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Case Report

Hepatogastric fistula: A rare complication of liver abscess

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Abstract

Rupture of amebic liver abscess into stomach is a rare complication. We report a case of a young male presenting with haematemesis due to a rupture of left lobe amebic liver abscess into stomach. We discuss the diagnosis and management of this rare clinical entity.

Key words

Amebic liver abscess, hematemesis, hepatogastric fistula

Introduction

Amebic liver abscess is a common cause of febrile illness in India. It usually presents with fever and right upper quadrant abdominal pain.[1] Upper gastrointestinal bleeding is not a commonly described clinical presentation of liver abscess. In liver abscess, upper gastrointestinal bleeding would usually be attributed to nonsteroidal anti-inflammatory drug-induced esophagitis, erosive mucosal disease, or duodenal or gastric ulcer, as would be expected in any febrile illness. We report a case of a young patient who presented with febrile illness and upper gastrointestinal bleed due to a ruptured amebic liver abscess perforating into the stomach.

Case Report

A 35-year-old male patient presented with sudden onset of massive hematemesis. There were six to seven episodes of vomiting, each containing around 250–300 ml of fresh blood. He had an antecedent history of high-grade fever for past 10 days. It was followed by dry cough and occasional chest pain which was pleuritic in nature. He had consulted a local physician and taken empirical antibiotics and antipyretics for the same.

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On examination, the patient had severe pallor, was febrile, and had a toxic appearance. He was tachypneic, had sustained tachycardia (pulse rate of 110/min) and hypotension (blood pressure of 90/60 mmHg). Per abdominal examination revealed mild tenderness in the epigastric and right hypochondriac regions. Respiratory system evaluation showed a decreased air entry in the left infrascapular and infra-axillary regions with dullness on percussion.

The patient was resuscitated with intravenous fluids and blood transfusion and broad spectrum antibiotics were started. Initial investigations showed an hemoglobin of 4.8 g/dl and total leukocyte count of 25,100 cells/mm^3 (neutrophils 86%). Platelet counts (156,000 cells/mm^3), prothrombin time (14 s, control 12 s), and activated partial thromboplastin time (38 s, control 32 s) were in normal range. Liver function tests and kidney function tests were normal.

Upper gastrointestinal endoscopy (UGIE) was done after initial stabilization. It revealed a large (3 cm × 2 cm) deep penetrating ulcer in the fundus of stomach with adherent clot and no active bleed at the time of examination [Figure 1]. The base of ulcer was quite deep giving an impression of transmural perforation. Considering the clinical presentation and endoscopic findings, the most likely diagnosis seemed to be a perforated gastric ulcer. An urgent contrast-enhanced computed tomography (CT) examination (with oral and intravenous contrast) of the

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abdomen was performed which showed a heterogeneously hypodense lesion in the left lobe of liver with multiple air foci within it. The liver lesion was extending directly into the body of stomach with a small amount of surrounding fluid [Figure 2]. The walls of fundus and lesser curvature of stomach could not be defined. These features were suggestive of rupture of left lobe liver abscess into stomach.

In view of gastric perforation and severe blood loss, the patient underwent an exploratory laparotomy. Intraoperative findings revealed dense adhesions of the left lobe of the liver with the anterior wall of stomach and diaphragmatic surface. There was a 4 cm × 2 cm defect on the anterior wall of stomach just beyond the gastroesophageal junction, along lesser curvature and it was communicating with abscess cavity in the left lobe of the liver [Figure 3]. There was no peritoneal soiling. Purulent material was aspirated from the liver abscess and sub hepatic drain was placed. The margins of the gastric perforation were freshened and primarily repaired with omental patch application.

Postoperatively, the patient was kept on nasojejunal tube feeds for 5 days and broad spectrum antibiotics along with metronidazole were continued. His clinical and laboratory parameters improved. Amebic serology was reported positive and the drained pus was sterile on culture. Normal diet was resumed on the seventh postoperative day and the patient was discharged in stable condition after drain removal.

