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REVIEW

Acute Gastric Dilation With Necrosis: Case Report And Literature Review

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Abstract

Acute gastric dilation is a rare pathology, which is met at diverse ages and has a high mortality rate if left untreated. This occurs due to several reasons, both due to mechanical obstruction of the stomach, and gastroparesis and due to extragastric causes such as eating disorders. Early placement of an oro- or nasogastric tube may help prevent serious complications such as ischemia and perforation of the gastric wall. The article presents a clinical case of a teenager with a severe degree of gastric dilation due to simultaneous overeating of bulky food and soft carbonated drinks. Due to the inefficacy of conservative measures and the development of severe ischemic alterations of the stomach, surgical intervention was performed.

Keywords: Acute gastric dilation, Gastric necrosis, Complicated gastric dilation, Overeating, Gastric decompression.

Introduction

Acute gastric dilation (AGD) is a rare urgent condition, which is met in diverse cohorts of patients from neonates to elderly people and requires early treatment to avoid life-threatening gastric rupture [1, 2]. AGD, even before gastric rupture, could develop signs of shock and lead to fatal outcomes with a mortality rate of 50-100% [3].

Etiologic factors of AGD also vary significantly and could depend not only on age but even on eating habits in different cultures [4, 5]. These features make AGD a difficult clinical diagnosis, which requires not only definite knowledge but a lot of experience. At the same time, a lot of clinical questions remain unsolved and require further discussion.

History and etymology

In medical publications, reports of AGD are more often represented by descriptions of individual clinical cases, which have been more common since the early years of the 20th century [6, 7]. Most likely, Karl von Rokitansky first drew attention to this pathology in 1842, when he described the syndrome of the superior mesenteric artery [8]. The superiority of the french surgeon S. E. Duplay in describing AGD in 1833 [3, 9] should be considered incorrect since according to biographic materials [10, 11], Simon Emanuel Duplay was born in 1836. In 1908 W.B. Laffer in his article "Acute dilation of the stomach and anterior-mesenteric ileus" [12] demonstrated a broad range of synonyms of previously diagnosed gastric dilatation pointing to its possible etiology:

Stomach-derived names as acute gastric dilation, post-operative acute dilation of the stomach, and post-operative gastric paralysis;

Mesentery caused entities as gastro-mesenteric ileus, mesenteric intestinal obstruction, post-operative arterio-mesenteric intestinal obstruction, anterio-mesenteric ileus

Proximal intestine obstruction as a cause of AGD as acute duodenojejunal intestinal obstruction, duodenal ileus, and duodenal compression. Here we also may notice the surgical intervention as a detached cause of AGD as a postsurgical complication.

The frequencies of variants "acute gastric dilation'' and "acute gastric dilatation" are seemed equal and more popular now.

In 1859, the English physiologist Winston Brinton put forward an atonic theory of the occurrence of AGD [3]. One of the first mentions of AGD with a rupture in neonates appeared in 1963, in a description of a clinical case of artificial lung ventilation in a newborn with a distal tracheoesophageal fistula and esophageal atresia [13]. A little later, G. Leonidas [14] showed the relationship between AGD, gastric rupture, and mechanical ventilation of the lungs in several clinical cases. At the same time, spontaneous AGD should be considered as a separate entity and distinguished from iatrogenic gastric rupture.

Etiology and pathogenesis of AGD

Spontaneous AGD has specific etiopathogenesis and the dynamics of clinical presentation. According to the literature data, the following factors have a certain etiological role in the development of AGD:

1. Compulsive overeating (binge eating) is an eating disorder characterized by episodes of eating a large amount of food with a loss of control over oneself [15]. This disorder may be a manifestation of neurological developmental delay, mental illness, or simply distress, but does not always lead to AGD [16, 17];

2. Direct trauma to the stomach [18, 19];

3. Dilation due to gastric volvulus [20] with a congenital laxity of its suspending ligaments or its absence;

4. Dilation of the stomach and part of the duodenum associated with compression of the last superior mesenteric artery [6, 12];

5. Massive invasive operations on the upper gastrointestinal tract and pancreas, which lead to gastroparesis [6, 12];

6. General anesthesia [21];

7. Neuroinfections (bulbar and pseudobulbar palsies, botulism, bulbar poliomyelitis) [22];

8. Overeating in a short period large meal of gas-producing foods and its combination (carbonated drinks, radish, grape, apples, etc.), foods rich in fiber [23];

