Case report

Acute gastric dilatation with infarction and perforation

Report of fatal outcome in patient with anorexia nervosa

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SUMMARY This is a report of a 22-year-old woman with treated anorexia nervosa who died of complications of acute gastric dilatation—that is, infarction and perforation with severe and irreversible shock. Binge eating and drinking, precipitated by emotional crises, contributed to her acute gastric dilatation. This complication of anorexia nervosa has been previously reported, but, unlike the others, this case ended fatally. The literature is reviewed.

Acute gastric dilatation is increasingly uncommon in clinical medicine¹ and is of varied aetiologies, one of these being anorexia nervosa.²⁻⁵ Gastric infarction as a complication of acute gastric dilatation is an unusual associated circumstance.³ We present a patient with such a course of events—that is, a history of anorexia nervosa in remission, with associated episodic irregular eating habits, acute gastric dilatation and infarction, leading to perforation and death. Awareness of this complication of anorexia nervosa may lead to its earlier recognition and more timely and favourable treatment.

Case history

The patient was a 22-year-old white female university student, who presented to the emergency room with the chief complaint of abdominal pain, constipation, and an inability to vomit. Here it was ascertained that she had been seen twice over a four month period for constipation and abdominal pain. She was treated with stool softeners and enemas. Information pertaining to recent oral food intake was not obtained. There was no

history of oral contraceptive use and the patient had taken no other medications nor did she admit to ingesting any toxic substances. Her last menstrual period was four months previously. Past medical history was significant for a cholecystectomy and appendectomy in 1967 for vague abdominal pain, with no reported pathological changes in either specimen.

On physical examination the patient was writhing in pain. Bowel sounds were present. Her abdomen was tympanitic, without rebound tenderness. She was given Ducolax tablets and was sent home.

She returned to the emergency room nine hours later with increasing abdominal pain, nausea, and an inability to vomit. Physical examination revealed a normally developed, well-nourished white female in severe distress. She was afebrile with respirations of 30/minute, pulse of 100/minute, and a blood pressure of 110/70 mm Hg. Upper extremity pulses were feeble. Femoral pulses were absent, while the lower extremities were cold and cyanotic with decreased capillary filling. The abdomen was distended and rigid with involuntary guarding and rebound tenderness. Bowel sounds were absent. Significant laboratory data included: haemoglobin and haematocrit of 10.8 g/dl and 29.3%, respectively; white blood cell count of 17 500 per mm³; with 71 neutrophils, 21 bands,

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and 8 lymphocytes; platelet count of 216 000 mm³; and prothrombin and partial thromboplastin times in the normal range. Serum electrolytes included sodium 137 mmol/l, potassium 4.2 mmol/l, chloride 104 mmol/l, bicarbonate 15 mmol/l, BUN 6.1 mmol/l/dl and a creatinine of 206 µmol/l.

Abdominal radiographs were interpreted as showing massive dilatation of the stomach (Figure) for the following reasons: (1) the cystic mass in the mid-abdomen extended into the pelvis, with displacement of gut inferiorly and laterally, outlined by serosa; (2) the presence of shifting collections of small floating air bubbles within homogeneous substance of water density, typical of retained gastric contents (not well reproduced in the Figure); and (3) a companion erect film, not illustrated, demonstrated a left subphrenic fluid level, typical of gastric fundus, continuous with the remainder of the abdominal mass. A nasogastric tube was seen radiographically to coil in the oesophagus despite repeated attempts to pass it into the stomach.

Figure Abdominal radiograph interpreted to show massive dilatation of stomach (supine film). Note that the cystic mass in the mid-abdomen extends into the pelvis, displacing gut inferiorly and laterally, outlined by serosa (arrows).