Discussion

Amebic liver abscess is endemic in India.[2] It is the most common extraintestinal complication of amebiasis with reported prevalence of 3–9%.[3] Though the classical description of an amebic liver abscess is a solitary lesion in the right lobe of the liver, a substantial variation in presenting pattern has been reported. In an ultrasonography analysis of 212 patients of amebic liver abscess, 35% had left lobe abscess.[4]

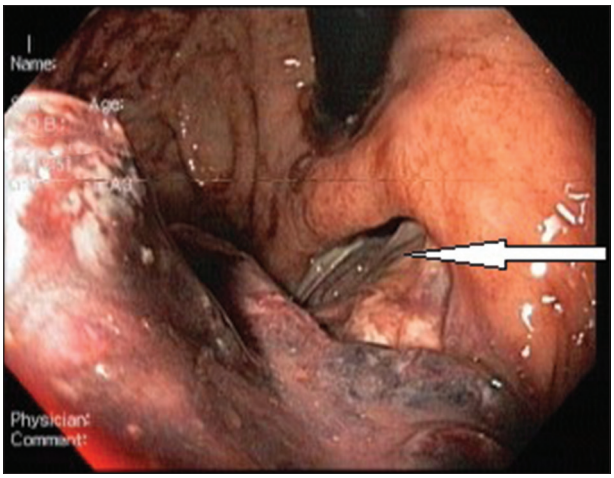

Figure 1: An upper gastrointestinal endoscopic view showing a deep penetrating gastric ulcer seen in the fundus of stomach with adherent clot and no active bleed

Rupture of an amebic liver abscess is a common cause of mortality in amebiasis. It is seen within the first 6 weeks of the disease.[5] Prevalence of rupture in amebic liver abscess irrespective of the site is about 22%.[6] Left lobe liver abscess are more prone to rupture.[7] The site of perforation depends on the location of the abscess in the liver; upper right lobe abscess often perforates in the right pleural cavity and right lung, left lobe abscess may perforate in the left lung, pericardium, or stomach whereas abscess in the lower portion of the liver can extend to the peritoneal cavity, gall bladder, colon, right kidney, or the inferior vena cava. In a large Chinese study on perforated amebic liver abscess in 110 patients,[6] the most common sites of perforation were in the pleural cavity (42.5%), lungs (20.2%), peritoneal cavity (15.6%), subphrenic space (5.7%), and thoracoabdominal wall (5.25%). The unusual sites of perforation included mediastinum, pericardial sac, duodenum, stomach, colon, common bile duct, inferior vena cava, right kidney, and spleen. Gastric perforation was found in only 0.3% of the cases.

The resultant fistulous communication between the abscess cavity in liver with the stomach cavity is termed as a hepatogastric fistula. Such a fistula has also been described following transarterial embolization, radiotherapy for hepatocellular carcinoma, percutaneous radiofrequency thermal ablation of hepatocellular carcinoma, pyogenic liver abscess, iatrogenic injury of the stomach, percutaneous catheter drainage of liver abscess, metal stent placement to decompress an obstructed biliary system in case of benign or malignant biliary obstruction, or by direct infiltration of stomach by hepatocellular carcinoma.[8]

Rupture of liver abscess into the stomach may present with persistent severe abdominal pain,[7,9] recurrent[10] or bilious vomiting,[8] or hematemesis[6] as in our case. Hematemesis in

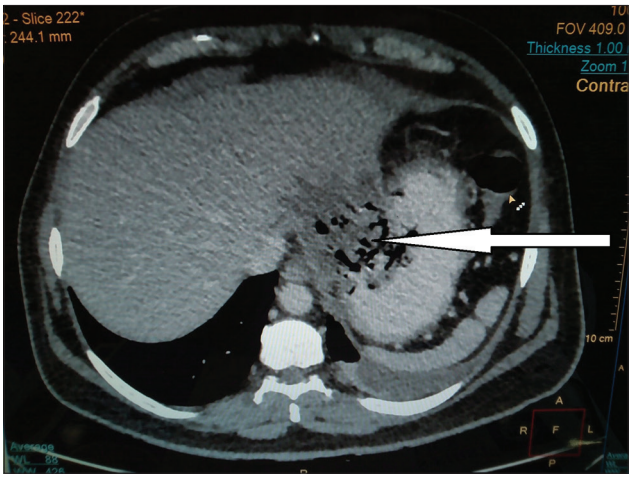

Figure 2: A contrast-enhanced computed tomographic scan of upper abdomen showing a heterogeneously hypodense lesion in the left lobe of liver with multiple air foci within, which are seen to extend into the body of stomach with small amount of surrounding fluid. The walls of fundus and lesser curvature are not well defined