9. Overeating after religious fasting - food abstinence in Ramadan or before Orthodox Easter [4, 5];

10. Intramural gastric neuropathy associated with chronic conditions (diabetic patients, alcohol abuse, etc) [24, 25].

11. As a result of intestinal obstruction [26].

The pathogenesis of AGD, leading to a perforation of the stomach wall and systemic disturbances, remains poorly understood. True obstruction of the alimentary tract of the AGD usually does not occur. In most cases of AGD, the mechanism of its development includes a vicious circle of increase in the gastric volume, leading to its rupture. Initially, the expansion of the stomach with liquid contents and food masses brings to compression of the neighboring organs of the upper abdominal cavity. When the transverse duodenum is compressed by the stomach itself or by the superior mesenteric artery, bile and pancreatic juice cannot enter the distal gastrointestinal tract. This flow back into the stomach causes additional gastric dilation (Fig 1) [6, 25, 27].

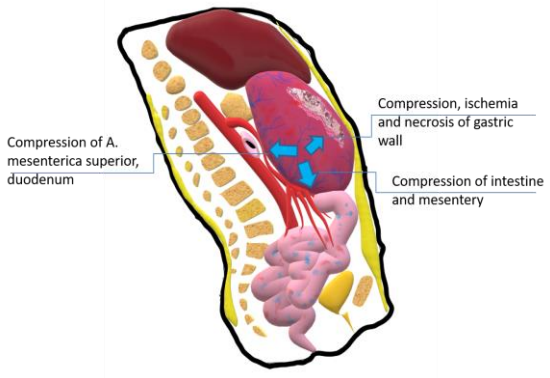

Figure 1: Vicious circle of AGD

At a certain stage, venous stasis occurs in the overstretched wall of the stomach, since malleable veins are easily compressed, and arterial blood flow is preserved for some time and decreases later with a possible complete cessation of blood flow through the clamped anastomoses or in areas free of them. Venous stasis, when intra-gastric pressure exceeds the gastric venous pressure (usually more than 20–30 cm of water column), promotes sequestration into the third space, which provokes the appearance of exudate in the peritoneal cavity and additional accumulation of fluid inside the stomach – thus, closing the vicious circle [25, 28].

With the expansion of the antrum of the stomach, gastrin secretion reflexively increases, which contributes to the secretion of hydrochloric acid in gastric juice. Hydrochloric acid, in turn, leads to subsequent damage of already ischemic, stretched, and thinned mucosa and the formation of erosions. This factor, along with altered intramural arterial blood flow in this area, causes its necrosis and subsequent perforation.

According to different authors [3, 25, 27], the mortality rate of patients with AGD is high even with the appropriate treatment. It is largely due to the rapid development of hypovolemic shock [16, 27]. Vomiting at the initial stages of the disease and local serum sequestration entails the significant loss of fluid and electrolytes, which leads to a decrease in circulating blood volume. Hypovolemic shock is also promoted by compression of the portal and inferior vena cava, which significantly reduces the venous return of blood to the heart. Also, from hemodynamic disorders, possible arrhythmias secondary to water-electrolyte disturbances and acid-base disbalance could be noted. Thus, AGD is one of the clearest examples of an intra-abdominal hypertension syndrome with a more rapid course, since the displacement of its center to

the upper floor of the abdominal cavity leads to direct compression of hemodynamically significant organs.

Clinical presentation – definite dynamics

In the described clinical cases [6, 11, 13] all authors point to a similar clinical picture, which has certain dynamics. The onset of the disease, as a rule, is accompanied by repeated vomiting, then, after a few hours, its frequency often decreases to the point of impossibility of vomiting. In the early stages of the disease, there is already a relative failure of the gag reflex, and the absence of vomiting in the later stages is associated with such factors as weakness of the wall of the distended stomach, intramural mediator imbalance - paresis, compression of the abdominal part of the esophagus by a full stomach like a one-way valve, electrolyte disturbances. The feeling of nausea by this time is also somewhat dulled. Electrolyte and fluid losses cause hypovolemia, which, alongside compression of inferior vena cava by increasing intra-abdominal pressure, decreases cardiac preload [23]. Without intravenous fluid resuscitation and correction of electrolyte balance, a patient develops signs of hypovolemic shock progressively. Some patients have low blood pressure, mottled skin, and different extent of impaired consciousness even before intra-abdominal hypertension. When intra-abdominal pressure reaches its possible limit, the ischemic and necrotic alteration of the stomach wall manifests with peritoneal irritation [33].

