

Case Report

Right hepatic artery injury: an unusual complication of penetrated duodenal ulcer disease

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ABSTRACT

Peptic ulcers generally appear in the stomach and the first segment of the duodenum as a result of mucosal erosion caused by pepsin and gastric acid secretion, with up to 70% of these occurring amongst patients aged 25-64. Currently, endoscopic procedures combined with proton pump inhibitors are considered the gold standard for managing complicated peptic ulcers, leaving surgical management as an option for endoscopic management failure or in scenarios such as incoercible bleeding, perforation, penetration and intestinal occlusion. Penetration of a gastric ulcer to adjacent organs is a rare complication; penetration to the liver and endoscopic diagnosis is even rarer. We have presented the case of a 54 year old diabetic male, who presented to the emergency room with upper gastrointestinal bleeding due to a chronic duodenal ulcer, with haemodynamic instability, requiring surgical management, revealing penetration to the liver with rupture of the right hepatic artery. The patient successfully recovered after surgery and was discharged 7 days after surgical intervention. We did not find any similar case reports in the current literature

Keywords: Duodenal ulcer, Gastrointestinal bleeding, Liver penetration, Penetrating ulcer, Peptic ulcer disease, Right hepatic artery

INTRODUCTION

Peptic ulcers usually occur in the stomach and the first portion of the duodenum, as a result of mucosal erosion caused by pepsin and acid secretion of the stomach.¹ 70% of these occur between 25 and 64 years of age.² Proton pump inhibitors radically altered the incidence of ulcers and their complications.³ The advancement of endoscopic techniques allowed these to be considered as the gold standard for the initial management of complicated ulcers. Surgery was relegated to the background, but it continues to play a fundamental role in the management of the most serious complications, which occur in up to 25% of cases, such as: incoercible bleeding, perforation,

penetration and obstruction of the outflow tract.⁴ Penetration of the duodenal ulcer to adjacent organs is a very rare complication; penetration to the liver and the endoscopic diagnosis of this entity is even rarer.⁵ We have presented the case of a chronic duodenal ulcer penetrated to the liver, which lacerated the right hepatic artery, causing massive bleeding. There are no similar reports in the world literature about this complication.

CASE REPORT

A 54-year-old male, diabetic, diagnosed with this condition 15 years before, in adequate glycemic control with metformin and glibenclamide, who one month prior

to admission underwent laparoscopic cholecystectomy due to lithiasis. During this hospitalization he presented an episode of upper gastrointestinal bleeding, attributed to a Forrest IIB gastric ulcer, which required endoscopic treatment and transfusion of 2 blood units.

