Abstract

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Acute gastric dilatation and hepatic portal venous gas in a patient with severe anorexia nervosa

D'Ambrosio D*[1], Mariniello A[2], Damiano S[1], Del Prete I[1], Giovine S[3], Iuliano D[4], Ievoli F[1]

[1]Internal Medicine Ward, S. Giuseppe Moscati Hospital, Aversa, ASL Caserta, Italy
[2]Digestive Endoscopy Unit, S. Giuseppe Moscati Hospital, Aversa, ASL Caserta, Italy
[3]Radiology Unit, S. Giuseppe Moscati Hospital, Aversa, ASL Caserta, Italy
[4]Gastroenterology and Digestive Endoscopy Ward, Marcianise Hospital, ASL Caserta, Italy

Background: Gas within the gastric wall is an alarming finding and a rare condition. Clinically, this condition is divided into two entities: gastric emphysema and emphysematous gastritis. These two diseases should be differentiated because they are characterized by different clinical symptoms, possible etiology, treatment and prognosis. Rarely, anorexia nervosa and bulimia nervosa have been described to be associated with acute gastric distension and duodenal obstruction induced by superior mesentery artery syndrome. So, gastric emphysema could be accompanied by acute gastric distension induced by anorexia nervosa.

Case report: A 31-year-old female with anorexia nervosa presented to the ED for epigastric pain. Physical examination revealed abdominal distension. Laboratory tests were unremarkable except for low normocytic anaemia. An abdominal X-ray showed a very large amount of gas in the fundus. Nasogastric decompression was applied and CT examination revealed a huge stomach, with intramural and hepatic portal venous gas, in absence of perforation of the luminal organs. Emergency EGDS highlighted mucosal edema and erosive and ulcerative areas compatible with ischemia all around the stomach. The patient was followed up with close radiologic and endoscopic surveillance under nasogastric decompression, parenteral nutrition and wide-spectrum antibiotic therapy. Clinical symptoms decreased dramatically on the 2° day, and radiological and endoscopic findings gradually disappeared, with discharge in the 8°day in good clinical status.

Discussion: The picture of acute gastric dilatation (AGD) and hepatic portal venous gas is a rare occurrence in patients with anorexia nervosa. Acute gastric distention is caused by a myriad of etiologies, which may cause ischemic injury to the stomach, such as gastroparesis, eating disorders, electrolyte imbalances, psychogenetic polyphagia, and obstructions. The diagnosis therefore needs to be promptly made in order to rule out gastric perforation and haemorrhage. Despite the extensive gastric collateral circulation, acute intragastric venous pressures greater than 14 mmHg may lead to impaired intramural blood flow and can therefore cause mucosal necrosis. One of the most common mechanistic theories for acute gastric dilation in patients with anorexia is gastroparesis, which develops frequently due to food restriction with weight loss. However, more nuanced physiological mechanisms have been proposed for the mechanism of acute gastric dilation in eating disorders, such as delayed gastric emptying, diminished gastric relaxation, diminished release of cholecystokinin and abnormalities in enteric autonomic function. However, the exact pathogenesis remains unclear. Our case demonstrates the complexities of the pathophysiology of AGD, as our patient had no obvious obstructive cause.

Conclusion: This report adds to an underreported but important complication of anorexia nervosa. The recognition and correct diagnosis of this condition is necessary for appropriate patient management.