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Acute gastric perforation after leaving against medical advice: A case presentation

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ABSTRACT

Introduction: Gastric perforation with necrosis is rare following acute gastric dilation (AGD) and can be fatal. We present a case of a patient with AGD due to a binge-eating episode who left the emergency department (ED) against medical advice (AMA) only to return with gastric perforation and necrosis requiring total splenectomy and partial gastrectomy.

Case: A 28-year-old female without a remarkable past medical history presented to the ED with diffuse abdominal pain and obstipation after a three-day “food crawl.” On admission, a computerized tomography (CT) scan revealed a markedly dilated stomach from the diaphragm to the pelvis with severe mass effect. The therapeutic plan at the time was gastric decompression via a nasogastric tube. The following day, the patient reported feeling better and left AMA only to return the same evening with worsening symptoms and peritoneal signs. The patient was then emergently taken to the operating room (OR). In the OR, laparotomy revealed frank spillage of partially digested food and necrosis along the greater curvature of the stomach that extended to the spleen. Damage control surgery was performed, which required a total splenectomy and a partial gastrectomy. The patient was admitted to the intensive care unit (ICU) and subsequently underwent five more trips to the OR due to severe edema that delayed the primary closure of the fascia. Once the patient was transferred out of the ICU, she was evaluated by psychiatry and diagnosed with a binge-eating disorder.

Conclusion: This case demonstrates the severity of acute gastric dilation and its potentially lethal consequences. In some cases, such as this one, the patient may present with mild symptoms and not comprehend the gravity of the situation. Therefore, it is important for clinicians to recognize this condition as a true emergency and perform immediate decompression and evaluation for surgery.

Case

A 28-year-old female with no past medical or surgical history was referred to our emergency department (ED) from an urgent care center due to a three-day history of diffuse abdominal pain with obstipation. The patient reported participating in a “food crawl” three days earlier, during which she visited a series of restaurants consuming a large amount of food at each.

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Social history includes employment as a cook, with no alcohol, tobacco, or illicit drug use. Upon arrival to the ED, the patient was afebrile and hemodynamically stable with abdominal pain, nausea, and constipation. Body mass index (BMI) was 22.4. Electrolytes, glucose, and liver function tests were normal. Lactic acid was 5.1 mg/dL, WBC 13.7 mL, and hemoglobin 14.2 g/dL. The abdomen was firm and severely distended. There was no peritonitis, but there was diffuse tenderness to superficial palpation and tympany on percussion. A nasal gastric tube (NGT) was placed with output of 1.5 L of liquid with particles resembling undigested food.

The patient underwent a CT with contrast which showed a markedly dilated stomach extending from the diaphragm to the pelvis with severe mass effect on the abdominal and pelvic structures. Oral contrast appeared within the compressed retro-gastric bowel loops and the colon appeared decompressed distally (Figs. 1, 2, 3).

The therapeutic plan was non-operative management with gastric decompression via NGT, but the following day, the patient left against medical advice (AMA) due to financial concerns. That same night, she returned to the ED with worsening abdominal pain. Upon arrival, she was tachycardic in the 160's. SpO₂ on 5 L nasal cannula was 86%. She was intubated due to overall deteriorating condition. Pertinent lab findings included WBC 2.1 mL and lactic acid 4.2 mg/dL. CT with contrast redemonstrated severely distended stomach, this time with a significant posterior defect at the gastric fundus and new moderate to large volume peritoneal fluid. Physical exam now demonstrated peritonitis. Due to gastric perforation concerns, the patient was taken emergently to the operating room (OR) for an exploratory laparotomy.

Laparotomy revealed frank spillage of several liters of partially digested food. Evaluation of the stomach showed a large area of necrosis and perforation extending along the proximal greater curvature to the spleen, which was adherent to the gastric wall. Due to hemodynamic instability, we made an early decision to perform damage control surgery and seal all perforations of the GI tract, which required a total splenectomy and a partial gastrectomy. The procedure was then concluded with temporary abdominal closure.

The patient was admitted to the intensive care unit (ICU) for stabilization and she returned to the OR five additional times, where a Wittman patch was placed for sequential approximation of the fascia, before it was closed primarily. The limiting factor for the primary closure was tension on the fascia caused by visceral edema. Eventually, enteral feeding was started, the patient was extubated, and she was transferred out of the ICU on hospital day 21. When she was better able to discuss what had happened, she stated that she and three male companions had eaten about five meals worth of food over three hours. She denied previous overconsumption of food or any other disordered eating. She denied weight consciousness, dieting, or purging. The family denied any previous extreme episodes but reported instances of heavy eating in one sitting with a bloated feeling and a long fast afterward.

The patient was evaluated by psychiatry and diagnosed with panic disorder and an unspecified eating disorder- most likely binge-



Fig. 1. Coronal section of contrast-enhanced CT showing the severely distended stomach on admission.