The patient was taken to the operating room where a laparotomy revealed a massively distended, gangrenous perforated stomach. No volvulus or adhesions were seen. Eight litres of fluid and undigested food were removed from the peritoneal cavity and stomach. The duodenum was dilated to the ligament of Treitz where there was a sharp cut-off. Focal areas of constriction, but no ischaemia, were present in both small and large bowel. Normal pulsations were seen in the superior mesenteric artery. Total gastrectomy was performed with closure of the oesophageal and duodenal stumps. The patient had a stormy post-operative course, became progressively hypotensive and developed renal failure. The platelet count dropped to 11 000 per mm³ and prothrombin and partial thromboplastin time became raised (14.5/11 and 42/32 respectively). Fibrin split products were absent. On the third hospital day the abdomen became distended with absent bowel sounds. Re-exploration of the abdomen revealed infarction of the entire small and large intestines. Resection was impossible and the patient died a few hours later.

In retrospect, the following history was obtained from the patient's mother and a former psychiatrist. At the age of 14, the patient first became conscious of her weight after being called 'overweight' by a peer. After her father's death, atypical dietary habits ensued. She would eat, sometimes large quantities, then would induce vomiting or exercise vigorously in order not to gain weight. When she was 17, she was hospitalised by her family doctor as her weight had dropped from 61 to 30 kg. She was described by her psychiatrist as a 'textbook case' of anorexia nervosa and was considered to have a severely distorted body image with many obsessive traits. Behaviour modification techniques resulted in the regaining of a significant portion of her lost body weight. At the age of 18 she was removed from therapy by her mother for no apparent reason. Over the past several years up to the present time the patient's mother related that she had periods of bulimia (overeating)—that is, 'she never knew when she was full.' Non-specific gastrointestinal complaints persisted, resulting in an appendectomy and cholecystectomy. Her mother had long made the association of exacerbations of gastrointestinal symptoms with emotional events. After being sent home the evening before her admission, the patient called her mother. Asked about any recent stressful experiences, she related that that morning she had been at the burial of a close friend who died of leukaemia.

PATHOLOGICAL FINDINGS

The total gastrectomy specimen was massively dilated and gangrenous (measuring 13.5 cm along the lesser and 39.0 cm along the greater curvature). The gastric wall was paper thin with a 1.0 cm perforation on the anterior wall. Microscopically, transmural ischaemic necrosis was evident.

At necropsy the patient had a mesomorphic habitus. The peritoneal cavity was soiled with gastric contents, including undigested vegetable matter. There was no occlusion of the coeliac, superior mesenteric, or inferior mesenteric arteries. The entire bowel was dilated and necrotic. The oesophagus was unremarkable.

Microscopically, fibrinoid necrosis of small mucosal and submucosal vessels of the bowel were present, but fibrin thrombi characteristic of disseminated intravascular coagulation were conspicuously absent. Most of the findings in the parenchymal organs were those seen in severe shock—that is, focal infarction of the liver, spleen, adrenals, and brain. A preferential infarction of the islets of Langerhans was striking in the pancreas. Minimal microscopic changes consistent with acute tubular necrosis were present in the kidneys. The hypothalamus was examined extensively, but only those findings attributable to shock were seen. The ovaries had markedly decreased numbers of primary follicles with conspicuous absence of follicular maturation. The endometrium had cystic atrophy. Postmortem blood cultures of heart, blood, pleural, and peritoneal fluid all grew *E. coli*, interpreted to be a manifestation of pre-terminal bacteraemia.

Discussion

Spontaneous rupture of the stomach (in the absence of a paraoesophageal hiatus hernia) is uncommon^{3-9,11}: 66 cases (including the present one) have been reported. This catastrophic event is more common in females (67%) and usually occurs along the lesser curvature (63%). It is uniformly fatal without operative intervention, while overall mortality is 73%. One half of the cases seem to be related to large meals and/or acute gastric dilatation. Infarction of the entire stomach, as a cause of spontaneous rupture, is a rare event (Table, cases 2 and 12).

