



Gastric outlet obstruction caused by focal nodular hyperplasia of the liver: A case report and literature review



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ABSTRACT

INTRODUCTION: Here, we present a case of gastric outlet obstruction due to focal nodular hyperplasia of the liver.

PRESENTATION OF CASE: A 23-year-old female presented to our emergency clinic with nausea, vomiting, and abdominal pain. Endoscopy showed that the prepyloric region of the stomach was externally compressed by a lesion. Computed tomography and magnetic resonance imaging revealed a 70 mm solid mass originating from the liver, extending caudally in an exophytic manner, and compressing the stomach. Laparotomy revealed an irregular and exophytic mass originating from the liver, which caused gastric outlet obstruction. The mass was resected with a 10 mm safety margin. The histopathology report of the mass returned as focal nodular hyperplasia.

DISCUSSION: Gastric outlet obstruction is a clinical syndrome characterized by abdominal pain, nausea, and postprandial vomiting. This clinical condition frequently develops as a result of peptic ulcer disease, pyloric stenosis, and obstruction of pylorus by foreign bodies including phytobezoars, congenital duodenal webs, malignant disorders, and various lesions externally compressing the stomach. Gastric outlet obstruction due to hepatic lesions is extremely rare; few cases have been reported.

CONCLUSION: This is the first reported case of gastric outlet obstruction that developed due to focal nodular hyperplasia of the liver.

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1. Introduction

Gastric outlet obstruction (GOO) is a constellation of symptoms of abdominal pain, nausea, and postprandial vomiting. The main etiologic factors of GOO include spasm of the gastric outlet, namely pylorus and pyloric obstruction by foreign bodies or benign/malignant lesions. The majority of these factors originate from the stomach or duodenum, although GOO may also develop due to external pyloric compression by benign/malignant lesions of adjacent organs.^{1–4}

Development of secondary GOO due to lesions originating from liver is rare. A comprehensive literature search from PubMed, Medline, and Google Scholar yielded only four case reports of GOO originating from the liver, while a similar search about development of GOO secondary to focal nodular hyperplasia resulted in zero publications.^{2,5–8} Focal nodular hyperplasia (FNH) is a common type of liver tumor, second only to hemangiomas in prevalence. Most cases of FNH are asymptomatic and are discovered

incidentally during physical examinations, abdominal surgeries, or autopsies.³ In this study, we present a case of GOO due to FNH of the liver.

2. Case report

A 23-year-old married female presented to our emergency clinic with complaints of nausea, vomiting without bile, abdominal pain, and bloating. The symptoms had been present for six months but recently worsened. In addition to these symptoms, she also lost 5–6 kg. Abdominal inspection revealed no pathology while palpation identified a mass of epigastric origin that extended to right hypochondrium. Complete blood count, liver function tests, hepatitis panel, and α -fetoprotein levels were normal. A contrast-enhanced abdominal CT scan was performed following a three-day fluid resuscitation and nasogastric decompression. The abdominal CT showed a 70 mm \times 65 mm lobulated mass with a dense contrast uptake, which was originating from segment 4b of the liver, extending to antero-inferior rim of liver (Fig. 1). Endoscopy revealed the relationship of the mass with the stomach following the abdominal CT scan. Endoscopy also showed that the prepyloric region of stomach compressed by externally lesion. The MRI examination

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Fig. 1. Contrast-enhanced abdominal CT showed a nearly 70 mm × 65 mm lobulated mass with a dense contrast uptake, which was originating from segment 4b of the liver, extending to the antero-inferior rim of liver.

