





Focal Nodular Hyperplasia of the Liver Mimicking Gastric Submucosal Tumor

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ABSTRACT

Focal nodular hyperplasia (FNH) is a common benign hepatic tumor, typically featuring a central scar with a spoke-wheel pattern on abdominal ultrasound. This case highlights a rare presentation of FNH causing extragastric compression that mimicked a gastric submucosal tumor on esophagogastroduodenoscopy.

A 32-year-old nulliparous woman presented with fatigue. She had experienced symptoms of Long COVID for more than 2 years and was admitted for further diagnostic evaluation of the underlying etiology. Physical examination and vital signs were unremarkable. Esophagogastroduodenoscopy (EGD) revealed a smooth, submucosal bulge on the midgastric body (Figure 1A). Abdominal ultrasound (AUS) identified a central scar with the spoke-wheel pattern (Figure 1B). Gadoxetic acidenhanced magnetic resonance imaging (MRI) of the abdomen demonstrated a well-circumscribed hepatic lesion in the left lobe causing extragastric compression, with homogeneous arterial-phase hyperenhancement (Figure 1C) and subsequent isoenhancement in the delayed phase (Figure 1D), with the central region showing no enhancement in either phase. The lesion was diagnosed as focal nodular hyperplasia (FNH). As she was asymptomatic from the FNH, we opted for conservative follow-up. After several months of outpatient follow-up, the patient reported no abdominal distention or pain suggestive of FNH progression or worsening extragastric compression.

FNH is the second most common benign hepatic tumor after hemangioma, occurring predominantly in women and often detected incidentally on imaging [1]. FNH is largely regarded as a hyperplastic reaction to a prior vascular lesion. The presence of this condition has been reported in pediatric patients with a history of various primary malignancies [2]. The characteristic histology of FNH features a central, stellate scar made of dense fibrous tissue and abnormally thick-walled arteries. Radiating from this central scar are fibrous septa containing abnormal vessels, which create the spoke-wheel pattern seen in imaging such as AUS. Gadoxetic acid-enhanced MRI offers greater diagnostic accuracy, while contrast-enhanced CT can serve as an alternative modality.

The differential diagnosis for submucosal lesions of the stomach is broad, most commonly including gastrointestinal stromal tumors (GISTs) and leiomyomas. On EGD, neither lesion type has specific characteristics, although GISTs may sometimes present with ulceration. On endoscopic ultrasound,

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Key Clinical Message

Focal Nodular Hyperplasia can cause extragastric compression that mimics a gastric submucosal tumor. Therefore, clinicians should include extragastric compression in the differential diagnosis when endoscopic findings resemble a submucosal tumor.

GISTs typically appear as hypoechoic masses arising from the fourth layer, while leiomyomas also appear hypoechoic but can arise from the second, third, or fourth layers [3].

Conservative management is recommended for asymptomatic patients with FNH, as lesions may regress or even disappear during long-term follow-up. Surgical intervention is considered if symptomatic or if malignancy cannot be excluded [1].

Extragastric compression can present as submucosal-like lesions. Extragastric compression identified during EGD necessitates ruling out malignancies such as pancreatic cancer or hepatocellular carcinoma. Reports of FNH causing extragastric compression are rare, as the lesion typically arises in the right hepatic lobe. However, this complication can occur when the FNH is located on the surface of the left lobe.

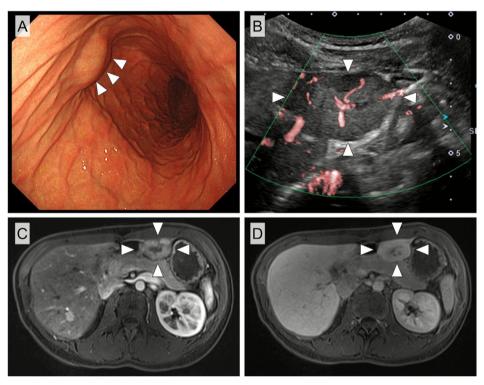


FIGURE 1 | (A) Esophagogastroduodenoscopy showed a smooth, submucosal bulge in the mid-gastric body. (B) Abdominal ultrasound identified a central scar with a spoke-wheel pattern. Gadoxetic acid-enhanced magnetic resonance imaging of the abdomen demonstrated a well-circumscribed hepatic lesion in the left lobe causing extragastric compression, with homogeneous arterial-phase hyperenhancement (C) and subsequent isoenhancement in the delayed phase (D).

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Author Contributions

Yohei Masuda: conceptualization, data curation, formal analysis, project administration, writing – original draft. **Yuki Otsuka:** formal analysis, writing – review and editing. **Fumio Otsuka:** supervision, writing – review and editing.

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Ethics Statement

Written informed consent was obtained from the patient to publish this case report in accordance with the journal's patient consent policy.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Data sharing not applicable to this article as no datasets were generated or analyzed during the current study.

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