The pathogenesis and pathophysiology of acute gastric dilatation and its complications have previously been studied in man and in animal models. Revilloid, in 1885, demonstrated that the stomach of cadavers had to be distended with at least 4 l of fluid to result in perforation.¹² It is stated that

intragastric pressure has to exceed gastric venous pressure to result in ischaemia and infarction.^{13,14} Increased intragastric pressure is usually the result of a closed loop, secondary to mechanical compression of the cardio-oesophageal and pyloro-duodenal or duodenojejunal junctions. Gastric infarction secondary to arterial insufficiency alone has not been recorded, because of extensive collateral circulation. Infarction is said not to occur even if all four major gastric arterial trunks as well as 80% of the smaller arterial branches are occluded. In fact, Somervell used this procedure in the treatment of 400 patients with duodenal ulcer to inhibit gastric secretion,¹³ without any complications. Gastric infarction, however, will occur in dogs if the gastric veins are also ligated.¹⁵

Volvulus of the stomach is rare¹⁶ but is the most common cause of infarction of this organ. However, it cannot be incriminated in this case, as it was not a finding at time of surgery. Spontaneous correction of a volvulus, though possible, is unlikely in view of the generalised gastric necrosis.

The so-called 'superior mesenteric artery' and 'ligament of Treitz' syndromes have been invoked by some to be the underlying causes of acute gastric and duodenal dilatation,¹⁷⁻²⁰ said to be particularly prevalent in emaciation. There are other conditions associated with acute gastric dilatation, including over-indulgence of food or drink, bicarbonate ingestion, volvulus, malrotation, diabetes mellitus, hypokalaemia, etc.^{1,21}

Psychogenic disturbances, specifically those related to abnormal eating habits, have been particularly mentioned as important aetiological factors in precipitating acute gastric dilatation.^{2-8,12,22} Eleven of 12 such cases from the literature (including the present one) had the diagnosis of anorexia nervosa (Table). All but one were young females, in keeping with the strong female predominance found in this disease.^{23,24} When mentioned, psychiatric problems tended to precede abdominal symptomatology.

All of the previous 10 cases of anorexia nervosa associated with acute gastric dilatation had histories of extensive recent weight loss (15 to 32 kg when stated) and were hospitalised to attempt refeeding. Related to this is the occurrence of acute gastric dilatation after rapid refeeding in emaciated prisoners of war.²⁵

Knowledge of the course of events in our patient is admittedly fragmentary, but, based on a few vital pieces of information, a highly plausible reconstruction is possible. Undoubtedly she had anorexia nervosa, even though a normal body

Table Summary of patients with gastric dilatation and associated psychogenic disturbances