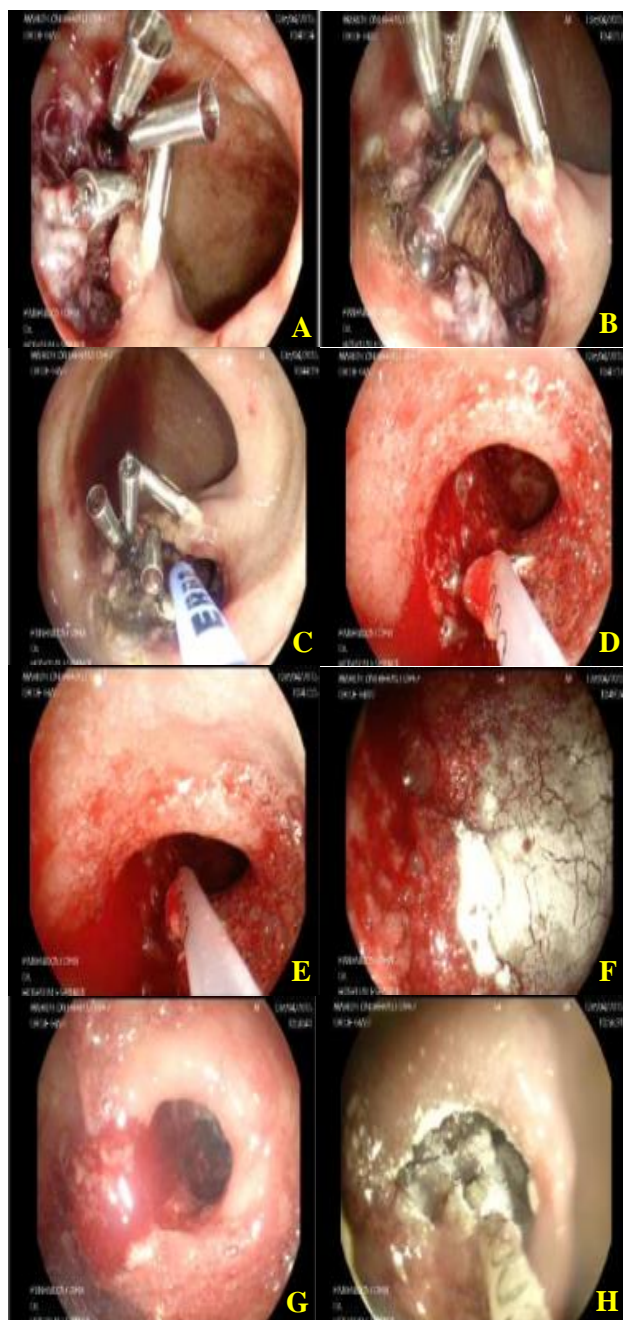


Figure 1: Esophagogastroduodenoscopy showing the presence of an ulcer of approximately 15mm with active arterial bleeding in the first portion of the duodenum.

On this occasion, he was admitted to the emergency room with mucosal pallor and diaphoresis. He was transported in an ambulance, and 30 minutes before he presented loss of consciousness, associated with 4 episodes of abundant hematemesis (610 cc) with sustained hypotension (60/40

mmHg). Grade IV hypovolemic shock secondary to upper gastrointestinal bleeding was diagnosed. Gastroenterology began emergency resuscitation with crystalloids, 2 blood units, sandostatin and omeprazole. The laboratories on admission showed hemoglobin of 10g/dL, blood glucose of 414g/dL and metabolic acidosis at the expense of hyperlactatemia on gasometric measurement. Urgent endoscopy consultation was requested. The endoscopic findings showed presence of a 15 mm Forrest IIa duodenal ulcer in the first portion, which underwent management with injection of 20 cc of adrenaline and application of hemoclips. The patient continued with hemodynamic instability in spite of the initial endoscopic treatment, with hemoglobin control reported at 5.9 g/dL, for which a second urgent endoscopy was performed, reporting a Forrest IA duodenal ulcer, with recurrent hemorrhage. The patient underwent hemostasis with argon plasma and hemospray. Due to inadequate control of bleeding and the possibility of perforation, surgical and/or endovascular management was suggested.

As a diagnostic complement, the general surgery department requested a CT scan of the abdomen and pelvis, where a 7 x 7 cm lobulated collection was documented with thick walls and suspicion of internal septa, heterogeneous content, with gas inside, adjacent to the duodenal knee, which appeared to be consistent with an abscess of the gallbladder bed.



Figure 2: Lobed collection on the vesicular bed. The presence of a hemoclip is observed in the upper sections of the liver.

He entered the operating room where he was initially submitted to a laparoscopic approach in an attempt to locate the collection or the ulcer, failing to do so, therefore the procedure was converted to open surgery using a Kocher incision. The duodenum was kocherized and a firm adherence of the first portion to the right lobe of the liver was observed. This adhesion was taken down to reveal a chronic ulcer, penetrated to liver. Primary closure of the duodenum with border remodeling was performed. In the hepatic cavity created by the ulcer, a

significant active arterial bleeding was observed, so a hepatorrhaphy was performed, with synthetic hemostat placed in the gallbladder bed.



Figure 3: 7 x 7 cm well limited lobed collection, with thickened walls presenting heterogeneous contents and gas within.

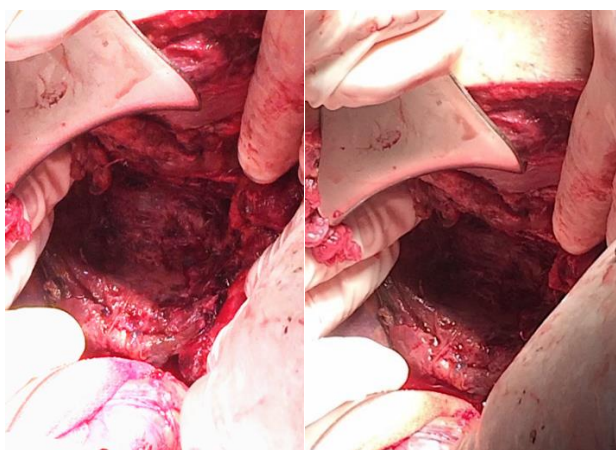


Figure 4: Hepatic cavity in the site of ulcer.

