ARCHIVES OF HELLENIC MEDICINE: ISSN 11-05-3992

CONTINUING MEDICAL EDUCATION
ΣΥΝΕΧΙΖΟΜΕΝΗ ΙΑΤΡΙΚΗ ΕΚΠΑΙΔΕΥΣΗ

ARCHIVES OF HELLENIC MEDICINE 2022, 39(4):571–572
ΑΡΧΕΙΑ ΕΛΛΗΝΙΚΗΣ ΙΑΤΡΙΚΗΣ 2022, 39(4):571–572

# Surgery Quiz – Case 41

**A. Kostouros,**
**V. Kyriakidis,**
**A. Kyriakidis**

*Department of General Surgery, General Hospital of Amfissa, Amfissa, Greece*

A 19-year-old boy with spastic paralysis due to congenital developmental disorders was examined at the emergency department because of acute abdominal pain, nausea, one episode of vomiting and epigastric distension. A bowel enema was administered at home without any relief of the symptoms. During initial assessment, the patient was afebrile and had $SpO_2$ of 94%, cardiac frequency of 150 bpm and blood pressure of 137/84 mmHg. Physical examination revealed profound abdominal dilatation, greater over epigastrium, metallic intestinal sounds, tympanic sounds during percussion and diffuse peritoneal abdominal pain during palpation. An abdominal radiograph (fig. 1) depicted great gastric dilatation. A nasogastric tube was inserted, 3 litres of mixed gastric and bilious content was suctioned and a new abdominal radiograph was performed (fig. 2). Nasal $O_2$ catheter was utilized and the patient was admitted in the Internal Medicine ward, where he quickly ameliorated.

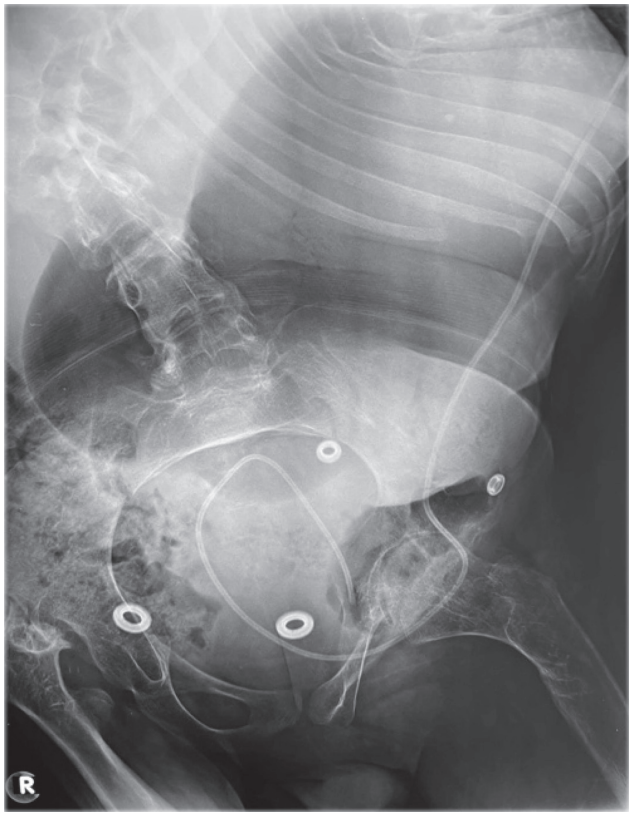

**Figure 1.** Abdominal radiograph, before nasogastric tube insertion.

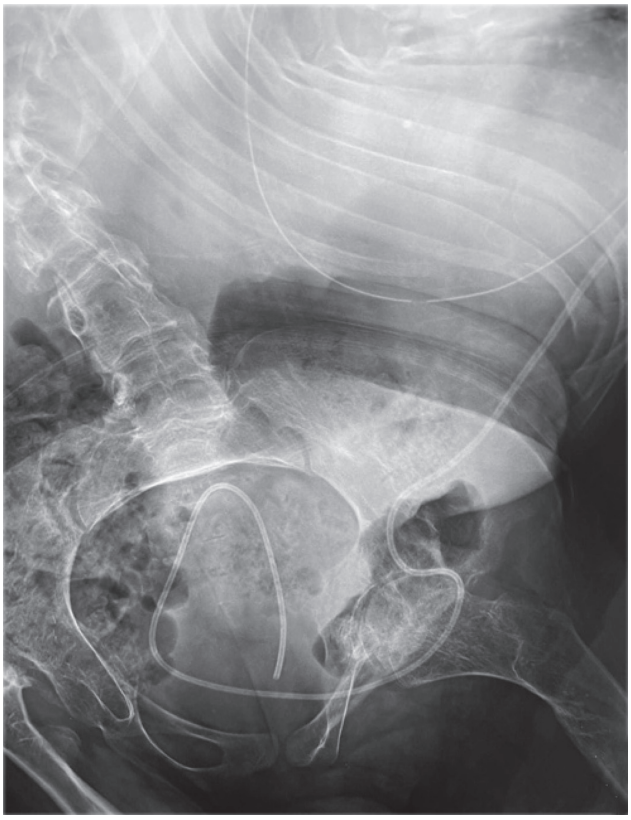

**Figure 2.** Abdominal radiograph, after nasogastric tube insertion.

## Comment

*Acute gastric dilatation (AGD) may concern only stomach, or expand until ligament of Treitz. It can deteriorate very quickly and may prove fatal in case it complicates with respiratory distress, compartment syndrome, gastric necrosis and or emphysema with or without rupture and sepsis. Possible etiology of AGD includes postoperative complications, acute vascular insufficiency, necrotizing inflammation and underlying morbidities combined with binge eating. In case of ischemia and perforation, mortality ranges between 80% and 100%.*

