Acute Gastric Dilatation and Acute Pancreatitis in a Patient with an Eating Disorder: Solving a Chicken and Egg Situation

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Abstract

A 26-year-old woman with an eating disorder presented to the emergency department with severe abdominal pain following binge eating. A plain film X-ray demonstrated a huge dilatation of the stomach with a high air-fluid level. Serum amylase was 2,265 IU/L, and serum lipase was 2,001 IU/L. Abdominopelvic computed tomography scan revealed a massive gastric dilatation and completely compressed duodenum. The distended right colonic loop and small bowel loops were reduced to the pelvic area and the displaced small bowel and mesenteries tightly pulled on the mesenteric vasculature. After nasogastric tube decompression and irrigation, her abdominal pain subsided. On the 15th day after admission, a follow-up abdominopelvic computed tomography scan demonstrated mild edematous changes of the pancreas compatible with pancreatitis.

Key words: anorexia nervosa, bulimia nervosa, eating disorder, gastric dilatation, pancreatitis

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Introduction

Severe gastric dilatation is an unusual but serious condition that has been reported in association with anorexia nervosa, particularly the binge/purge subtype. Episodes of acute gastric dilatation may result in serious sequelae, including gastric necrosis (1), gastric perforation (2), and even death (3). There have been four peculiar cases reported presenting with the simultaneous occurrence of massive gastric dilatation and acute pancreatitis in a patient with an eating disorder (4-6). Three previous reports have proposed possible mechanisms for elucidating which situation preceded the other, the acute gastric dilatation, acute pancreatitis, or a hidden event causing both phenomena (4-6). However, the information from these cases did not provide a sufficient amount of clear evidence to support any of these theories. Here, we clarify the sequential events of acute gastric dilatation and acute pancreatitis in a patient with an eating disorder through objective images and laboratory findings.

Case Report

A 26-year-old woman presented to the emergency department with severe abdominal pain lasting for approximately 40 minutes following binge eating. The patient came from a middle class family and was the second of three sisters. She had been obsessed with dieting and had frequently taken laxatives for losing weight since middle school. The patient also performs binge eating during times of stress. Following graduating from a university, she was not able to find employment and was under significant social stress. Six months prior to presentation, she began to undergo alternating episodes of anorexia nervosa and bulimia nervosa. Six hours before admission, she ate 300 grams of pork, 1 packet of ramen, 80 slices of bread, 1.5 liters of Coke, and 1.5 liters of another soda. She then fell asleep but awoke from a deep sleep due to intense abdominal pain. Physical examination revealed a distended abdomen, epigastric tenderness, and hypoactive bowel sounds. Her body weight was 48.5 kg and, height was 173 cm. The initial electrocardiograph was unremarkable. She had blood pressure of 110/80 mmHg, heart
rate of 125 beats per minute, respiratory rate of 16 times per minute, and body temperature of 36.7°C. Intravenous fluid replacement was immediately initiated and a nasogastric tube was positioned. A plain film X-ray demonstrated a huge dilatation of the stomach with a high air-fluid level (Fig. 1). Serum amylase was 2,265 IU/L, and serum lipase was 2,001 IU/L. Laboratory data were notable for the following; white blood cell count, 1,1100/mm³; C-reactive protein, 3.6 mg/dL; erythrocyte sedimentation rate, 29 mm/hr; AST, 21 IU/L; ALT, 14 IU/L; ALP, 116 IU/L; γ-GTP, 49 IU/L; PT, 91%; total bilirubin, 0.53 mg/dL; arterial oxygen pressure, 95 mmHg; base excess, -2 mEq/L; blood urea nitrogen, 20 mg/dL; creatine, 0.8 mg/dL. Abdominopelvic computed tomography scan revealed a massive gastric dilatation occupying the entire abdominal cavity from the diaphragm to the pelvis with a large volume of food inside the stomach (Fig. 2A). The markedly dilated stomach compressed the transverse portion of the duodenum completely (Fig. 2B). The distended right colonic loop and small bowel loops were reduced to the pelvic area and the displaced small bowel and mesenteries tightly pulled on the mesenteric vasculature (Fig. 2C). There were no findings compatible with acute pancreatitis. Nasogastric tube decompression and irrigation was initiated. A total of 6,000 cc of greenish semisolid material and food particles were evacuated, and the acute gastric dilatation might follow, and the increased intra-abdominal pressure may cause passage of duodenal contents retrogradely along the gastric lumen. However, an animal

**Figure 1.** Plain film X-ray upon admission. A huge gastric dilatation with a high fluid level was observed.

Since acute gastric dilatation was first described by Duplay (7) in 1883, there have been four peculiar cases simultaneously presenting with acute gastric dilatation and acute pancreatitis (4-6). Three previous reports have linked acute gastric dilatation with acute pancreatitis during the refeeding phase in anorexia nervosa patients and described a possible mechanism between the two acute diseases (4-6). However, there was not much information to assess the relationship between the two diseases, such as serial serum amylase and lipase levels and computed tomography scans. In contrast to previous reports, the present patient provided us with sufficient information to reveal the relationship between acute gastric dilatation and acute pancreatitis.

