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Acute Gastric Dilatation: "A Gluttony or a Crises in a Pandemic" An Update with review of Current literature

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ABSTRACT

Gastrectasia, Ectasia, or Acute gastric dilatation is a surgical emergency. Historically it was associated with binge eating, branded as gluttony, wolf eating and today goes by a refined name BED or binge eating disorder. