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CASE REPORT

Gastric dilatation and perforation due to binge eating: a case report

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Abstract

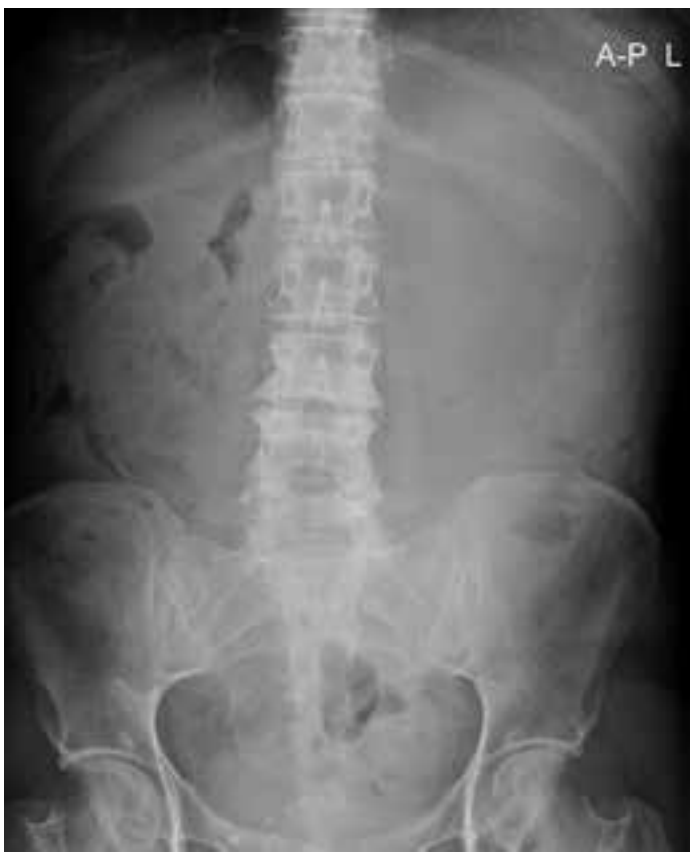
We present a case of massive gastric dilatation and necrosis in a patient with psychogenic polyphagia. The patient developed a *Candida albicans* sepsis due to gastric perforation and was treated with antifungal therapy and multiple surgical interventions.

Case

A 43-year-old female attended the emergency department with complaints of severe abdominal pain. The pain, mainly localized in the epigastric area, arose after the ingestion of a large amount of food one day prior to admission. The patient was suffering from nausea but was unable to vomit. She was afebrile and did not complain of altered bowel movements. Her medical history reported a lumbar sympathectomy, psychosis and an eating disorder (periods of polyphagia alternated with periods of extreme anorexia). An erect abdominal X-ray (*figure 1*) showed a remarkably distended stomach. The patient was admitted to the surgical ward with the suspected diagnosis of gastric distension, possibly caused by delayed gastric emptying provoked by her anti-psychotic drugs, namely *Olanzapine* and *Clomipramine*. She was put on 'nil by mouth' and was given a nasogastric tube, which immediately drained 2.5 litres of gastric fluid. However, in the next few hours the patient's condition deteriorated, showing signs of shock with progressive tachypnoea, tachycardia, cold extremities and falling blood pressure. At emergency laparotomy, the ventral side of the stomach was found to be completely necrotic and had perforated from the distal oesophagus to the pylorus. Further, 3.5 litres of gastric fluid and undigested food particles had leaked into the abdominal cavity. Extensive abdominal lavage was performed, followed by resection of the necrotic ventral side with reconstruction of a "tube-like" stomach, using the vital dorsal side of the stomach.

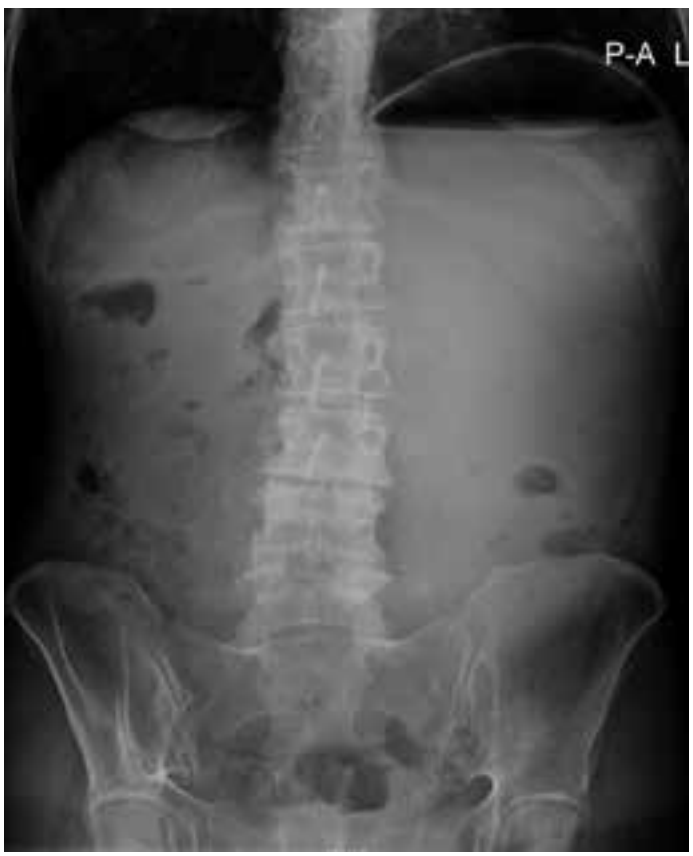
After surgery, the patient was admitted to the Intensive Care Unit (ICU), where she was mechanically ventilated and treated in accordance with the ICU's sepsis protocol (inotropes, cefotaxime, metronidazole and a single dose of gentamicin), continuous venovenous hemofiltration and administration of amphotericin, colistin and tobramycin in mouth and stomach (routine selective decontamination of the digestive tract). Although at this stage

