Abdominal aortic occlusion and vascular compromise secondary to acute gastric dilatation in a patient with bulimia

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ABSTRACT

Acute gastric dilation is a rare but recognised complication in patients with bulimia and anorexia following binge episodes owing to decreased bowel motility. We present a rare case of acute gastric dilation secondary to bulimia in an otherwise healthy 18-year-old female patient that resulted in compression and complete occlusion of the abdominal aorta, leading to acute mesenteric and bilateral lower limb ischaemia. This resolved immediately following a laparotomy and gastric decompression.

Management of these patients is very challenging owing to the lack of a successful precedent. To our knowledge, such a catastrophic complication has only ever been reported once in the literature and the outcome was fatal. Our case is of additional importance as it offers a successful management strategy for these patients.

KEYWORDS

Acute gastric dilation – Aortic occlusion – Bulimia

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Case History

An 18-year-old woman with bulimia presented to the emergency department with sudden onset of severe abdominal pain and distension over the previous 2 hours. Within 30 minutes of arrival, her pain started to radiate to her back, she became unresponsive and developed extensive mottling of the skin from the waist down. Further examination revealed a grossly distended abdomen, engorged neck veins and absent femoral pulses bilaterally. She had a blood pressure of 190/132mmHg, a pulse rate of 100/min, a respiratory rate of 28/min, oxygen saturation of 98% on air and a temperature of 37.2°C.

Assuming a diagnosis of aortic dissection, the on-call vascular surgeon was bleeped to the emergency department while the patient was being intubated and resuscitated. A venous blood gas sample showed she was in profound metabolic acidosis with a pH of 7.1 and a lactate of 7.6.

As the patient was haemodynamically stable, urgent computed tomography was performed. This revealed a massively dilated stomach occupying the majority of the abdominal cavity from the epigastrium down into the pelvis with gas extending into the liver and spleen (Fig 1). Alarming, it also showed that the abdominal aorta was being compressed and completely occluded above the superior mesenteric artery origin (Figs 2 and 3).

The patient was taken to theatre for an emergency laparotomy and on opening the abdominal cavity, she was also found

to have extensive small and large bowel ischaemia. A gastrotomy was performed and the stomach was decompressed, with a total of 15l of gastric content being emptied. This resulted in an immediate restoration of circulation with reperfusion of the bowel and return of femoral pulses bilaterally. She was transferred to the intensive care unit (ICU) with a laparostomy and a plan for a 'relook' laparotomy in 48 hours.

Unfortunately, over the next 24 hours, the patient developed disseminated intravascular coagulopathy and liver failure, and went into a profound metabolic acidosis. She was returned to theatre and an extended right hemicolectomy with an end ileostomy was performed for necrosis of the ascending and transverse colon. The stomach was also noted to be slightly dusky so a plan was made to review this again in six hours, at which point a total gastrectomy with cross-stapling of the distal oesophagus was performed.

Following this, the patient remained on the ICU for three weeks. She had five further relook laparotomies, a tube oesophagostomy and tube jejunostomy were formed, and, finally, a polyglactin mesh was used to close the abdomen.

The patient was intubated and monitored closely on the ICU for the duration of her stay. She received total parenteral nutrition, her coagulopathy was treated and she was started on continuous venovenous haemofiltration.

Twenty-three days following the patient's initial procedure, all of her biochemical parameters had returned to within normal range. She was haemodynamically stable and a referral was made to the intestinal failure unit for further care.

Figure 1 Coronal section of abdominal computed tomography showing: acute gastric dilation with extension of the stomach from the diaphragm to the pelvic floor (A); extensive presence of gas in the portal, splenic and mesenteric veins secondary to ischaemia (B); and absence of any contrast in the femoral arteries due to occlusion of the proximal abdominal aorta (C)

Discussion

Bulimia nervosa and anorexia nervosa (binge/purge subtype) are disorders characterised by a refusal to maintain adequate body weight with intermittent binge/purge episodes. These affect 0.3–1% in the UK.¹

Gastric dilation is a rare but recognised complication in these patients owing to motility dysfunction and delayed gastric emptying following binge episodes. The pathophysiology of this process is poorly understood but is thought to be due to gastrointestinal smooth muscle atrophy and autonomic nervous system disturbances resulting in gastric rhythm abnormalities.²

Acute gastric dilation (AGD) is a life threatening surgical emergency owing to the risk of gastric perforation, necrosis, shock and aspiration.^{3,4} It is usually managed with nasogastric tube decompression of the stomach, rehydration and correction of electrolyte disturbances.⁴ Occasionally, nasogastric decompression is not possible because of the presence of large retained food items, in which case a gastrotomy and surgical decompression may be performed.⁵ However, if vascular compromise is suspected, then non-operative treatment is not an option and an urgent laparotomy is necessary as it allows for rapid surgical decompression and evaluation of any ischaemic bowel segments.

Figure 2 Sagittal sections of contrast abdominal computed tomography at different levels showing: gross dilation of the stomach with displacement of bowel loops (A); contrast in the abdominal aorta proximally in the top two images, which then disappears at the level of L1 owing to compression and occlusion by the dilated stomach (B); extensive presence of gas in the portal, splenic and mesenteric veins secondary to ischaemia (C); and absence of contrast in the femoral arteries (D)

Aortic compression and occlusion secondary to AGD was reported in a similar case in Hungary in 2006 involving a 22-year-old female anorexic patient who developed AGD following a binge episode, which she initially denied.⁶ In that case, there was also rapid return of circulation following gastric decompression, the bowel appeared viable and the abdomen was closed. Unfortunately, the patient died 36 hours later, having developed severe metabolic acidosis and disseminated intravascular coagulation secondary to reperfusion injury.

AGD should be considered very early on in any young thin/malnourished female patient presenting with acute abdominal pain and distension, and the assessing clinician should enquire directly about any history of eating disorders or binge episodes even though these may be denied by the patient. If a diagnosis of AGD is being considered, every attempt should be made to rule out vascular insufficiency and, if suspected, a formal laparotomy should be considered as a matter of urgency. Rapid assessment with a high index of suspicion is key in diagnosing and managing these patients as delays in treatment have been associated with increased mortality.⁵

Any ischaemic bowel segments should be resected and any attempt at anastomosis should be deferred until the patient has recovered from the reperfusion injury as the additional risk of sepsis following an anastomotic leak would probably prove fatal.

A surgical laparostomy is preferred as there is a high risk of abdominal compartment syndrome following reperfusion

Figure 3 Coronal section of abdominal computed tomography showing absence of contrast in the abdominal aorta below the level of L1 due to compression and occlusion by the massively distended stomach (A), and abdominal aortic compression and occlusion at the level of L1 (B)

and these patients will likely need to have several relook procedures by an experienced senior bowel surgeon to reassess the bowel for ischaemia. Close monitoring on the ICU with organ support and early parenteral nutrition is a vital part of the management of these patients as it is to be expected that they develop acid base disturbances, disseminated intravascular coagulation and multiple organ failure.

Conclusions

AGD is a life threatening emergency that affects young patients, and carries significant morbidity and mortality. Having a high index of suspicion allowing early diagnosis and management of these patients is key in preventing an adverse outcome. Early surgical intervention coupled with close monitoring on the ICU and organ support is crucial.

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