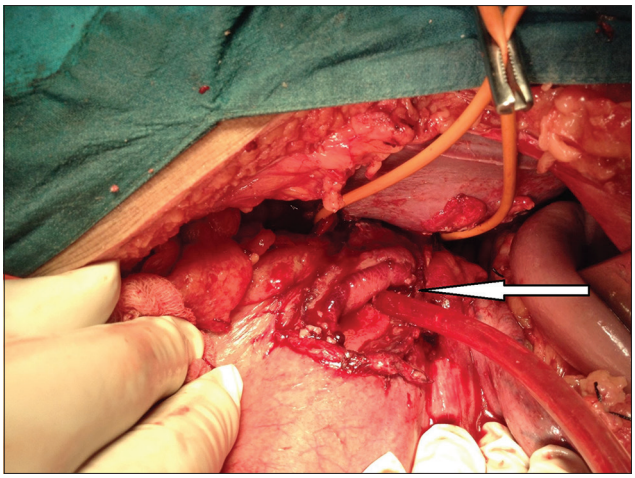

Figure 3: A 4 cm × 2 cm defect on the anterior wall of stomach just beyond the gastroesophageal junction. The edges of defect appear inflamed. Yankauer suction tip is placed in the defect. The left lobe of retracted liver is visible with the site of perforation

these cases is usually due to tearing of the mucosal surface. Involvement of hepatic artery or portal vein branches has not been reported.

In cases of liver abscess rupturing into the stomach, abdominal CT scan and UGIE usually establish the diagnosis. The presence of multiple air foci within the abscess cavity on imaging is a significant suggestive finding as it is seen only in few other conditions such as infection with gas-forming organisms, bland tissue infarction with necrosis, and recent instrumentation or surgery.[11] Endoscopic picture may be mistaken for a gastric ulcer and a high index of suspicion is required for making this diagnosis. Visibility of liver or peritoneum may point to such a complication, but still differentiation from a perforated gastric ulcer can only be made by CT scan.[12]

There is no clear consensus regarding the management of hepatogastric fistula due to rarity of the condition and variable presentation.[9,10] Conservative management includes keeping patient nil per oral, percutaneous drainage of liver abscess, antibiotic therapy, proton pump inhibitors, and decompression of the stomach.[13] A nasojejunal tube may be passed distally for initial feeding purpose or alternatively a distal feeding jejunostomy can be made. Spontaneous closure of the fistula can be anticipated within 4–6 weeks in the majority.[13] Involvement of main hepatic ducts may result in the formation of internal biliary fistula. In such situation, use of endoscopic retrograde cholangiopancreatography and stenting of common bile duct has been reported.[8] If the general condition of the patient permits, a trial of conservative management should be given prior to definitive surgical therapy.[13,14]

Early surgical intervention may be required in patients with peritonitis. Many authors have recommended early surgical intervention for amebic abscess rupturing into the gastrointestinal tract.[15] This may give definitive one stage solution of the problem with closure of perforation. Omental patch repair of gastric perforation done in this patient leads to an early resumption of oral intake. In addition, the presence of massive upper gastrointestinal hemorrhage causing hemodynamic compromise in this case prompted us to go with the surgical option.

Conclusion

Gastric perforation of liver abscess is a very rare complication of liver abscess. Clinical presentation is varied and it may present with upper gastrointestinal bleed. Endoscopic picture usually mimics gastric ulcer and a high index of suspicion is required to make this diagnosis. Diagnosis is confirmed by CT scan and UGIE. A definitive consensus regarding management is still lacking and approach must be individualized according to clinical presentation.

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Conflicts of interest

There are no conflicts of interest.

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