Figure 1. Abdominal X-ray showing a massively distended stomach



there was only slight clinical suspicion of candidemia, fluconazole was added as a precautionary action because of the severity of the sepsis. On the fifth day of admission, abdominal cultures taken at the laparotomy as well as a blood culture showed *Candida albicans* and fluconazole was replaced by caspofungin. All intravenous and arterial lines were replaced. Fundoscopy of both eyes by an ophthalmologist did not show signs of Candida endophthalmitis. Subsequent blood cultures (9 sets) on different days were negative for *C.albicans*. Despite the antifungal treatment with caspofungin, the patient's abdominal fluid remained positive for *C.albicans.* After seven days of caspofungin monotherapy, fluconazole was again added to the antifungal regimen in order to augment the treatment with higher tissue levels. *C.albicans* was found in abdominal samples up to 3 weeks after admission. The *C.albicans* found in the blood and peritoneal fluid was sensitive to fluconazole, tested in the Vitek system (Biomerieux). A total of 16 re-laparotomies were performed in the following five weeks because of recurrent gastrointestinal leakage and inadequate source control. This eventually resulted in a subtotal gastrectomy. Both the oesophagus and the leaking antrum stump were drained by Foley catheters. Also the left hemicolon was resected together with a large part of the small bowel due to ongoing leakage from various fistulas. The patient had a surgical procedure for end colostomy of the transverse colon. Eventually the patient's clinical condition stabilized. She was weaned from mechanical ventilation and no longer in need of inotropic agents. All antifungal therapy was stopped after 29 days and cultures remained negative thereafter.

Figure 2. Abdominal X-ray showing a massively distended stomach



After 46 days of ICU-admission the patient was transferred to the surgical ward for further recovery. Eventually, 118 days after admission, the patient was transferred to a university hospital for home parenteral nutrition (TPN) training. After almost one year of TPN, bowel continuity was restored in a ten hour surgical procedure in which, after extensive adhesiolysis, a Roux and Y oesophagojejunostomy was created with reconstruction of the abdominal wall with a Ramirez-plasty. The patient is currently recovering from this surgery.

Discussion

It is well known that the stomach has rich vascularisation and collateral blood supply. As a result, gastric infarction due to an insufficient perfusion is a rather rare condition and its exact pathophysiology remains unclear. The reported causes of gastric infarction are diverse and include volvulus, acute necrotizing gastritis, intrathoracic herniation, the ingestion of caustic materials, vascular compromise and acute gastric dilatation[1]. Experimental models have shown that gastric infarction will only appear when both venous and arterial occlusion is present[2]. In a massively distended stomach, the intragastric pressure can reach over 30cm H_2O exceeding the gastric venous pressure, therefore compromising venous drainage of the stomach[3]. In healthy individuals, satiety mechanisms prevent over-distension of the stomach. However, in patients suffering from eating disorders, these satiety mechanisms are often insufficient or absent. Geliebter et al[4] showed that patients with bulimia nervosa have a significantly larger stomach capacity, possibly due to repetitive expansion of the stomach during binge eating. Some studies have found evidence of delayed gastric emptying in bulimic patients, possibly due to smaller gastric contractions in the enlarged stomach[5,6,7]. These gastrointestinal changes in patients with eating disorders might lead to a higher risk of developing gastric infarction. The incidence of acute gastric dilatation in anorexia and psychogenic polyphagia seems to be higher in females (67%)[8]. A distended abdomen and the urge to vomit are the most common early signs of gastric distension[9]. With progression of the gastric distension, continuous abdominal pain and the inability to vomit are the predominant findings. An abdominal X-ray may show gastric dilatation (and, in rare cases of recurrent gastric distension, gastric pneumotosis caused by gas-forming pathogenic bacteria infiltrating the injured stomach wall[10]). With clinical signs of gastric distension, prompt decompression by nasogastric suctioning should be initiated.

When gastric decompression is delayed, progression of the gastric distension may lead to gastric infarction and subsequently gastric perforation. The overall mortality of gastric infarction is 73%[11]. Mortality can often be attributed to severe sepsis and multi-organ failure as a result of gastric perforation.

In our case, necrosis and perforation of the stomach resulted in an ongoing peritonitis and candidemia. Candidemia requires systemic antifungal treatment. Studies show a high mortality rate associated with candidemia, which is highest in those patients who are not treated with an antifungal drug[12,13]. In a patient who is clinically suspected of candidemia (e.g. presenting with classic skin or eye lesions), preemptive antifungal therapy should be initiated while awaiting the return of blood cultures. Yet most patients have no obvious signs suggesting the presence of candidiasis. In those cases, depending upon the acuity of the patient's condition, clinicians should consider whether it is appropriate to initiate empiric treatment with an antifungal agent. The 2009 Infectious Diseases Society America (IDSA)[14], and the 2008 Dutch SWAB[15] treatment guidelines for candidiasis recommend an echinocandin for the treatment of candidemia in patients who are considered to be 'moderately severe' or 'severely' ill[14].

Conclusion

Massive gastric dilatation caused by psychogenic polyphagia can progress to gastric necrosis and perforation. Therefore, physicians should always consider the possibility of acute gastric dilatation when a patient complains of abdominal pain after the ingestion of a large meal, especially in patients with psychiatric co-morbidity. Gastric dilatation can often be treated conservatively, but progressive gastric dilatation may result in ischemia, gastric perforation and subsequent multiple organ failure.

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