The patient was admitted to the intensive care unit, where he underwent a resuscitation due to massive bleeding, and several hours after the first surgery, he presented a new episode of hemodynamic deterioration and underwent laparotomy for the second time. In the second surgery only superficial bleeding in the layer of the hepatic bed and of the omentum was documented. An open drainage was placed and the duodenorrhaphy was reinforced. He was readmitted to the intensive care unit where once again he presented active bleeding through the drainage with hemodynamic deterioration, so he had to be reintervened a third time. The hepatorrhaphy was dismantled, a Pringle maneuver was performed, and the walls of the hepatic cavity created by the ulcer were incised with Harmonic ACE scalpel, revealing a large

cavity of 10 x 7 cm which involved practically the entirety of the right hepatic lobe; the source of was found to be a laceration of the right hepatic artery, which was then ligated in its origin.

The patient had a postoperative hemoglobin of 2.6g/dl, and in total was transfused 15 blood units, 18 fresh frozen plasmas and 8 cryoprecipitates. His postoperative evolution was satisfactory, without hepatic or intestinal complications, and he was discharged with complete tolerance to oral feeding.

DISCUSSION

Complications associated with acid-peptic disease, although less frequent since the introduction of proton pump inhibitors, are potentially fatal. Risk factors that contribute to increased mortality have been identified, such as: advanced age, concomitant conditions, shock before hospital admission, delayed treatment for more than 24 hours, ASA III-IV classification and localization of the ulcer.⁴

In Mexico, 70% of patients go to the hospital 24 hours after the onset of the disease, and 56.1% after 48 hours.⁴

It is estimated that bleeding mortality from a duodenal ulcer reaches between 7 and 10% of cases. Bleeding is the most common indication for surgery, but only 4% of bleeding ulcers should be surgically treated. The clearest indications for submitting the patient to a surgical procedure are: transfusion of 6 or more blood units, Failure to achieve endoscopic control in more than 2 occasions, failure of endovascular intervention or evidence of perforation.⁶

In order of frequency, the sites where perforation most frequently occur are: duodenum (60%), gastric antrum (20%) and gastric body (20%). Up to 1/3 of the cases of perforation are related to chronic consumption of NSAIDs.² The first case of a peptic ulcer penetrated to the liver was published in 1887. It is considered a rare complication of peptic acid disease, although its incidence is actually unknown. Between gastric ulcers and duodenal ulcers, the latter has been most frequently reported to penetrate the liver.⁸

Penetration to the liver can lead to 4 other complications: pain, formation of liver abscesses, obstruction of the outflow tract or bleeding of the upper gastrointestinal tract. The organ most frequently penetrated is the pancreas, usually by antral or duodenal ulcers. 80.5% of these patients will have recurrent pancreatitis. In order of frequency, the sites to which the penetration occurs are listed: Pancreas, minor omentum, bile ducts, liver, major omentum, mesocolon, colon and vascular structures.⁵

The vascular structure most frequently affected by the penetration of a peptic ulcer is the cystic artery, a situation that will lead to the presence of hematemesis.

However, in posterior duodenal ulcer penetration, the most affected artery is the gastroduodenal artery.⁸

In the world literature, there are only about 30 reported cases of peptic ulcers penetrated to the liver. All reported cases have manifested with gastrointestinal bleeding. Only 6 cases of duodenal ulcers penetrated to the liver have been reported, which were diagnosed by endoscopy, after histopathologic report of liver tissue in duodenal biopsies.⁵ Most cases are diagnosed by tomography or ultrasound. The usual tomographic finding is a cavity adjacent to the liver and duodenum with gas present inside.

The penetration to the liver of a peptic ulcer, in most cases requires surgical management, but there are cases reported in the literature, handled conservatively with PPIs.⁹

We present an exceptionally rare case of a duodenal ulcer penetrated to the liver, a penetration that could be documented through endoscopic images, without a positive biopsy for hepatic tissue due to the urgency of endoscopic management, which also resulted in a vascular laceration of the right hepatic artery evidenced during open surgery, a situation that has not yet been reported in the world literature.

CONCLUSION

Although the complications derived from acid-peptic disease are becoming less frequent, the surgeon should be familiar with them, because there are clear indications for surgical management. In the present case it is possible to discuss the possibility of having offered the patient an endovascular management of the lesion, despite its deteriorated hemodynamic status, and then consider hepatic and duodenal repair. Our patient undoubtedly had several factors identified as key in the worsening of the prognosis of a duodenal ulcer (age, diabetes, state of shock at admission, location), and in spite of it, its evolution was very favorable. The lack of association with the consumption of NSAIDs in our patient is striking. Undoubtedly, it is a case that helps us to reflect and rethink the diagnostic and therapeutic possibilities in the context of a massive bleeding of the upper digestive tract, associated with a duodenal ulcer.

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