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Case report

Recurrent Peptic Ulcer Perforation: A Case Report

Maather Al Abri, MD, Ghaitha Al Mahruqi [*], MD, Hani Al Qadhi, FRCSC

Department of Surgery, Sultan Qaboos University Hospital, 123, Oman

*Corresponding author: Ghaitha Al Mahruqi; *almahruqighaitha@gmail.com*

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Abstract

Background: Refractory peptic ulcers are ulcers in the stomach or duodenum that do not heal after eight to twelve weeks of medical/surgical treatment or those that are associated with complications despite medical tssreatment. We herein present a case of a 44 year old man with a recurrent perforated duodenal ulcer requiring emergent surgical intervention.

Keywords: ***Peptic ulcer, duodenum, perforation***

Introduction

Peptic ulcer disease (PUD) is a major public health problem and it is caused by acid injury which results in a fibrin-covered break in the mucosa of the digestive tract [2]. PUD can be complicated by haemorrhage, perforation, penetration, or obstruction. These complications represent the most common indications for surgery in PUD. The estimated worldwide incidence of PUD is 0.1-0.3% with a prevalence of 5-10% , duodenal ulcers is complicated by perforation in 5-15% of patients and the long term recurrence rate of perforation after surgical management was found to be 12.1% [1-3]. While the use of Non-steroidal anti-inflammatory drugs (NSAIDs) and H. pylori are frequent causes of peptic ulcer perforation (PUP), demographic differences in age, gender, perforation location and etiology might potentially influence the rate of PUP recurrence and post-operative mortality [4].

Case presentation

44-year-old male patient, smoker (15 pack - year) and heavy alcohol consumer with past surgical history of duodenal ulcer perforation that was repaired laparoscopically using falciform ligament a year ago, presented to the emergency department with a chief complaint of asthenia and an acute onset of burning sensation in the epigastric area radiating to the back and is associated with nausea and vomiting. On examination, the patient was in distress, tachycardic; 120 beats per minute, tachypnoeic; 26 beats per minute, blood pressure of 110/90 mmHg, maintaining SpO_2 of 98% on 2L oxygen. His abdomen was rigid and tender over the epigastric area. Preliminary laboratory results were notable for leukocytosis; WBC of $13X10^9$ with neutrophil predominance. Arterial blood gas revealed an element of metabolic acidosis; pH of 7.1, HCO_3- of 19 mmol/L, PCO_2 of 43 mmHg, base excess of -3 mmol/L and lactate of 5 mmol/L. Erect chest x-ray revealed air under the left diaphragm (**Figure 1**). The patient was resuscitated; he received 2L of normal saline, intravenous (IV) metronidazole, cefuroxime and omeprazole were administered. CT abdomen with IV and oral contrast demonstrated pneumoperitoneum, free fluid all over the abdominal cavity and fat stranding around the duodenum. However, there was no contrast leak. The patient was taken for an exploratory laparotomy; the abdominal cavity was entered through a midline incision revealing voluminous amount of purulent fluid and undigested food particulates in all four quadrants. The fluid was sampled for cultures and removed via suction. Once unobscured, dense adhesions between the stomach, liver, and Omentum as well as the anterior surface of the duodenum were identified. Adhesiolysis and takedown of the falciform ligament exposed a duodenal ulcer immediately distal to the pylorus in the first part of the duodenum (**Figure 3**). Prior sutures on the anterior aspect of the duodenal lumen were also noted from the previous surgery that was performed for duodenal ulcer perforation. An extensive four-quadrant washout using warm saline was performed until irrigation was noted to be clear. Ulcer evaluation revealed a 2 cm defect with raised, thickened and hyper-emic edges. Graham patch omentopexy was performed, full-thickness bites from serosa to mucosa were taken around the entire perimeter of the exposed duodenal ulcer with initial placement in the duodenum, an Omentum pedicle was parachuted into place as a Graham patch. Once the anchoring sutures were completed and tied, the fascia was closed in a simple running fashion using looped PDS and the skin was approximated using a skin stapler with overlying sterile dressing. The patient was shifted to high dependency unit; he maintained normal vitals, nasogastric tube was placed by the anaesthesiologist during the operation and was set to low intermittent suction and an intravenous Proton pump inhibitor (PPI) was initiated. We opted to investigate the patient for Zollinger Ellison syndrome. However, the patient left against medical advice the next day, he was discharged on H. pylori eradication therapy and was given a day-care appointment with the gastroenterologist for an upper GI endoscopy.

