Although extrinsic compression of the gastric wall by an intact splenic artery is a common observation, the characteristic endoscopic ultrasound (EUS) findings of small splenic artery aneurysm (SAA) have not yet been established.

We present four symptom-free patients who were diagnosed as having extragastrentric compression from a small SAA in the early stage, at the posterior wall of the fundus, by EUS.

Table 1 summarizes the clinical characteristics of the patients and the findings of the various investigations. All patients underwent esophagogastroduodenoscopy (EGD), EUS using an electronic radial scanning echoendoscope (EG-530UR, Fujifilm Corp., Saitama, Japan) with color and power Doppler flow-mapping capabilities, and three-dimensional spiral computed tomographic angiography (3D-CTA) using intravenous contrast agents. The final diagnosis was based on the EUS and 3D-CTA findings and the results of the clinical follow-up (5–16 months, mean 12 months).

Screening EGD seemed to reveal a submucosal tumor on the posterior wall of the fundus in all the patients (Fig. 1). However, EUS revealed a normal gastric wall compressed by a focally dilated aneurysm (Fig. 2), and an arterial pulsation signal was detected by pulse-wave Doppler ultrasound (Fig. 3). 3D-CTA revealed these submucosal masses to be small SAAs. Patient 2 had an aneurysm (15-mm diameter) at the hilum of the splenic artery (Fig. 4). There was no change in the SAAs in any of the patients at a 3-month follow-up with 3D-CT.

SAA is the most common visceral artery aneurysm [1, 2], and although asymptomatic when small, 3%–10% of SAAs are at risk for rupture [3, 4]. Aneurysms should be considered in the differential diagnosis of endoscopically detected submucosal lesions to avoid potentially
harmful outcomes of EUS-guided fine needle aspiration or biopsy. EUS may be a reliable initial diagnostic modality for the diagnosis of even small SAAs (≤15-mm diameter), primarily to differentiate between true submucosal tumors and extrinsic compression of the gastric wall caused by normal or pathological structures.

Competing interests: None

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N. Higuchi1, K. Akahoshi1, K. Honda1, N. Matsui1, M. Kubokawa1, Y. Motomura1, K. Nakamura2, R. Takayanagi2
1 Department of Gastroenterology, Aso Iizuka Hospital, Iizuka, Japan
2 Department of Medicine and Bioregulatory Science, Graduate School of Medical Sciences, Kyushu University, Fukuoka, Japan

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Bibliography
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Corresponding author
K. Akahoshi, MD, PhD
Department of Gastroenterology
Aso Iizuka Hospital
3-82 Yoshio
Iizuka 820-8505
Japan
Fax: +81-948-298747
kakahoshi2@aol.com