

Nonoperative Management of a Perforated Peptic Ulcer in the Setting of Decompensated Alcoholic Cirrhosis: A Case Report

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Abstract

Perforation of peptic ulcers is typically considered a surgical emergency, but management must account for patient comorbidities. Surgical management of perforated ulcers in the setting of decompensated cirrhosis can be complicated by coagulopathy and delayed wound healing, leading to increased morbidity and mortality. This case outlines the nonoperative management of a perforated peptic ulcer in a 47-year-old male with decompensated alcoholic cirrhosis. Computed tomography scan revealed pneumoperitoneum, and further imaging showed a large perforated ulcer of the superior fornix of the duodenal bulb. Due to the patient's high preoperative risk, a nonoperative approach was chosen by his multidisciplinary care team. Initial management included nil per os with nasogastric tube decompression, antibiotics, and therapeutic paracentesis. Due to the patient's declining hemoglobin, an urgent esophagogastroduodenoscopy was performed, and the hemorrhage was treated with epinephrine injection and clipping, followed by placement of a percutaneous pigtail catheter adjacent to the site of perforation. An upper gastrointestinal series completed 11 days later showed no leak from the site of perforation. The patient tolerated dietary advancement and continued to improve until discharge on hospital day 36. In this case, a nonoperative approach yielded resolution of the patient's bleeding perforated ulcer and resulted in a favorable outcome for our patient.

Keywords: Peptic ulcer disease, Bleeding peptic ulcer, Decompensated cirrhosis, Alcoholic cirrhosis, Nonoperative management

1. Introduction

Cirrhosis with portal hypertension is associated with a high risk of morbidity and mortality following abdominal surgery. Although elective operations can be delayed or avoided in many cases, emergent surgery is often accompanied by substantial risk. Peptic ulcer perforation is largely considered an operative emergency; however, some case reports and series have reported the feasibility of nonoperative management (1, 2). We describe herein the successful nonoperative optimal medical management of a complex patient with a perforated ulcer in the setting of decompensated alcoholic cirrhosis.

2. Case Description

A 47-year-old Hispanic male with no available medical or surgical history presented to the emergency department with fatigue and progressive abdominal distension for a few weeks, having developed encephalopathy over the preceding days. He reported melena in the distant past but not within the past six months. Further questioning elucidated a history of heavy alcohol use. On evaluation, he was afebrile, hypotensive, and demonstrated a heart rate of 90 bpm. He was jaundiced with visible superficial umbilical varices, and his abdomen was non-tender to palpation with a fluid wave. His laboratory studies revealed the following: hemoglobin, 6.8 g/dL; white blood cell (WBC) count, $18.5 \times 10^9/L$; platelets, $271 \times 10^9/L$; sodium, 126 mmol/L; creatinine, 1.28 mg/dL; albumin, 2.0 g/dL; aspartate aminotransferase (AST), 110 U/L; alanine aminotransferase (ALT), 34 U/L; alkaline

phosphatase (ALP), 343 U/L; total bilirubin, 12.2 mg/dL; prothrombin time (PT), 19.5 seconds; and international normalized ratio (INR), 1.7. Liver ultrasound revealed reversal of flow in the left portal vein, hepatomegaly, and ascites. The patient's model for end-stage liver disease-Na (MELD-Na) score was 30, Child-Pugh Class C with 12 points.

He underwent diagnostic paracentesis and was admitted for presumed spontaneous bacterial peritonitis in the setting of decompensated alcoholic cirrhosis. On hospital day (HD) 1, the patient reported increasing abdominal discomfort, which progressed on HD 2 to severe diffuse pain with episodes of rust-colored emesis. Therapeutic paracentesis was performed, and 4 L of ascitic fluid was removed, providing immediate relief of symptoms. The ascites showed $> 2,000$ WBCs, and cultures grew gram-positive cocci that eventually speciated to *Streptococcus viridans*.