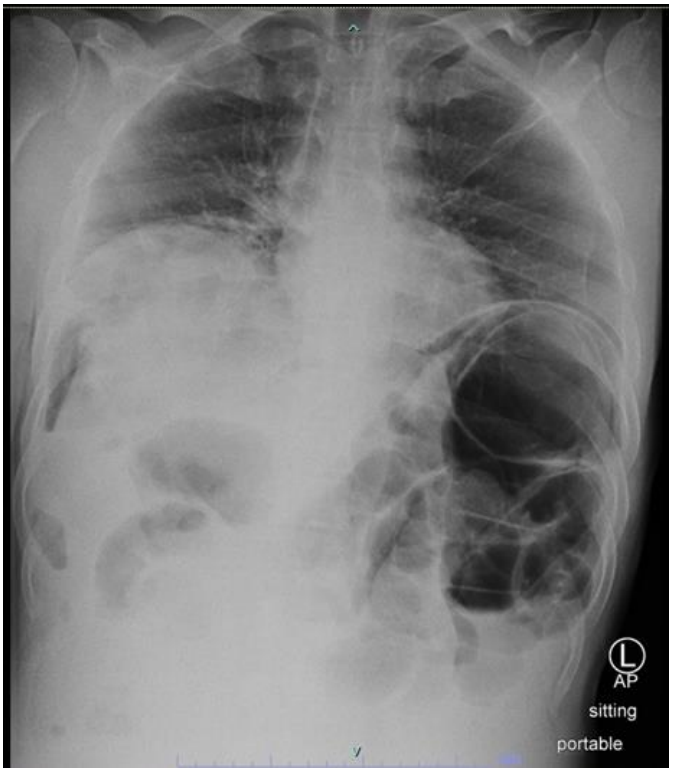

Figure 1: Erect chest x-ray demonstrates pneumoperitoneum with free air under the left hemidiaphragm.

