

## Case report

# Acute clostridial enteritis – or pig-bel?

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**SUMMARY** Acute postprandial gastric dilatation and associated clostridial enteritis necroticans is a well recognised but unusual clinical condition. Non-pathological overeating, gastric distension, and clostridial enteritis, termed pig-bel, has been reported in Papua, New Guinea.<sup>1-3</sup> A similar condition (Darmbrand) was reported from Germany after the second world war, but it is not a condition seen in our society today. Gastric dilatation alone may be seen in individuals with anorexia nervosa, who may occasionally indulge in episodes of overeating (bulimia).<sup>6</sup>

We wish to report a case of gastric dilatation associated with a fulminating enteritis, and discuss the similarities with enteritis necroticans (pig-bel).

### Case history

A 23 year old Caucasian woman was seen in the casualty department at 8 00 am one Sunday morning. She had walked in complaining of abdominal pain and distension. She told the medical staff that between midnight and 4 00 am that morning she had eaten a large meal, gone to sleep, and then awoken with abdominal pain. The meal she had eaten had consisted of 681 g of poorly-cooked liver, 907 g of kidney, 226 g of steak, two eggs, 226 g of cheese, two large slices of bread, one whole cauliflower, 454 g of mushrooms, 907 g of carrots, 10 peaches, four pears, two apples, four bananas, 907 g of plums, and 907 g of grapes. She was a photographic model and over the years deliberately tried to keep very thin. She periodically indulged in food binges, but not usually of the magnitude of her most recent one.

The patient was in obvious discomfort, with considerable abdominal distension. An abdominal radiograph confirmed a diagnosis of acute gastric dilatation. Attempts to aspirate the stomach with a wide bore nasogastric tube were unsuccessful. The patient's condition deteriorated, with increasing abdominal distension, laboured breathing, and a fall in blood pressure. With the worsening clinical picture, urgent gastric decompression was performed. As the abdomen was opened the grossly distended, ischaemic stomach protruded into the

wound. Approximately three litres of semi-solid food material was evacuated from the stomach, but there was considerable gaseous distension of the proximal small bowel, and the wall appeared ischaemic in places. The bowel was decompressed, and left for some minutes to see whether or not the appearance would improve. After 20 minutes, the bowel was once more distended with gas, its colour had not improved, and small gas bubbles were seen in the subserosal and mesenteric veins. At this stage, most of the small bowel was affected and appeared to be infarcted. No further operative procedures were thought to be practical, and the patient died a short while later.

Necropsy was performed 16 hours after death. The stomach and large intestine were extremely distended, and extensive haemorrhage had occurred into the small intestinal mesentery and the abdominal wall, spreading up over the lower chest wall. There was extensive haemorrhage into the mucosa of the lower oesophagus, stomach, small intestine, and the right side of the colon. Numerous submucosal gas bubbles were seen in the stomach and small intestine.

Histological sections from the stomach and small intestine showed microthrombi in the submucosal vessels, and mucosal necrosis was associated with an early neutrophil response. There were areas of patchy degeneration, necrosis, and haemorrhage in the liver parenchyma. Gram positive bacilli (probably Clostridia) were seen in the mucosa and

submucosa of the lower oesophagus, stomach, and proximal small bowel. *Clostridium perfringens* was cultured from the stomach, duodenum, and other regions of the small bowel, but no organisms were isolated from a blood sample.

### Discussion

This was a case of acute gastric dilatation after an episode of bulimia, followed by a Clostridial gastroenteritis and toxæmia.

The features of this case are strikingly similar to pig-bel, a disease described in the native inhabitants of Papua, New Guinea,<sup>1</sup> resulting from feasting on large quantities of pork, and found to be associated with B-toxin producing strains of *Clostridium perfringens*.<sup>2</sup> The B-toxin is particularly susceptible to breakdown by pancreatic proteases, the output of which may be markedly reduced by involuntary starvation.<sup>3</sup> In addition, the staple diet of the New Guinea population is the sweet potato, which contains a heat-stable trypsin inhibitor, rendering the individual more susceptible to the effects of the B-toxin. If large quantities of poorly-cooked meat containing the toxin are eaten by protease deficient individuals, an overwhelming toxæmia may occur.<sup>4</sup>

Acute gastric dilatation may also be produced by rapid feeding after periods of involuntary starvation, as was seen in prisoners of war after the second world war.<sup>5</sup>

It is probable that this patient suffered from bulimia nervosa, which involves an irresistible urge to overeat (followed by self-induced vomiting or purging), together with a morbid fear of becoming fat.<sup>6</sup> Episodes of bulimia are usually not fatal but perforation of the stomach may occur.<sup>7</sup>

This patient was unable to vomit, and nasogastric

aspiration, which is usually effective in decompressing the stomach in this situation, was unsuccessful. The rapid deterioration in her condition was related to proliferation of *Clostridium perfringens* in the upper gastrointestinal tract, in which there was damage to the mucosal barrier due to gross mechanical distension, followed by a toxæmia, indicated by liver damage and demonstration of intravascular coagulation, known effects of Clostridial toxins.<sup>8</sup>

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### References

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