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Intrahepatic Abscess Secondary To Duodenal Ulcer Perforation- Role Of UGI Endoscopy And Literature Review.

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Abstract:
Duodenal ulcer perforation /penetration into liver causing intrahepatic abscess is a rare event. To the best of our knowledge only 6 cases of intrahepatic abscess due to perforated duodenal ulcers has been reported. Our case will be the seventh one. We have presented a case of intrahepatic abscess secondary to a perforated duodenal ulcer diagnosed by endoscopy, which we were able to completely resolve by repeated percutaneous drainage of abscess and anti H. pylori eradication therapy.

Key words: *Intrahepatic abscess, duodenal ulcer, perforated duodenal ulcer*

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I. Background:

The incidence and prevalence of PUD is decreased in recent decades, possibly due to use of proton pump inhibitors (PPI) and *H. pylori* infection eradication therapy.[1] Liver penetration by perforated peptic ulcer (both gastric ulcer and duodenal ulcer) is rare, only several case reports are documented. But not all penetrating duodenal ulcers were associated with intrahepatic abscess or cavity. Intrahepatic abscess following perforated duodenal ulcer is a serious complication and more rarely documented. Endoscopic diagnosis is also very difficult. Here we report a patient with intrahepatic abscess due to perforation of duodenal ulcer disease diagnosed by endoscopy with supportive CT scan evidence.

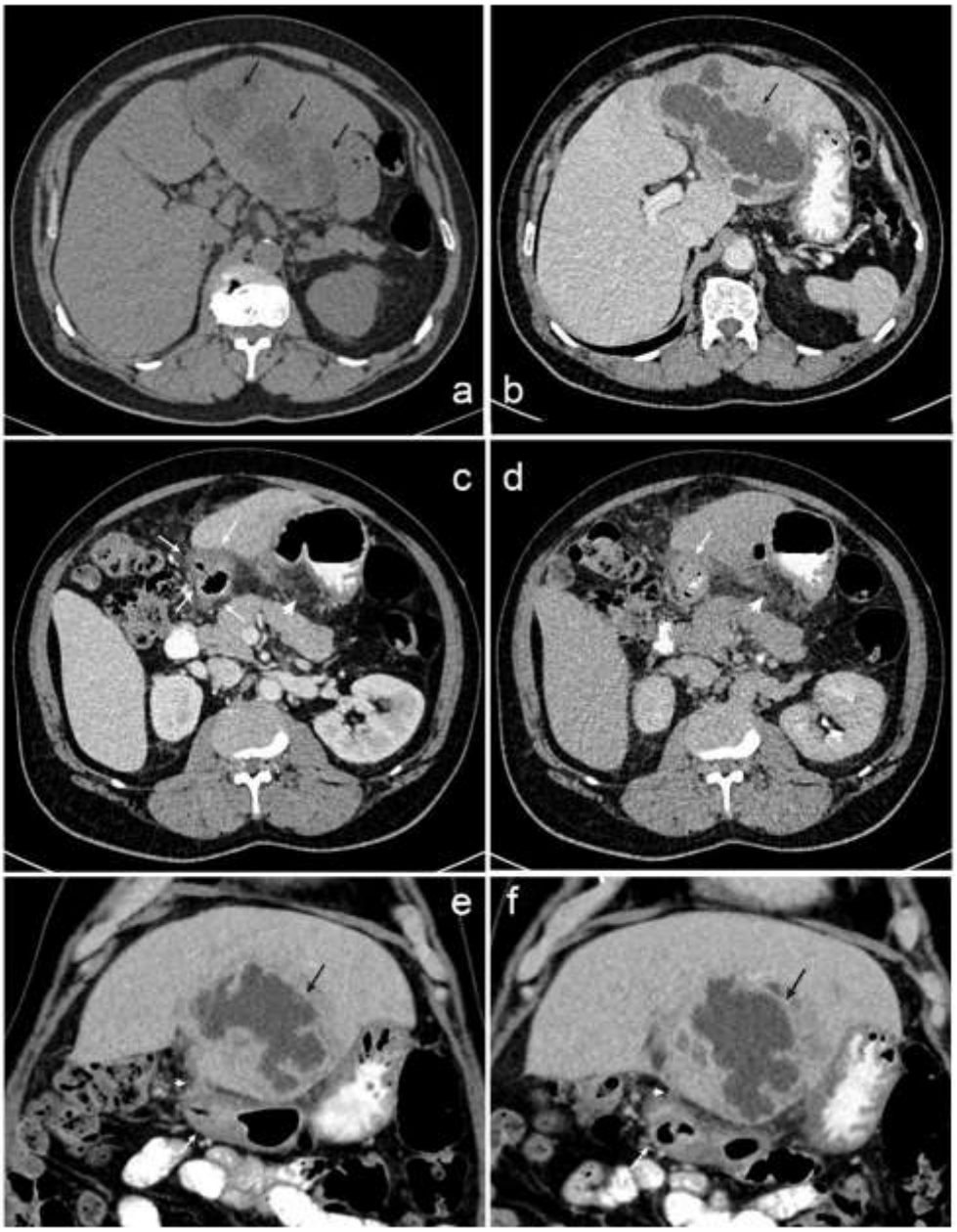
II. Case presentation:

A 62 years male, normotensive, nondiabetic, non smoker, nonalcoholic, Muslim retired service holder, hailing from Jashore, far away from capital city Dhaka, Bangladesh, presented on July, 2023 with history of two episodes of melaena and generalized weakness without any prior history of NSAID drug intake or other systemic complains. At that time investigations revealed Hb-7.4 g/dl (reference range 11-15 g/dl), ESR 110 (reference range 0-10) mm in first hour, Hct-20.5% (reference range: 35-55%), MCV 65.5 fl (reference range:75-100 fl), MCH-23.8 pg (reference range: 25-35 pg), MCHC 36.3 g/dl (reference range:31-38 g/dl). Total WBC and platelet count, liver function tests (LFT), renal function tests (RFT) were within normal range, USG of whole abdomen revealed normal. Patient was treated conservatively with 2 unit of blood transfusion, proton pump inhibitor (PPI), 20 mg, twice daily and was completely alright for next one week after which he developed fever and abdominal pain. Fever was high grade, maximum recorded temperature was 103^0 F, with chills and rigor, continuous, not responding to antipyretic, associated with night sweats, anorexia and abdominal pain in epigastric and right hypochondriac region which was constant, dull ache without any radiation, not related with food and posture. There was no preceding history of jaundice, haematemesis, altered consciousness, altered bowel habit, weight loss, joint pain, skin rash, cough or contact with known TB patient.

On examination, he was ill looking, toxic, mildly anaemic, nonicteric, no peripheral lymphadenopathy, no stigmata of chronic liver disease, temperature was 102^0 F, pulse 110/minute, blood pressure 100/70 mm of Hg, BMI was 22 kg/m^2. Abdomen examination revealed tender soft hepatomegaly without any intrabdominal lump or testicular atrophy. So our clinical diagnosis was hepatic abscess and empirical injectable antibiotic, ciprofloxacin 500 mg, twice daily and metronidazol 500 mg thrice daily were started.