Patient no.	Author and reference	Age (yr)	Sex	Psychogenic disturbance	Degree of recent weight loss (kg)	Treatment of gastric dilatation and/or operative findings	Outcome	Comment
1	Russell ²	16	F	AN*	32	Nasogastric suctioning. Acute removal of 2560 ml fluid from stomach	Recovery	Admitted for refeeding. Developed nausea and vomiting
2	Evans ³	20	F	AN	15	Gastrectomy for gastric infarction with perforation	Recovery	Admitted for refeeding. Treated with diet, insulin, and chlorpromazine. Developed acute abdominal pain
3	Scobie ⁴	18	F	AN	20	Laparotomy: grossly dilated stomach and proximal duodenum. Gastric evacuation and duodenojejunostomy performed	Recovery	Admitted for refeeding. Developed abdominal pain
4	Scobie ⁴	20	F	AN	31	Nasogastric suctioning: acute removal of 500 ml gastric fluid	Recovery	Follow-up at one year. Proximal duodenum dilated
5	Jennings and Klidjian ¹	14	F	AN	25	Nasogastric suctioning: Acute removal of 3500 ml gastric fluid	Recovery	Admitted for abdominal pain and vomiting. Developed shock
6	Jennings and Klidjian ²	25	F	AN	Unknown, however weight on admission was 25 kg	Nasogastric suctioning: acute removal of 3000 ml gastric fluid	Recovery	Developed sudden abdominal pain after meal and was subsequently admitted
7	Kerstein <i>et al.</i> ^{1,2}	18	F	Psychogenic polyphagia	Unknown, described as thin	1500 ml gastric fluid removed acutely. Poor response. Subsequent oesophagojejunostomy for infarction of stomach, duodenum, and distal oesophagus	Stormy postoperative course: pancreatitis, subphrenic abscess and pneumonia. Eventual recovery	Patient had continuously ingested food without chewing for eight hours. History of binge eating and self-induced vomiting
8	Brook ⁵	17	F	AN	Unknown	Nasogastric suctioning:	Recovery	Admitted for refeeding. Episodes of binge eating (3000 cal. diet plus 1 kg fruit cake) followed by acute abdominal pain
9	Bossing-ham ⁶	16	F	AN	Unknown	Nasogastric suctioning. Acute removal of 7000 ml gastric fluid	Recovery	Admitted for dietary and chlorpromazine treatment of AN. Hoarded food and secretly over-ate. Subsequently developed abdominal pain
10	Bossing-ham ⁶	19	F	AN	Unknown	Nasogastric suctioning: Acute removal of 4000 ml gastric fluid	Recovery	Admitted for AN. Secretly over-ate when found she wouldn't be released for Christmas holidays. Developed abdominal pain
11	Froese <i>et al.</i> ²²	16	M	AN	40% of his normal body weight	'Appropriate medical management'	Recovery	Admitted for refeeding. Sudden onset of nausea and vomiting. Upper gastrointestinal series revealed dilatation of stomach and distal duodenum
12	Saul <i>et al.</i> (present case)	22	F	AN	Negligible	Inability to pass nasogastric tube. Laparotomy: gastric and proximal duodenal dilation with gastric infarction and perforation. 8000 ml fluid and undigested food present in stomach and peritoneal cavity	Fatal	Presented to ER with abdominal pain and nausea. Sent home to return nine hours later with intensification of symptoms and rigid abdomen

*Anorexia nervosa.

habitus was found. However, peculiar eating habits were retained, which were especially exacerbated by emotional upsets. We assume that she ate and drank massively before her final admission, attested to by the 8 l of fluid and undigested food in her dilated, infarcted, and perforated stomach. The most prominent clinical finding was the closed loop associated with the massive acute gastric dilatation. The failure of the patient to decompress her stomach spontaneously by vomiting may in part be due to its presence, since no decompression tube could be passed through the obstructed cardio-oesophageal junction into the stomach. Death is attributed to bacteraemia and shock, the latter resulting in the extensive generalised ischaemic changes found at necropsy.

When compared with previously reported patients, our patient is unusual in several respects. First, this appears to be the first fatality in a patient with anorexia nervosa and acute gastric dilatation. Second, the previously reported cases were in the semi-starvation phase of their anorexic illness. This patient, as mentioned, was not emaciated and in that regard resembles patient no. 7 (Table). Bemis noted that patients with this disease may have alternating periods of semi-starvation and massive food intake (bulimia).^{23 26} Four patients (7, 8, 9, and 10: Table) had bulimic episodes in relation to their gastric dilatation. Self-induced vomiting and laxative abuse may be associated with binge eating. Long-term surveys of these patients show that less than half have satisfactory life adjustments and the menstrual cycle of a small number will not become normal again (evident from the abnormal ovarian and endometrial histological findings in our patient) when they regain normal body weight.^{23 24} Thus the chronic nature of the physical and/or emotional symptoms of this disease is stressed.

In conclusion, this paper directs attention to the fact that, although in anorexia nervosa the usual life-threatening condition is starvation,²⁷ acute gastric dilatation, an uncommon but well-recognised complication of this disorder, may be a cause of death, preventable by early recognition and treatment.

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