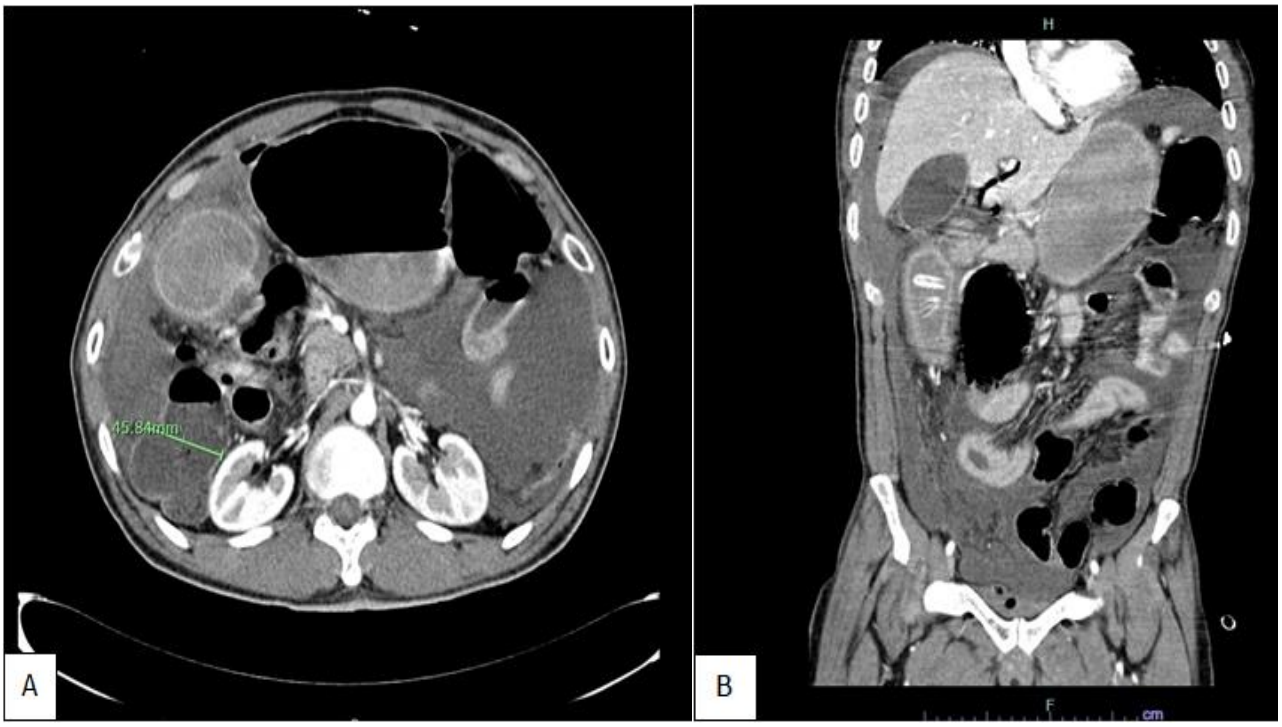

Figure 2: CT abdomen, (A-axial section, B - coronal section) with IV and oral contrast, demonstrates significant free fluid and pneumo-peritoneum in the upper abdomen and porta hepatis suggestive of viscus perforation, there is no evidence of focal bowel wall defect or oral contrast leak.

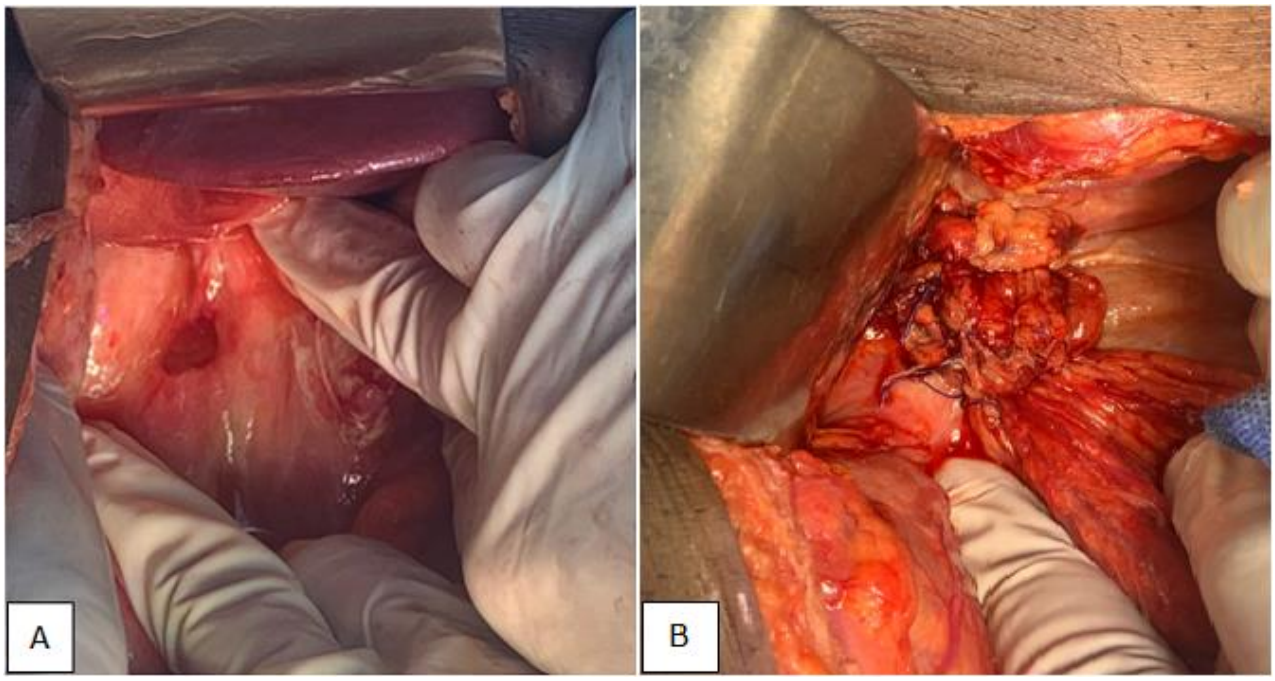

Figure 3: A-2 cm defect with raised, thickened and hyper-emic edges in the first part of the duodenum; B-Omental pedicle parachuted into the defect as a graham patch

