Case Report

Emergency Operations for Bleeding Duodenal Ulcer:A simple option to consider: Case Report

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Abstract

We report a 46 years-old man who had severe bleeding from a posterior duodenal ulcer (DU) that was diagnosed but could not be treated endoscopically in another health facility. He went into shock as he was being admitted to the casualty at Khartoum North Teaching Hospital (KNTH). His haemoglobin (Hb) dropped to five gram/dl. He required resuscitation and transfusion of six units of blood overnight. Emergency surgery was performed.

Over sewing (OS) of the ulcer was done and the stenosed first part of duodenum was closed transversely (pyloroplasty). No acid-reducing procedure (ARP) was done. The patient received anti-helicobacter therapy via the intravenous route preoperatively and continued postoperatively. This was later given orally after he started taking by mouth. He made an uneventful recovery with no recurrence of bleeding and was discharged home one week latter. Endoscopy was done at KNTH six weeks later. This showed complete healing of the ulcer with no evidence of Helicobacter pylori in the biopsies taken.

We found simple OS of the bleeding DU together with anti-helicobacter therapy safe, efficient, and not associated with re-bleeding. We discuss the rationale of this simple treatment. We propose the need for a randomized controlled study comparing it with acid-reducing procedure (ARP) as options in the surgical treatment of bleeding DU.

Key words

Bleeding duodenal ulcer, Emergency surgical operations, Peptic ulcer, Helicobacter pylori, Anti-helicobacter therapy,

Case Report

A 46-year old male presented to the University unit in the casualty at Khartoum North Teaching Hospital (KNTH) with haematemesis and melaena for two days. This was preceded two days before with dizziness and general fatigue. The patient was a heavy smoker (30 cigarettes a day) for many years. He admitted to taking Aspirin for headache.

Upper Gastrointestinal (GI) endoscopy was performed at another health facility. This showed an ulcer in the posterior wall of the first part of the duodenum, covered with a blood clot.

It was also reported that the ulcer was not injected, and surgical treatment was recommended if bleeding recurred. Afterwards, the patient unfortunately had a recurrent bleed and slipped into a hypovolaemic shock when he presented to our casualty. He was admitted to the ICU for resuscitation. This consisted of intravenous (IV) fluid and blood transfusion. He was also given Cimetidine 200 mg i.v. 8 hourly and antibiotics (cephalosporin, metronidazole, and amoxicillin).

His initial investigations showed haemoglobin (Hb) of Five (5) gm/dl. The patient received six units of blood during the night and the following morning, after which his Hb increased to 11gm/dl. He was taken to theatre where laparotomy was done. Dense adhesions were found in the supracolic compartment, between the greater omentum, duodenum, gallbladder, and liver. The

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adhesions were released. The first part of the duodenum was stenosed and indurated. Kocherisation of the duodenum was done, and a longitudinal incision was made along the pylorus and the first part of the duodenum. The posterior ulcer was identified. There was no active bleeding, but a vessel was seen in the floor of the ulcer. This was over sewn with No. 2/0 Vicryl sutures. The pyloric incision was sutured in 2 layers transversely as a pyloroplasty using 2/0 vicryl as well. Vagotomy was not done.

The patient had a smooth uneventful postoperative recovery and was discharged home after one week. He received anti-helicobacter quadruple therapy orally consisting of Amoxicillin 500 mg 8-hourly, Metronidazole 500 mg 8-hourly, and Clarithromycin 500 mg twice a day, for 14 days. In addition, he had Omeprazole 20 mg bd for 4 weeks.

The patient underwent upper GI endoscopy 6 weeks after his discharge. This revealed complete healing of the ulcer, and prepyloric biopsies did not show evidence of Helicobacter. He remains asymptomatic 18 months after discharge.

Discussion

Bleeding and perforated DU have the same aetiology and pathogenesis. They differ only in the duodenal site of the pathology. The former erodes the duodenal wall posteriorly into the gastro-duodenal artery, and the latter erodes anteriorly into the peritoneal cavity. Both ulcers may occur simultaneously or at different times in the same patient. The finding of dense adhesions anterior to the duodenum in this patient may indicate the occurrence of a silent perforation some time earlier.