Management of AGD

Clinical presentation could be vague and may not allow diagnosing an AGD alone and the underlying condition early. Imaging an extremely extended stomach either by plain radiography, MRI, or CT scans is a keystone of the diagnosis [26]. MRI and CT have advantages in the further diagnostic search for an impaired gastric outlet (tumor, volvulus, intestinal obstruction, etc) [4, 9, 12, 20, 26], although an abdominal X-ray could be used as a screening method.

Blood biochemistry analysis can reveal abnormal glucose levels and diabetes as an underlying cause of gastroparesis [24, 25, 30]. Electrolyte disturbances could be an early sign of underlying gastroparesis or late signs of electrolyte loss. A complete blood count will not show anything specific, however, could indicate the extent of dehydration and possible necrotic complications.

According to a few studies [6, 24, 25, 27, 30], early active gastric emptying by aspiration through the nasogastric tube could be determinative and crucial in the resolution of the AGD vicious circle. Correction of the water-electrolyte balance is the other main part of non-operative management. The administration of parenteral proton pump inhibitors, prokinetics, antispasmodics, and antibiotics is almost always included but not standardized. The effectiveness of early conservative measures in absence of mechanical obstruction is shown in 50-70% [30].

Although there are reports of conservative treatment of AGD, the main articles present descriptions of clinical cases that required surgical treatment due to perforation of the gastric wall and intractable shock signs. According to literature data the surgical approach distinguishes significantly and depends on the surgical status. Some authors described primary repair with local debridement of the stomach wall and suturing [29], which sometimes was combined with a feeding jejunal tube [31]. In the other cases, partial stomach resection was necessary [29]. The reports about total or subtotal gastrectomy are rare [16, 29]. The esophagostomy and feeding jejunostomy [3, 29], as well as tube gastrostomy [32] for

decompression in cases of AGD, are also described.

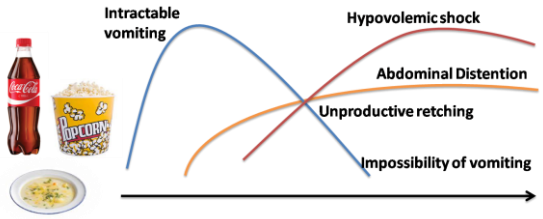

Figure 2. Dynamics of clinical presentation

Case report

Patient K., the the 13 years old girl, was admitted to the emergency department presenting a major complaint of a distended, tender abdomen. The patient reported that about 8 hours before she ate traditional Russian cold soup – "Okroshka", the usual components of which are bread kvas, fresh cucumbers or radish, dill, hard-boiled egg, and sour cream. After a while, she drank Coca-cola and had popcorn as a snack. Sometime later she had nausea followed by intractable vomiting and unproductive retching at admission. Her past medical history was uneventful. She refused constipation or contact with contagiously infected people. There were no known allergies and she did not receive any medications regularly. The dynamics of the patient's condition could be summarized in a diagram (Fig.2).

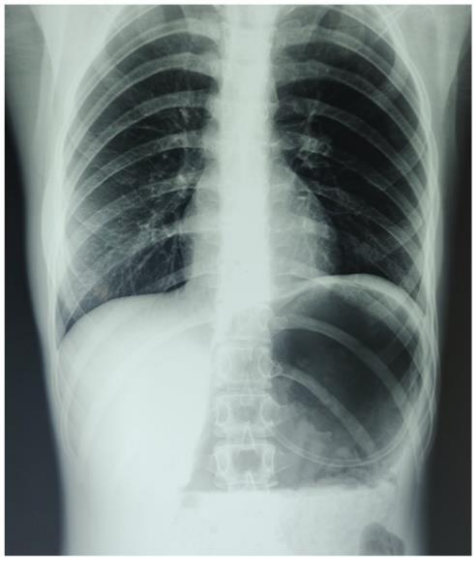

Figure 3. Abdominal radiograph

On examination her vital signs were stable, although she was slightly hypotonic (BP 94/72), tachycardic (HR 114), and tachypneic (RR 22). Generally, she appeared uncomfortable and fatigued. The abdomen was asymmetric, distended mostly in the epigastric and left flank area. Generalized tenderness was most prominent in the umbilical region. There were no signs of peritoneal irritation or guarding. Pertinent laboratory values showed leukocytosis – 17,000 and hypokalemia – 2,6 mmol/l. The bedside US was unremarkable due to a gas-filled stomach and intestine. The AP chest and abdomen radiographs revealed a significantly distended stomach with a level of fluid (Fig.3). CT scan of the abdomen confirmed the markedly distended stomach, fulfilled with heterogeneous masses (Fig. 4). No mechanical causes of obstruction were found. Immediate active aspiration through a nasogastric tube did not allow evacuating all of the containing. Considering conservative measures as not completely effective, after stabilization of hemodynamics, diagnostic laparoscopy was performed.