The patient's pain later recurred, and a computed tomography (CT) scan of the abdomen and pelvis was performed with oral and intravenous (IV) contrast (Fig. 1). Imaging revealed anterior pneumoperitoneum, a moderate volume of abdominal and pelvic ascites with increased density in the pelvis, suggesting hemoperitoneum or extravasated contrast. There was lobulated gastric antral wall thickening with findings suggestive of a large ulcer and adjacent complex fluid collection containing air. The liver appeared grossly cirrhotic with recanalized umbilical vessels, and splenic and gastric varices were noted, consistent with portal hypertension. The volume of pneumoperitoneum was more than expected following paracentesis and in an unusual distribution. Thus, our working diagnosis based on these findings was that of a small perforated ulcer.

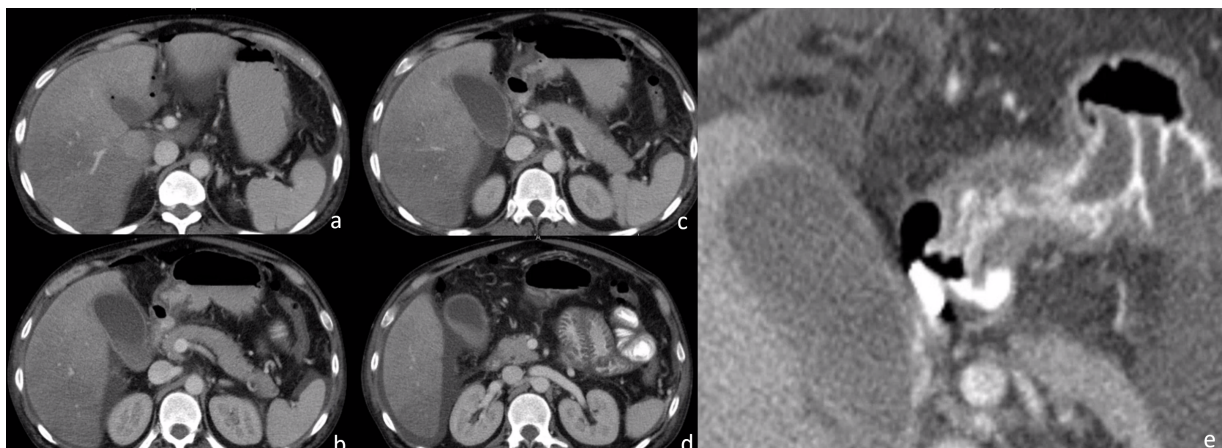


Figure 1. CT axial images showing a) free air locules in gallbladder fossa and anterior to 1st portion of duodenum, b) free air and site of duodenal perforation, c) extravasated oral contrast adjacent to site of perforation, d) free fluid along liver margin, and e) second CT scan with oral contrast showing contained perforation.

Further confirmation of the diagnosis was achieved with an upper gastrointestinal (GI) series with water soluble contrast, which demonstrated a contained perforated ulcer of the superior fornix of the duodenal bulb with a small adjacent collection measuring 1.6 x 1.8 cm. Serologic test for *Helicobacter pylori* IgG was negative. Given the high perioperative morbidity and mortality and his improving clinical status, our team elected to pursue nonoperative management. Of note, the patient was not a candidate for liver transplantation due to ongoing alcohol abuse and lack of social support.

He was made nil per os (NPO) with nasogastric (NG) tube suction and was administered broad-spectrum antibiotics (vancomycin, piperacillin/tazobactam, and fluconazole). The care plan also included twice daily IV esomeprazole, albumin boluses, octreotide drip, thiamine, multivitamins, folic acid, and daily vitamin K. During his hospital course, the patient's hemoglobin slowly decreased, followed by a drop from 7 to 4.2 g/dL on HD 5 associated with hypotension and hematochezia. An urgent esophagogastroduodenoscopy (EGD) was performed, which showed large non-bleeding varices in the lower esophagus. Two cratered duodenal ulcers were identified in the duodenal bulb, one with active bleeding (Forrest Class Ib). The lesions measured 10 mm x 5 mm and 8 mm x 5 mm (Fig. 2).