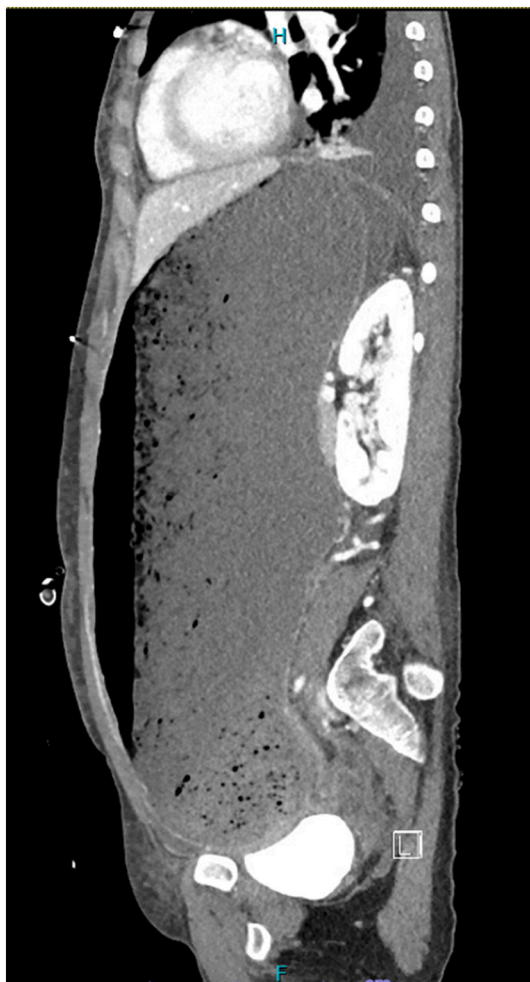


Fig. 2. Sagittal section of contrast-enhanced CT showing the severely distended stomach on admission.



Fig. 3. Axial section of contrast-enhanced CT showing the severely distended stomach on admission.

eating disorder. They recommended outpatient therapy. The patient was discharged home on hospital day 28. She left tolerating a regular diet, having regular bowel movements, and ambulating with a wheeled walker. On a routine clinic visit 2 weeks later, she reported living independently and eating normally. Her BMI was measured at 15.6, which may represent her premonitory weight without the gastric contents and edema associated with a critical illness. She was then discharged from the surgical service with plans for outpatient psychiatric follow-up.

Discussion

We present an unusual case of hyperphagia leading to massive gastric dilation and perforation, with novel psychological and surgical features. Acute gastric dilation is a rare phenomenon with a variety of potential mechanisms. Some proposed etiologies include a heavy meal, infection, diabetes mellitus, trauma, and psychiatric conditions such as anorexia nervosa and bulimia [1].

In this case, the gastric distention followed a documented episode of binge eating. Given the patient's psychological profile and lack of identifiable organic cause, binge eating disorder seems to be the most likely etiology. The patient's low BMI on follow-up may be related to catabolic effects of a critical illness but may also reflect a component of anorexia. Binge eating disorder is one of the three major types of eating disorders and is the most common, with a lifetime prevalence in the United States of 2.6% [2]. The patient's diagnosis of panic disorder is typical. Eating disorders are frequently seen in association with other psychiatric illnesses, especially mood and anxiety disorders [2,3].

The pertinent behavioral question is why the patient consumed such a massive quantity of food and why she felt well enough to leave AMA, which nearly resulted in her death. Seeking medical care, refusing treatment, and leaving the hospital due to massive gastric distention have not been reported previously. Denial of the seriousness of a medical condition is consistent with an eating disorder. Patients with eating disorders often exhibit cognitive dysfunction, including impaired decision making [3].

Disordered satiety has also been described in patients with eating disorders. Typically, when one eats, there is an interplay of hormones in the brain that interact with the neuroendocrine system of the gut. Neurotransmitters such as dopamine and endogenous opioids are associated with appetite, while serotonin and acetylcholine are associated with satiety. In certain animal models, eating disorders modify the way these neurotransmitters are expressed. Brain imaging studies on humans have shown similar findings. For those having a binge-eating disorder, positron emission tomography (PET) scans have shown an increase in dopamine dysregulation [5]. In our patient, secondary endocrine dysfunction may have limited the ability to perceive satiety. Mishima [6] reports that, in addition to reduced feeling of satiety, patients with eating disorders often do not feel severe abdominal pain even with massive gastric distention [6]. This may explain why our patient waited to come to the ED and why she left AMA. Additionally, eating disorders may lead to secondary effects on GI motility, specifically gastric emptying [3].

Once the stomach dilates excessively, potential complications are gastric ischemia, necrosis, and perforation. Due to the rich vascular network of the stomach, these complications are rare. However, when extreme intraluminal pressure exceeds circulatory perfusion pressure, necrosis ensues [2,6]. First-line treatment for acute gastric distention includes NGT decompression and fluid resuscitation. Once the condition progresses to gastric necrosis, perioperative mortality rate is 50% to 65% [7]. Our patient required splenectomy as part of a damage control procedure necessary to expeditiously close the gastric perforation. Splenectomy has long been recognized as part of damage control surgery for trauma [8], and gastric necrosis has been reported as a complication of splenectomy [9]. However, this is the first report of a damage control splenectomy for non-traumatic gastric necrosis. In patients with eating disorders, signs of impending gastric rupture may be masked by cognitive deficits and chronic physiologic changes in the GI tract. Clinicians must maintain a high index of suspicion to recognize this complication early.

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