During the diagnostic laparoscopy, about one liter of dark cloudy yellowish effusion with fibrin and a hemorrhagic component was found in the peritoneal cavity - aspirated. Further exploration revealed a subserous rupture of the stomach along the greater curvature and the anterior surface of the stomach - the wall of the stomach from the antrum to the bottom, gray-dirty color - extremely thinned, in some places represented only by the visceral peritoneum, soldered with loose fibrinous overlays to the anterior abdominal wall. Taking into account the need for further thorough revision and the volume of the upcoming resection of the stomach, a conversion to the upper midline laparotomy was performed (Fig. 5-6). After assessing the

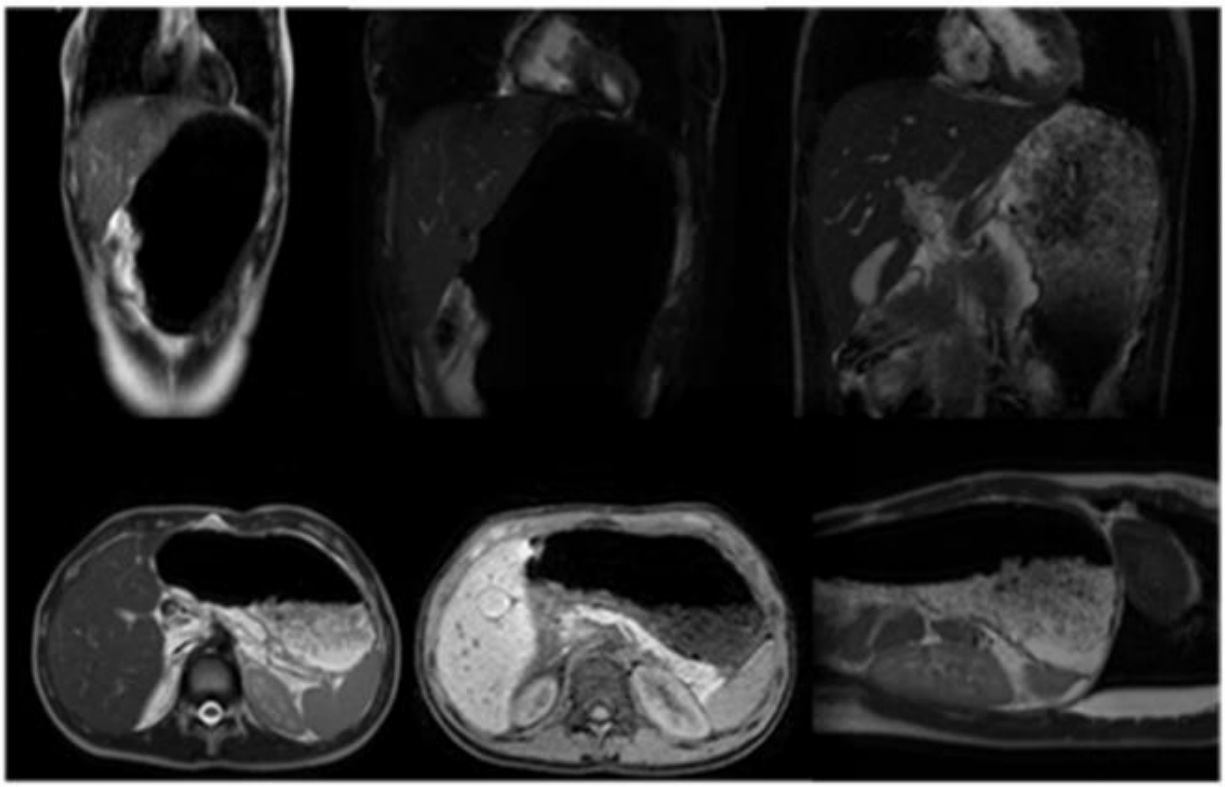

Figure 4. CT findings

viability of tissues, an atypical resection of the stomach was performed with the removal of necrotic tissues. The defect was sutured in two layers with a noninterrupted Resorba 3/0. Two drains were inserted - one in the pelvic cavity and the other in the left subdiaphragmatic space. Hemostasis was ensured. Postoperative diagnosis: Acute gastric dilatation with incomplete subserous rupture, diffuse peritonitis.