The other difference between bleeding and perforated DU is in their current surgical treatment strategies. Whereas the management options of perforated DU have been reviewed from time to time particularly in the post-Helicobacter era, those of bleeding peptic ulcer remained largely the same. At one point of time, the standard treatment of perforated DU consisted of over- sewing the perforation together with an acid-reducing procedure (ARP), such as truncal vagotomy (TV). This necessitated addition of a drainage procedure, such as pyloroplasty or gastro-enterostomy because TV would not only inhibit gastric acid secretion but also the motility of the gut. As time went by, less radical ARPs were developed such as selective and highly selective vagotomy whereby the vagal motor branches (Nerves of Latarjet) to the pyloric area were preserved. Eventually, with the discovery of Helicobacter pylori as a major cause of DU and the development of efficient anti-helicobacter therapy, ARPs are rarely performed nowadays for perforated DU. Moreover, non-operative management is now an established option as part of the management of perforated DU in selected cases.

In contrast to perforated DU, the management of bleeding complications has not changed much in the post-Helicobacter era, and is still widely variable and radical. This may be due to the fact that bleeding DU has a more dramatic presentation and carries a more serious risk of death from exsanguinations and hence surgeons are still carrying out radical surgery.

The indications of surgery in bleeding DU that has been mostly agreed upon are, age of 60 years or above, recurrent bleeding during the same hospital admission and the need to transfuse more than half the volume of the patient blood (5-6 units) to maintain a haemodynamic stable state, and Hb of 10 gm/dl at least.

The best surgical procedure for treating bleeding duodenal ulcer is unknown. Reuben from the USA reported wide variations in the management of bleeding DU that depended on the surgeon's experience and individual patient's factors[1]. The surgical options include: (i) over sewing (OS) of the bleeding ulcer plus an acid-reducing procedure (ARP), such as TV and pyloroplasty (ii) gastric resection (GR) with ulcer excision. Gastric resection (GR) includes operations such as Billroth II with gastro-jejunal anastomosis, or antrectomy and vagotomy. All these procedures are characterized by a long

operative/ anaesthetic time, and each carries significant risks and life-long side effects.

Comparative studies showed that the recurrence rate for bleeding is higher in OS + truncal vagotomy (17%), than GR (3%), whereas the anastomotic leak rate is lower in OS +TV (3%) than GR (13%)[2]. However, some studies had either small number of patients or were stopped because of slow recruitment.

The surgical options in bleeding DU depend on the patient condition, the expertise of the surgeon[1], and the size of the ulcer[3]. Some authors suggest that GR would be the operation of choice for (giant) ulcers of two cm or more in internal diameter[3].

We believe that the discovery of Helicobacter pylori and the development of efficient anti-helicobacter therapy should also change the treatment options of bleeding DU as it did for perforated DU.

Gisbert et al have shown that the incidence of H. pylori infection in DU bleeding is almost 100% and that after H. pylori eradication there was no recurrence of bleeding after one year[4]. The H. pylori re-infection rate was <3% after one year. It was also shown that maintenance therapy with H2 blockers or proton pump inhibitors was not necessary after eradication of H pylori. Another study has shown that Helicobacter eradication therapy has reduced ulcer relapse rate in patients who had perforated DU treated by simple OS to < 5% after one year[5].

Our patient received intravenous anti-helicobacter therapy on admission. Emergency surgery was indicated to stop the bleeding as he presented in shock and we didn't have emergency endoscopic services at the time. The patient recovery was smooth. Oral anti-helicobacter therapy was continued when the patient started to take oral fluids on the 3rd postoperative day. The patient was given some health education and advised to stop smoking. He was followed up in our outpatient clinic. Six weeks after discharge from the hospital he underwent a check upper GI endoscopy at KNTH. This showed complete healing of the ulcer. Pre-pyloric biopsies showed no evidence of Helicobacter pylori. The patient continued to be well with no recurrence of bleeding 18 months after the operation.

We agree with Milat, et al[6] that randomized controlled studies are needed to compare ARPs with minimal surgery (stopping bleeding) combined with antisecretory drugs and eradication of Helicobacter pylori. These studies may be difficult to carry out due to the small numbers of such patients.

Conclusion

This case showed that, simple over sewing of bleeding duodenal ulcer combined with anti-helicobacter therapy is safe, and efficient. It was not associated with re-bleeding with long-term follow up. This case also highlighted the need to include this simple procedure in randomised controlled studies to compare it with the radical acid-reducing procedures that were developed prior to the Helicobacter era.

Note:

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