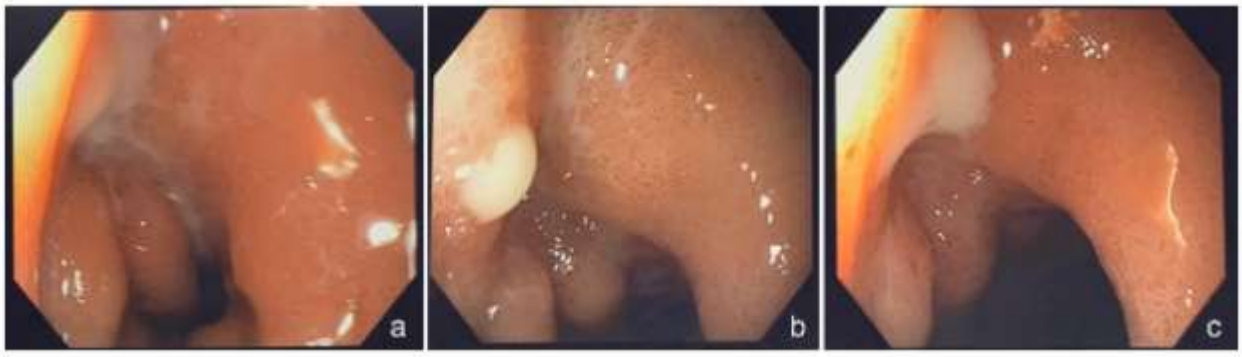
Investigations revealed Hb-8.4 g/dl (reference range:11-15 g/dl), ESR 124 (reference range:0-10) mm in first hour, Hct-31% (reference range:35-55%), MCV 79 fl (reference range:75-100 fl), MCH 25 pg (reference range:25-35 pg), MCHC 32 g/dl (reference range:31-38 g/dl), total WBC count 24,500/Cmm ((reference range: 4,000-11000/Cmm), neutrophils 86% (reference range: 40-75%).. PBF showed normocytic anaemia with neutrophil leucocytosis, LFT, RFT was within normal range. USG of whole abdomen revealed anechoic area

containing internal echoes measured about 97 x 68 mm in size, in left lobe of liver suggestive of liver abscess, rest of hepatic parenchyma appeared normal. CT scan of whole abdomen revealed multiple irregular hypodense lesions in the segment III of left lobe of liver. After contrast peripheral hyperdense rim enhancement of these lesions were observed which were surrounded by hypodense areas representing edema. These are compatible with typical "double target sign" of pyogenic liver abscess. Enhanced thickening of duodenal cap with surrounding fat stranding were also noticed adhering segment III of left lobe of liver suggesting duodenal ulcer perforation involving adjacent liver parenchyma (Panel A); free air in the abdominal cavity was not detected.



Panel A: CT scan of abdomen with noncontrast (a) and contrast (b,c,d,e,f), multiple axial views (a,b,c,d) and coronal views (e,f) showing multiple irregular hypodense lesion (a) with rim enhancement after iv contrast (b,e,f) in segment III, left lobe of liver (black arrows). Duodenal cap (c,d,e,f) is thickened, enhanced (white arrows) with surrounding fat stranding (white arrow head) adherent to segment III, left lobe of liver suggesting perforation.

As patient had initially a history of melaena, we performed UGI endoscopy and duodenal bulb was found to be grossly deformed, had edema and inflammation and frank pus was seen coming out spontaneously from its anterior wall suggestive of duodenal ulcer perforation in healing stage (Panel B). So finally we concluded that actually it was duodenal ulcer perforation which eventually developed liver abscess.



Panel B: Endoscopy showing grossly deformed duodenal bulb (a), frank pus coming out from partially healed ulcer suggesting duodenal ulcer perforation (b,c).

Patient was managed conservatively with antibiotics, proton pump inhibitor and percutaneous drainage of liver abscess followed by anti H. pylori eradication therapy. Initially empirical injectable antibiotics was started (ciprofloxacin 500 mg bd & metronidazol 500mg tds) which was continued for 2 weeks followed by oral antibiotics for next one month. USG guidance 250 ml of foul smelling pus was aspirated, colour of pus was suggestive of pyogenic abscess. After drainage of pus general well being of patient was improving, pain subsided and temperature also touched the base line. Follow up USG revealed irregular cystic lesion containing echogenic debris measuring about 124 x 62 x 103 mm in left lobe of liver. Second aspiration of around 150 ml foul smelling pus was done after 12 days of first aspiration. Aspirated pus was sent for culture sensitivity which revealed no growth.

III. Discussion:

Intrahepatic abscess is an unusual complication of peptic ulcer disease. Less than 10% of all ulcer penetration involved liver and many of these are extensions of subhepatic or perihepatic collection. Liver penetration is usually due to lesser curve gastric ulceration because of gastrohepatic ligament can serve as a pathway. The duodenal bulb lies just inferior to quadrate lobe of liver.[2] Duodenum and portal structures are in direct association with the liver through the hepatoduodenal ligament (inferior aspect of the lesser omentum) and porta hepatis which may explain the pathway of penetration of a duodenal ulcer into liver.