*Common symptoms of AGD include acute abdominal pain, increased or absent intestinal sounds and nausea. It is noteworthy that patients are often unable to vomit or vomit only small amounts of content because of closure of the gastroesophageal junction. This occurs as esophagus gets compressed by the right diaphragmatic crus due to dislocation by the neighboring fundus of the distended stomach. Patients with AGD exhibit compression of aorta with subsequent absent femoral pulses and cold, cyanotic lower extremities, whereas inferior vena cava compression reduces venous return and lead to hypotension. Third space fluid loss and hepatic and splenic congestion also contribute to the decrease of preload that diminishes blood pressure. In the beginning, vagus nerve stimulation provokes bradycardia but abdominal pain and hypotension eventually cause tachycardia. AGD also induces elevation of diaphragm, which in turn decreases lung compliance and increases airway resistance, giving rise to progressively worsening dyspnea.*

*AGD pathophysiology is not definite. According to atonic theory, long periods of starvation lead to gastric atony and muscular atrophy. Therefore, rapid consumption of great amount of food cumbers the already weak stomach. Furthermore, mechanical theory assumes that the cardioesophageal and either the pyloroduodenal or the duodenojejunal junction get compressed. Probably those mechanisms occur simultaneously in some degree. Gaseous distension usually precedes fluid distention. Fluid comes mainly from congested capillaries whereas gastric, pancreatic and bilious secretions, as well as shallowed saliva, food and drink accumulate and contribute to the total fluid volume as they cannot be pushed through duodenum towards jejunum. During operations, anesthesia and or hypoxia relaxes upper esophageal sphincter and positive pressure ventilation may force air towards stomach, potentially initiating the sequel of events of AGD.*

*Stomach exhibits vascular abundancy. As a result, gastric ischemic necrosis requires not only left and right gastric and gastroepiploic artery occlusion but loss of more than 80% of collateral arterial supply as well. Gastric ischemia presupposes that intragastric pressure exceeds gastric venous pressure. Gastric pressure of 14 mmHg or more than 3 litres of fluid are sufficient to lead to moderate ischemia. If pressure augments greater than 120 mmHg or if fluid accumulation surpasses 4 litres, gastric wall may rupture. Greater curvature and fundus are more vulnerable to ischemia and necrosis than lesser curvature and pylorus.*

*Diagnosis is based on abdominal radiograph, which usually features gastric distension, air-fluid level and displacement of adjacent structures laterally, downwards or upwards around stomach. Computed tomography (CT) can also be used. Early diagnosis is of upmost significance because early treatment is needed in order to decompress stomach and prevent the life-threatening complications. High suspicion index is required in order not to miss such a diagnosis.*

*Initial treatment comprises insertion of nasogastric tube to remove gastric content and abrogate AGD, permitting gastric blood flow. Further conservative measures are IV fluids and nasal $O_2$ in case of respiratory distress and hypotension. If these measures prove unsuccessful or if gastric rupture is suspected, laparotomy is implemented. If necrosis is found, total gastrectomy and esophagojejunostomy is the procedure of choice in most cases. However, if limited patchy necrosis and gangrene is evident, partial resections may also be effective. During hospitalization, parenteral nutrition is implemented, monitoring is necessary and intravenous antibiotics may be needed.*

## References


1. BYRNE JJ, CAHILL JM. Acute gastric dilation. *Am J Surg* 1961, 101:301–309
2. FRANKEN EA Jr, FOX M, SMITH JA, SMITH WL. Acute gastric dilatation in neglected children. *AJR Am J Roentgenol* 1978, 130:297–299
3. KYRIAKIDIS AV, KONTIZAS IS, ATHANASIOU K, BOVIATSI P, ALEXANDRIS H. Acute gastric dilatation as a rare cause of acute abdomen concerning patients with Alzheimer's disease. *Hellen J Surg* 2015, 87:276–278
4. SAHOO MR, KUMAR AT, JAISWAL S, BHUJABAL SN. Acute dilatation, ischemia, and necrosis of stomach without perforation. *Case Rep Surg* 2013, 2013:984594
5. TURAN M, SEN M, CANBAY E, KARADAYI K, YILDIZ E. Gastric necrosis and perforation caused by acute gastric dilatation: Report of a case. *Surg Today* 2003, 33:302–304



*Corresponding author:*

A. Kostouros, 51 Christou Maltezou street, 180 30 Methana, Greece
e-mail: kostouros.a@yahoo.com