There are three possibilities explaining the relationship between acute gastric dilatation and acute pancreatitis in a patient with an eating disorder (4, 8). First, acute gastric dilatation could be one of the complications caused by acute pancreatitis in a patient with an eating disorder. The eating disorder itself has also been thought to provoke acute pancreatitis (9-12). Gryboski et al. (11) described children with pancreatitis associated with refeeding among anorexia nervosa patients with elevated lipase activity. Isaacs et al. (12) reported a patient with a well-documented eating disorder who developed severe pancreatitis following binge eating. However, the patient reported by Isaacs et al. (12) did not develop gastric dilatation despite severe pancreatitis. One patient, reported by Gryboski et al, developed both diseases, but the acute gastric dilatation did not seem to result from acute pancreatitis, but rather it resulted in acute pancreatitis. Second, superior mesenteric syndrome can link acute pancreatitis and acute gastric dilatation. If duodenal compression by the superior mesenteric artery is the initial event, then gastric dilatation might follow, and the increased intraduodenal pressure may cause passage of duodenal contents retrogradely along the pancreatic duct. However, an animal

**Discussion**

Since acute gastric dilatation was first described by Duplay (7) in 1883, there have been four peculiar cases simultaneously presenting with acute gastric dilatation and acute pancreatitis (4-6). Three previous reports have linked acute gastric dilatation with acute pancreatitis during the refeeding phase in anorexia nervosa patients and described a possible mechanism between the two acute diseases (4-6). However, there was not much information to assess the relationship between the two diseases, such as serial serum amylase and lipase levels and computed tomography scans. In contrast to previous reports, the present patient provided us with sufficient information to reveal the relationship between acute gastric dilatation and acute pancreatitis.

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A study found that duodenal obstruction did not cause acute gastric dilatation (13). Therefore, it seems not reasonable to follow this idea. Third, acute gastric dilatation can cause acute pancreatitis through duodenal obstruction. The introduction of food following a prolonged period of reduced intake and severe emaciation may lead to gastric dilatation (6). Accordingly, compression by the distended stomach and traction of the vascular pedicle by the small intestine and mesentery could cause duodenal obstruction. The resultant rise in duodenal pressure may produce reflux into the pancreatic ducts, and initiate the inflammatory reaction (6).

McCutcheon and Race (14) demonstrated in canine experiments that duodenal contents under pressure can be forced past normal pancreatic duct papillae, causing pancreatitis.

The clinical course of the present case supports the third theory that acute gastric dilatation causes acute pancreatitis when the two diseases co-exist in patients with eating disorders. After binge eating, our patient clearly developed huge gastric dilatation, and the displaced small bowel and mesenteries, deep in the pelvic cavity, significantly pulled down the mesenteric vasculature. Huge gastric dilatation and traction by displaced mesenteries compressed the duodenum,

Figure 2. Abdominopelvic computed tomography scan on admission. (A) A massive gastric dilatation occupied the entire abdominal cavity from the diaphragm to the pelvis with a large volume of food found inside the stomach, 38 cm × 23 cm in size (white dotted lines). (B) The markedly distended stomach compressed the transverse portion of the duodenum (white arrows). Fluid retention in the proximal duodenum and the distinctive features of valvulae conniventes were observed (white arrowheads). (C) The distended right colonic loop (white arrows) and small bowel loops (white arrowheads) were reduced into the pelvic area. The mesenteric vasculature was pulled down by the displaced small bowel and mesenteries (black arrows).
and finally caused obstruction. These are visualized by radiologic images (Fig. 2). Finally, a rise in duodenal pressure caused reflux into the pancreatic duct and provoked pancreatitis. The presence of pancreatitis, which is clearly supported by highly elevated serum lipase and amylase, with abnormal liver function tests, lasted for over 2 weeks; follow-up computed tomography scan revealed an edematous pancreas with a swollen lymph node. The first theory that acute pancreatitis causes acute gastric dilatation cannot be supported by our case. Although the pancreatitis persisted for over at least 14 days, acute gastric dilatation was resolved in a day after nasogastric decompression and irrigation. If acute pancreatitis had been the cause of acute gastric dilatation, acute gastric dilatation would have been resolved after the resolution of acute pancreatitis. The second theory cannot be supported by our case either. Our patient did not have superior mesenteric syndrome though her body mass index was 16.2 kg/m². Duodenal obstruction was caused not by the superior mesenteric artery but by the distended stomach.

When encountering this rare situation, a physician should perform nasogastric decompression and irrigation as soon as possible. Rapid gastric decompression resolves duodenal obstruction and finally eradicates the possibility of pancreatitis. After gastric decompression, initiating refeeding should be started carefully as the introduction of food following a prolonged period of reduced intake and severe emaciation may lead to gastric dilatation again. The present patient started drinking a small amount of liquid diet on the 12th hospital day and the amount was gradually increased. The simultaneous occurrence of acute huge gastric dilatation and acute pancreatitis in patients with eating disorders is quite rare. To date, only 5 cases have been reported in the literature, including the current case. However, what makes this case more valuable is that the findings prove the theory that acute gastric dilatation causes acute pancreatitis in patients with eating disorders. Duodenal obstruction by the distended stomach and the subsequent traction on the vascular pedicles, due to the displaced small bowel and the mesenteries, was clearly visualized; if we had used magnetic resonance imaging, we could have visualized the compression of the third portion of the duodenum by the mesenteric vasculatures more clearly. In light of these findings, we conclude that acute gastric dilatation precedes acute pancreatitis, although the two diseases can seem to occur simultaneously.

The authors state that they have no Conflict of Interest (COI).

References


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