The most common complication of duodenal ulcer is gastrointestinal bleeding. Perforation is less common.[3] Classic triad of duodenal ulcer perforation is sudden onset of abdominal pain, tachycardia and abdominal rigidity (peritonitis). By contrast, the confined perforation of a peptic ulcer is defined as the penetration into and confinement within the tissue of an adjacent structure or organ by peptic ulceration,[4] commonly pancreas, followed by gastrohepatic omentum, biliary tract and liver.[5] It can present in a more latent form, giving rise to an intra-abdominal abscess.[3] Peptic ulcer penetration into the liver is a rare complication that is poorly understood.[5]

In February 2021, by a computerized search from Pubmed, in a systematic review, peptic ulcer penetration/perforation into liver was found a rare event, among 41 publications presenting 42 patents with peptic ulcer (both gastric ulcer and duodenal ulcer), only 20 patients were identified to have liver involvement with a perforated/penetrated duodenal ulcer.[6] Diagnosis was also difficult – usually made during laparotomy or by endoscopic biopsy. Among 42 patients, only 13 cases were diagnosed by the direct observation of a peptic ulcer that perforated into the liver during surgery.[6] To the best of our knowledge, till date only 12 cases of the spontaneous duodenal ulcer penetration to liver have been diagnosed by endoscopy.[7-18]

But not all penetrating duodenal ulcers were associated with intrahepatic abscess or cavity. To the best of our knowledge only 6 cases of intrahepatic abscess due to perforated duodenal ulcers has been reported.[2,8,17,19,20,21] Our case will be the seventh one. Heathfield (1942) described one case (diagnosed post mortem) of liver abscess following definite perforation of ulcer.[19] In 1946 another similar case was recorded which was complicated by suppurative peritonitis, pleural effusion, femoral vein thrombosis.[20] In 1955, another case was confirmed by laparotomy that a massive blood clot in large hepatic abscess cavity due to duodenal ulcer (1.5 cm) perforation on its anterior wall.[21]

Radiological diagnosis of ulcer penetration is difficult.[8] In only eight cases of peptic ulcer disease, movement of oral contrast or air bubble or a fistula formation between liver and stomach or duodenal ulcer could be recorded.[6] Other CT findings include duodenal wall thickening, fat stranding, inflammatory changes in adjacent fat tissues and periduodenal fluid collection.[22,23] which is compatible with our present case.

The mortality rate for duodenal and peptic ulcers were 13.3% and 23.5%, respectively. Mean age of patients with liver penetration due to duodenal ulcer was 62.65 years (95% CI: 53.40–71.90) with slight male

predominance (1.5:1). The majority of the duodenal ulcers occurred in the first portion of the duodenum (n = 17) on its anterior wall[6] which is compatible with our case.

Our case is unique on its documentation of an intrahepatic abscess along with enhanced thickening of duodenal cap with surrounding fat stranding adhering to segment III of left lobe of liver on CT scan in a patient with a prior history of melaena and on endoscopy spurting of pus from anterior wall of an inflamed deformed duodenal bulb confirmed perforation of the duodenal ulcer into the liver as an aetiology of intrahepatic abscess and patients responded with repeated percutaneous aspiration of pus and anti-.H Pylori therapy and PPI without need for surgery.

The endoscopic diagnosis of ulcer perforation is not frequent, because endoscopy is considered an inadequate diagnostic tool by many authors.[24] Insufflation during upper endoscopy could aggravate free perforation and even convert a confined perforation to a free one. With a minimal level of insufflation and an operating theatre on the alert, the role of endoscopy in the management of suspected ulcer perforation could be reconsidered.[13]

We recommend UGI endoscopy in all patient with intrahepatic abscess or intaabdominal abscess in contact with liver or stomach and duodenum for diagnosis of peptic ulcer or duodenal ulcer perforation / penetration into liver as an aetiology of abscess.

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