
Proximal small bowel obstruction caused by a massive intraluminal thrombus from a stress ulcer

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Location: Huddersfield Royal Infirmary, Huddersfield, UK

Citation: Siddiky AH, Gupta P. Proximal small bowel obstruction caused by a massive intraluminal thrombus from a stress ulcer. JSCR 2012 1:6

ABSTRACT

We describe a case of proximal small bowel obstruction caused by an occlusive thrombus as a result of bleeding from a duodenal ulcer, which is likely to be stress induced. Initial presentation was confused as a bleeding duodenal ulcer and resultant ileus. Such reports are incredibly rare in the literature and never has one been reported as a result of a stress ulcer.

Obstructive symptoms in the acute postoperative patient may be confused for an ileus but mechanical causes must be excluded. The presence of upper gastrointestinal bleeding must not detract from that possibility. Future identification of an occlusive clot on endoscopy that is impossible to circumnavigate may benefit from intraluminal injection of thrombolytic agents to prevent obstruction but this must be weighed with the risk of exacerbating any bleeding. Clearly a risk versus benefit analysis will be necessary on an individual basis.

INTRODUCTION

Small bowel obstruction caused by a thrombus has been described in cases following laparoscopic Roux-en-Y gastric bypass surgery as a result of suture line bleeding (1-3) and in coagulopathic patients following emergency surgery (4) or from a bleeding Meckels diverticulum (5). The paucity of cases in the literature suggest that this is a rare occurrence and to our knowledge, has never been described following a spontaneous haemorrhage from a duodenal ulcer. There is currently no specific guidance on the management of such occurrences.

CASE REPORT

A 68 year Caucasian male was admitted electively for an open anterior resection of rectum. Preoperative investigations for anaemia had identified no oesophageal, gastric or duodenal pathology but did identify a mid-rectal lesion. Biopsy confirmed an adenocarcinoma and

radiological staging put this as pT₃ N₁ M₀.

The operation proceeded without difficulty with the formation of a colorectal anastomosis. It was decided that a defunctioning loop ileostomy was not necessary. The patient was managed postoperatively following the Enhanced Recovery Programme for colorectal surgery. Haematological investigations demonstrated a rise in his inflammatory markers in the early days postoperatively and this was assumed to be as a consequence of the surgery. No bowel activity was noted but as the patient was not complaining of any nausea or vomiting, oral nutrition continued. Seven days postoperatively, the patient developed pyrexia of 38.3 °C and a significant rise in his C-reactive protein (CRP) and white cell count, although there was no evidence of any abdominal peritonism. Computed tomography (CT) identified an anastomotic leak with free fluid in the pelvis and the patient underwent an emergency Hartmann's procedure and was transferred to the intensive care unit (ICU). He was kept intubated for 48 hours before returning to full consciousness. Conservative management of oral intake ensured a nil per oral regime followed by gradual build-up of fluids to solids. The patient appeared to be making progress with a decline in his inflammatory markers and an increase in his stoma activity. Eight days following the emergency Hartmann's procedure, the patient developed a tachycardia and a drop in his haemoglobin of 4 g/dL. Blood was noted in his nasogastric aspirate. He underwent an emergency upper gastrointestinal (UGI) endoscopy which identified evidence of blood in the stomach and a large thrombus at D₁. There was no active bleeding seen. Attempts to circumvent the thrombus were unsuccessful and a decision was made not to disturb it as the patient had stabilised. An intravenous PPI infusion was commenced and the patient returned to the ICU. Three days later, there was a further acute deterioration with a sudden reduction in stoma activity, development of colicky abdominal pain and another drop in the haemoglobin. It was presumed the patient was experiencing an acute bleed and was transferred to the operating theatre under a general anaesthetic for another UGI endoscopy. The second endoscopy identified similar findings as the first, with no active gastric bleeding and a large clot in D₁, which was impossible to navigate round. The decision was made for a re-laparotomy on the assumption the patient was bleeding distal to the thrombus. Intraoperative assessment found distended duodenum and proximal jejunum, collapsed ileum and colon and no free fluid or blood. An enterotomy in the jejunum removed a significant solid thrombus which had completely occluded the duodenum and formed an exact replica of the duodenal bulb. No bleeding was identified. The ulcer was oversewn with an omentum patch, the enterotomy closed and abdomen washed with saline before completing the operation.

The patient continued to do well from this point and was transferred to the ward after a further three days on ICU and discharged home seven days later.

DISCUSSION

The development of duodenal ulceration as a consequence of the physiological response to stress such as surgery is well known. The incidence of significant haemorrhage, however, is

relatively rare but for that to develop into a thrombus that then causes obstruction is almost unknown. Although not apparent in this particular case, the development of obstructive symptoms in the acute postoperative state may be confused for ileus and vigilance is necessary to identify alternative causes. It would be difficult to suggest recommendations to avoid this specific occurrence as much debate exists on the role of PPI prophylaxis in preventing stress ulceration in postoperative patients, with the reported risks of associated hypernatraemia and *C. difficile* infection opposed to routine use. Future identification of an occlusive clot that is impossible to circumnavigate with the endoscope may benefit with intraluminal injection of thrombolytic agents to prevent obstruction but this must be weighed against the risk of exacerbating any further bleeding. Clearly a risk versus benefit assessment would be required on a patient by patient basis.

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