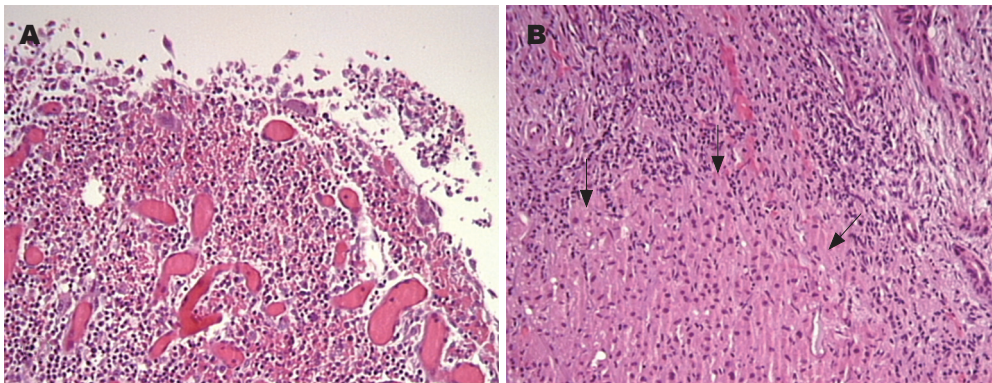
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**Figure 2.** A: Endoscopic ultrasound-guided fine needle aspiration (EUS-FNA) of the lesion; extraluminal growth of the mass, adhering to the liver, is also observed (arrow); B: Thickening of the gastric wall, disruption of layer configuration and breaching of the serosa (arrow) was found on radial EUS, B-mode; C: Contrast harmonic EUS displayed a hypoechoic, inhomogeneous pattern of uptake (arrowheads).



**Figure 3.** Histopathology of the surgical specimen (x10). A: the gastric wall presents a mixed population of inflammatory cells - granulocytes, lymphocytes, plasmocytes and macrophages - and total loss of epithelial cells; B: The inflammatory and fibrotic tissue (upper part of the image) creates a tight adherence (arrows) to the hepatic tissue (lower part). Staining for CD 3, 10, 20, and 138; cytokeratines 8 and 19; cyclin D1; Ki-67 was negative.

both cases.

## DISCLOSURE

All authors have no conflicts of interest, relevant to this paper, to disclose.

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