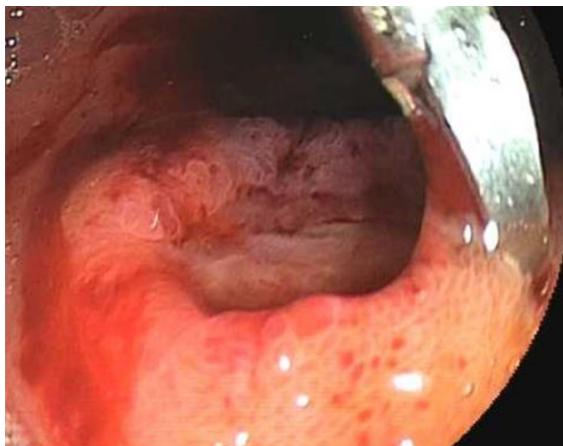


Figure 2. Upper endoscopy photo showing a large, cratered ulcer with clip applied to the exposed bleeding vessel.

The larger ulcer was successfully treated with epinephrine injection and clip application. The smaller had an adherent clot, which was washed, revealing a clean ulcer base. The patient's hypotension, tachycardia, and acute blood loss anemia resolved with transfusions and supportive care. On HD 6, the patient experienced worsening

abdominal pain and distention. A repeat CT scan of the abdomen showed increased free air and some extraluminal oral contrast. A percutaneous pigtail catheter was placed by interventional radiology adjacent to the site of perforation, which initially drained ascitic fluid and upper GI contents. Total parenteral nutrition was initiated. The patient was kept NPO with a NG tube to suction, and his pain gradually resolved. The insufflation during endoscopy may have initially worsened pneumoperitoneum but was essential to control active bleeding. We suspect that subsequent evacuation of ascites allowed the omentum to re-adhere and seal the perforation.

Over time, output from the pigtail catheter became serous. On HD 17, the patient underwent another upper GI contrast study, which showed no leak from his prior site of perforation. He was gradually advanced to a regular-consistency, salt- and fluid-restricted diet. He tolerated this well until discharge. Our patient's care was provided at a quaternary care facility with expert intensive care and the ability to perform timely bedside and interventional radiology procedures. The patient was closely monitored in the intensive care unit (ICU) by a multidisciplinary team that included hepatology and transplant surgery. Informed consent was obtained for the publication of this report.

3. Discussion

The management of perforated and bleeding peptic ulcers can be complex depending on additional patient factors. No clear consensus guidelines have been formulated for operative versus nonoperative management as cases are often multifactorial and rely heavily on clinical judgment. The traditional and most common approach for patients presenting with perforation is emergent operative repair; nevertheless, there have been attempts to assess the feasibility of nonoperative management (2). Reports suggest that roughly half of the cases of duodenal ulcers with perforation are self-sealed by the time the patient is admitted to the hospital, with the associated hypothesis that the seal may be broken by surgeons during abdominal entry and dissection (3, 4). Optimal medical management consists of NG tube suction, IV antibiotics, IV histamine-2 receptor (H2) blockers or proton pump inhibitors (PPIs), and serial abdominal examinations by trained staff (1, 3, 5). PPIs show more favorable results than H2 blockers in this setting, with one study reporting a 11% mortality rate and 16%

morbidity rate among the PPI group compared to a 64% mortality rate and 73% morbidity rate within the H2 blocker group (6). In a randomized control trial of 83 patients, Crofts et al. found no difference in the morbidity and mortality rates between operative and nonoperative management for perforated peptic ulcers. Results of a prospective study of 241 patients completed by Cao et al. also found no difference between the two groups. These two studies disagreed on mean length of hospital stay. Cao et al. reported no difference between 11.2 days in the nonoperative group and 10.9 days in the operative group ($p = 0.089$) while Crofts et al. reported a mean hospital stay of 7.8 days among the operative group and 12.0 days among the nonoperative group ($p < 0.001$) (1, 5). Despite this evidence showing equal risk of morbidity and mortality between the two groups, surgical repair of perforated ulcers is the more popular treatment among good surgical candidates, possibly due to the high success rates of current operative techniques and/or inherent distrust in self-sealing ulcers. Despite the concern for the durability of self-sealing ulcers, it was reported that among 152 documented cases of self-sealing ulcers confirmed by upper GI study, only two cases developed repeat leak (3).