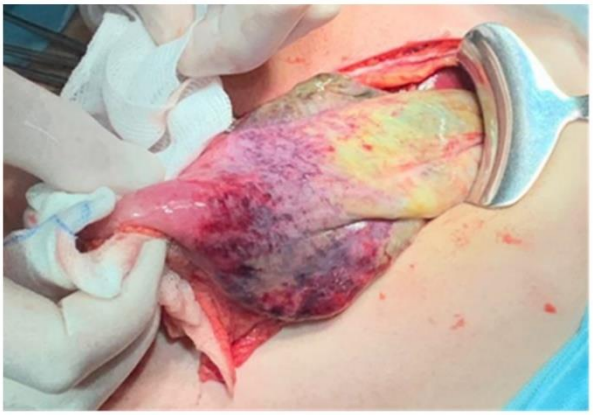

Figure 5. Laparotomy view

During the postoperative period, the girl was in the pediatric intensive care unit for 6 days, where antibacterial, antisecretory, infusion, hemostatic, and symptomatic therapy was carried out. Total parenteral nutrition was carried out until the 10th postoperative day, after which, with a gradual expansion, feeding with the Peptamen mixture was started. The child was discharged on the 22nd postoperative day. Currently, she is under the supervision of a gastroenterologist, receives

Figure 6. Removal of necrotic tissues

courses of antisecretory and enzyme replacement therapy, and follows an easily digestible diet.

Discussion

The rare incidence of AGD and heterogeneous etiologic factors leave the management of this condition not standardized. In the pediatric population there are two peaks in incidence in the neonatal period (mostly iatrogenic or due to congenital reasons) [13, 14] and adolescence (due to the most diverse causes such as overeating disorders, direct trauma, diabetes, etc.) [21, 24, 33]. No articles about spontaneous AGD in toddlers, preschoolers, or children of young school age, except due to gastric volvulus [20], were found.

Initial signs of AGD usually are vague and not specific [26]. Currently, there is no optimal algorithm of diagnosis for better management of AGD. That the first step to the correct diagnosis is obtaining the instrumental image of an extremely expanded stomach in a patient with the pertinent clinical presentation [4, 9, 12, 20, 26]. When the presence of AGD is undoubtful, the next step is to differentiate the cause by more accurate imaging studies and laboratory investigation. The possibility and necessity of upper endoscopy on the dilated stomach are discussible.

Simultaneously with the diagnostic search aggressive conservative treatment should be started [28, 30], as early gastric decompression decreases the rate of surgical interventions and mortality significantly. It is clear from every report that early diagnosis is a mainstay of successful treatment of AGD.

Indications for surgical exploration are not identified. At the early stage of assessment, it is extremely important to define the presence or absence of ischemic complications of AGD, as it is one of the most common reasons for surgery. Not many clinical signs and tests can directly point to the destructive process of the stomach wall and reflect its extent. Classic clinical and instrumental signs of gastric perforation in patients with AGD are late and can be vague in case of shock. In presence of gastric distention, properly obtained and properly studied in the laboratory, pentraxin, 3 and ischemia-modified albumin biomarkers could be possible markers of developing ischemia, which can help to distinguish between uncomplicated AGD and complicated [34-40]. However, these acute phase proteins are not specific and their significance in AGD requires further investigations. Therefore, there are a few groups of indications for exploratory laparoscopy or laparotomy in AGD [4, 9, 12, 20, 24, 25, 26, 30]:

1. AGD + unclear cause + clinical deterioration;

2. AGD + persistent shock (after stabilization);

3. AGD + ischemic complications.

The follow-up of the patients after AGD also remains not determined and the rate of recurrence stays unclear. In the presented case of AGD, this aspect is highly important as the patient will come under the control of medical institutions for adults soon, where necessary precautions should be undertaken.

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Writing the article: **SAK**
Critical revision of the article: **IVG, MA**
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