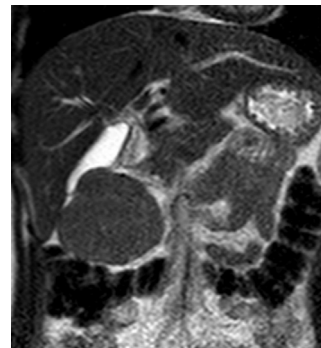


Fig. 2. MRI examination revealed a 78 mm × 55 mm × 56 mm solid mass originating from the liver and extending caudally in an exophytic manner.

revealed a 78 mm × 55 mm × 56 mm solid mass originating from the inferior rim of segment 4b of liver, extending caudally in an exophytic manner (Fig. 2). The lesion appeared hypointense on T1-weighted images and mildly hyperintense on T2-weighted images. Radiologic and endoscopic findings showed that the external compression of the stomach was due to the liver mass. Based on these findings, a laparotomy was performed detecting an irregular mass originating from the immediate vicinity of the falciform ligament. The lesion was adhered to the gall bladder and exerted external compression on stomach at the level of pylorus (Fig. 3A–C). Cholecystectomy was performed after the dense adhesions between the compressive tumoral lesion and gall bladder were removed. Then, the tumoral mass was removed together with a 10 mm intact liver tissue (Fig. 3D). No complications developed after the surgery. The mass was confirmed to be focal nodular hyperplasia by histopathological and immunohistochemical studies.

3. Discussion

Gastric outlet obstruction is a constellation of symptoms that develops as a result of any disease process that poses a mechanical obstacle to gastric emptying. Independent from the etiologic factors, the most common symptoms are abdominal pain, nausea, and postprandial vomiting without bile. The pattern and the rate of symptom onset, on the other hand, may depend on the underlying pathology. As in our case, weight loss may also be observed in cases when the obstruction develops slowly. The basic pathophysiologic mechanism of GOO is an obstruction of pyloric canal or proximal duodenum by intrinsic or extrinsic (external compression) factors. The malignant causes of GOO are upper gastrointestinal cancers. The benign lesions causing GOO are peptic ulcers, gastric polyps, caustic material ingestion, pyloric stenosis, duodenal web, gallstone obstruction (Bouveret's syndrome), gastric tube complications, pancreatic pseudocysts, and various bezoars. In addition to

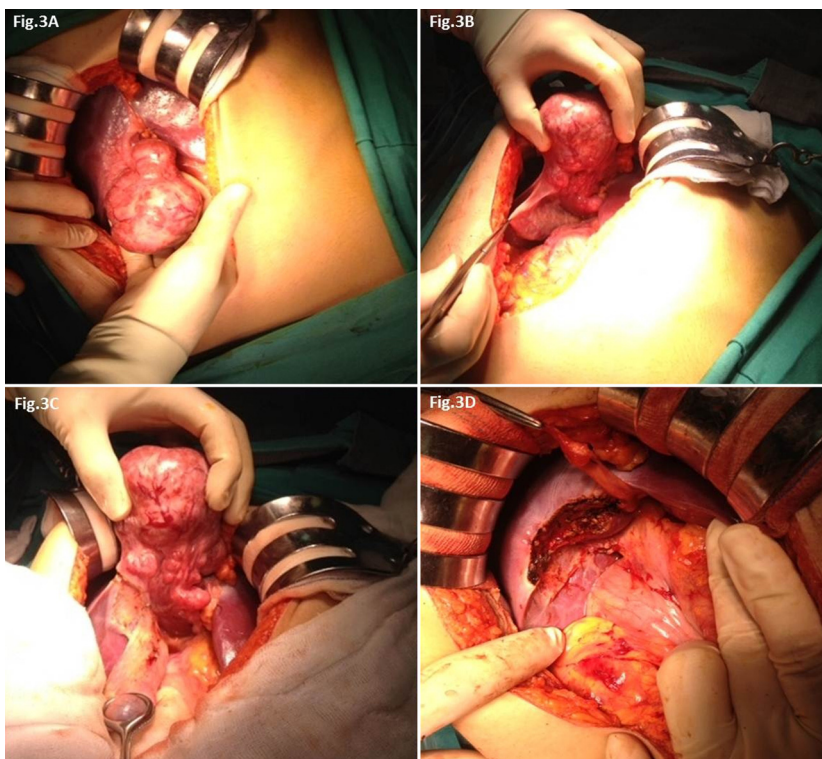


Fig. 3. Intraoperative images. The general appearance of the exophytic mass that was originating from liver and compressing pylorus from outside (A), the image of the dense adhesions between the mass and gall bladder (B), removal of the adhesions between the gall bladder and the mass with blunt and sharp dissections (C), the image of liver after the mass was resected (D).

these classical factors, there are also rare causes including the presence of a Riedel's lobe, exophytic giant hemangioma of the liver, worm ball, or amoebic liver abscesses originating from liver.^{1–6} There are no published case reports establishing FNH as a secondary cause of GOO.