With evidence demonstrating similar success between the two treatment modalities, it begs the question which patients can benefit from nonoperative treatment and which patients will ultimately require a delayed operation. Several factors that were suggestive of failure included an elevated heart rate greater than 94 bpm, volume of pneumoperitoneum score greater than 1, abdominal distention, pain on digital rectal exam, and age greater than 59 years (2). Another study reported that age >70 years, acute physiology and chronic health evaluation II (APACHE II) >8, fluid seen on ultrasound, and contrast extravasation found on water soluble gastroduodenal imaging were factors that selected against nonoperative management on univariable and multivariable analyses (5). Based on these factors, our patient would be considered higher risk for failure of nonoperative management. He had a volume of pneumoperitoneum greater than expected, abdominal distension, contrast extravasation on imaging, and APACHE II >8. Our institution's resources facilitate close continuous monitoring of the patient as well as engagement in frequent multidisciplinary evaluations and discussions. Therefore, this higher-than-average risk was acknowledged but accepted given our team's

confidence in our ability to identify and quickly manage any signs of decompensation.

Surgery among patients with hepatic cirrhosis poses serious risks to the patient due to several factors. Decreased production of coagulation factors, thrombocytopenia, and increased fibrinolysis contribute to coagulopathy that renders perioperative hemostasis difficult to achieve. The physiologic stress of surgery may also promote decompensation of liver failure, resulting in severe morbidity or death. Postoperatively, ascites and distention can prevent wound healing, resulting in breakdown of repairs and anastomoses. Hypoproteinemia and platelet dysfunction also hinder healing (7). 30-day postoperative mortality rates of 10% in patients with Child-Turcotte-Pugh-A (CTP-A), 30% with CTP-B, and 76-82% with CTP-C have been reported across patients with cirrhosis. These rates were also found to increase by three-fold for emergent surgery in patients with a similar CTP class (7-9). A retrospective case series of peptic ulcer surgery by Lehnert and Herfarth, which included 69 patients with liver cirrhosis, saw a mortality rate of 54% (37/69) with the highest mortality rates among patients with post-hepatic cirrhosis (9/15) and alcoholic cirrhosis (19/32). They found that hemoglobin less than 12 mg/dL, systolic blood pressure less than 100 mmHg, PT above 60% higher than normal, and portal hypertension were all statistically significant prognostic factors present in the patients with cirrhosis who underwent surgery for peptic ulcer disease (10).

Ultimately, our patient was at high risk of morbidity and mortality with either approach as the factors found to place patients at higher risk of failure of ulcer self-sealing also tend to convey poorer healing following surgical intervention. Due to the high mortality risk associated with this patient's degree of liver disease, therapeutic efforts were taken to promote self-sealing of the ulcer, including evacuation of the ascites via paracentesis, decompression of the GI tract via NG tube suction, endoscopic control of hemorrhage when the patient could no longer maintain his hemoglobin, and nutritional optimization.

4. Conclusion

Despite the high risk of failure, nonoperative management may provide the safest treatment approach for some patients with cirrhosis. The historically high morbidity and mortality of nonoperative management among patients with severe liver disease may be partially mitigated by

the availability of advanced critical care and less invasive interventional procedures now available. Further contemporary studies are warranted to examine the optimal management of perforated ulcers among this complex patient population. It also is critical to treat such patients in a certified tertiary or quaternary liver transplant center with robust multidisciplinary team involvement to promote the best possible patient outcomes.

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