Discussion

The incidence of peptic ulcer has decreased in the recent years because of the availability of H. pylori eradication therapy. However, the use of non-steroidal anti-inflammatory drugs, smoking and alcohol consumption has become more widespread and, therefore, there may have been no corresponding decrease in peptic ulcer complications [1]. Re-perforation is considered a predictive factor for mortality from peptic ulcer disease and therefore an average recurrence rate of 12.1 % should not be underestimated.

It was found that the use of immunosuppressant agents and corticosteroids, presence of malignancy (adenocarcinoma and lymphoma), preoperative shock and admission to intensive care unit at the time of presentation were associated with increase rates of perforation recurrence [5]. In our case, none of these factors were present but reasonable factors which might be responsible for recurrent perforation were that the patient often consumed both excessive alcohol and tobacco in addition to irregular use of proton pump inhibitors. Smoking should always be addressed in patients with refractory peptic ulcers as it is responsible for impaired tissue healing throughout the body and it can impair the healing of peptic ulcers through decreased prostaglandin synthesis [2]. A population-based cohort study reported that the risk of ulcer re-perforation is threefold higher in smokers compared to non-smokers [6]. Systemic conditions such as Crohn's disease, sarcoidosis, Behcet's disease and polyarteritis nodosa can increase the risk of peptic ulcer re-perforation [7]. Zollinger Ellison syndrome is associated with increased acid production and usually presents as a refractory duodenal ulcer that doesn't heal with standard doses of PPI [2]. The recurrent ulcer tends to recur at the original site, as in our case. However, H. pylori eradication therapy may prevent recurrence after surgical management [8].

Different surgical techniques have been proposed for the management of perforated duodenal ulcer (PDU) but the main surgical treatment is the closure of the perforation using an omental patch or falciform ligament.

A study that compared the results of patients who underwent falciform ligament repair and omentopexy for PDU concluded that there is no significant difference in the general post-operative morbidity and mortality between the two groups [9]. In Graham patch omentopexy several interrupted sutures are taken through the defect and a well vascularized omental flap is brought between these sutures. In the setting of previous several operations in which there is unhealthy or deficient omentum, the falciform ligament could be used in the repair of perforated duodenal ulcer [10].

With the advent of proton pump inhibitors (PPI), truncal vagotomy for acid reduction has become obsolete. However, a population-based cohort study concluded that a simple closure procedure has a higher rate of repeated ulcer associated surgery in non-H. pylori infected peptic ulcer perforation patients than in truncal vagotomy patients. Therefore, postoperative PPI might not be sufficient to prevent the recurrence of duodenal ulcer perforation in non-H. pylori infected patients. (8)

Conclusion

Peptic ulcer disease remains a substantial health care problem globally. Swift diagnosis and treatment of PUP along with adequate therapy and duration of PPI is likely to decrease postoperative morbidity and mortality. Moreover, vigilant attention should be paid to the treatment of ulcer etiology.

List of abbreviations

PUD: Peptic ulcer disease
NSAIDs: Non-steroidal anti-inflammatory drugs
IV: Intravenous
PPI: Proton pump inhibitors

Declarations

Ethics approval and consent to participate

Not applicable

Data Availability

Not applicable

Conflicts of Interest

No conflicts of interest

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Authors' contributions

MA did the literature review about recurrent peptic ulcer perforation and went through case details.
GA analyzed and interpreted the patient's data and went through the surgery details and follow up of the patient.
HA revised the article critically for important intellectual content and finally approved the version to be published.

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