Focal nodular hyperplasia constitutes eight percent of primary liver tumors and is the second most common type of liver tumor.^{3–5} While it is common in women and men of all age groups, it is 8–12 times more prevalent in women.⁵ It is most common between individuals between 30 and 50 years of age. FNH was originally known as a hamartoma, neoplasm, a response to ischemia, or a focal area of regeneration. However, it has recently been classified as that it is a regenerative (hyperplastic) response to hypoperfusion created by the nodule around the artery located at the center of the liver. The actual etiopathogenesis of FNH remains unclear.

The patient in this case report used oral contraceptives for the last year. While the relationship between FNH and oral contraceptives is not completely understood, conflicting data does exist suggesting FNH development correlates with estrogen and progesterone. Further, the predominant view in the literature suggests that long-term oral contraceptive use and pregnancy may lead to an increase in nodule size or cause an asymptomatic lesion to become symptomatic.^{3,5} In contrast, existence of the disease in men and children, and in women years before the invention of oral contraceptive drugs indicate that such a relationship cannot be established.

The majority of patients with FNH are asymptomatic and FNH is usually detected incidentally during physical examination, radiologic examinations, autopsies, or laparotomic operations performed for other indications. Symptomatic cases may present with abdominal pain, nausea, and vomiting. Unlike other liver tumors, hemorrhage, necrosis, and infarction are very rare. Therefore, circumstances like spontaneous rupture or intraperitoneal bleeding that necessitates urgent surgical intervention are extremely rare.⁶ Furthermore, liver function tests are almost always normal unless intrahepatic or extrahepatic bile ducts or hepatic vascular tree are subject to external compression.⁵

Diagnosis of FNH is generally made by showing the characteristic features in radiological imaging studies and ruling out other potential causes such as hepatic adenoma, HCC, fibrolamellar HCC, large regenerative nodules, hemangioma, and hypervascular metastases.⁵ Lesions are usually well-demarcated, homogenous, and hypoeschoic or isoechoic and sometimes hyperechoic when viewed using ultrasonography. CT imaging studies should include a non-contrast phase, an arterial phase, and a portal venous phase. The lesion that is hypodense or isodense at the non-contrast phase becomes hyperdense at the arterial phase, which indicates the hypervascularity of FNH. When visualized using MRI, the lesion usually looks isointense or mildly hypointense in T1-weighted images, and isointense or mildly hyperintense in T2-weighted images. The sensitivity and specificity of MRI can be increased using dimeqlumin.^{4,5} FNH is composed of hepatocytes, bile ducts, and Kupffer cells, and it is characteristic for Kupffer cells to cause the lesion to appear as a hot spot at a ratio of 70–80% in Tc-sulfur colloid SPECT examination.

The most appropriate approach to asymptomatic patients is conservative management. A close follow-up is very important

in patients contemplating pregnancy or prescribed oral contraceptives. Certain criteria must be established in managing symptomatic cases. According to these criteria, surgery should be performed when patients are symptomatic, a radiological change in lesion size occurs, refractory and resistant symptoms are present, compression of biliary or vascular tree of liver presents, localization of the tumor is atypical, or the tumor is indistinguishable from other liver tumors by radiology.⁵ Our decision to perform surgery on this patient was based on the presence of the exophytic extension of the lesion and the obstruction caused by pyloric compression.

In conclusion, disorders of liver rarely cause gastric outlet obstruction. However, it should be considered in the case of gastric outlet obstruction differential diagnosis.

Conflict of interest

The authors declare that there is no conflict of interest.

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None.

Ethical approval

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Author contributions

Eris C., Odabasi M. and Yildiz M.K. performed the surgical procedure. Akbulut S., Abuoglu H. and Ozkan E. contributed to a comprehensive search and review of the literature, as well as writing the article. Akbulut S. and Eris C. designed and prepared the manuscript.

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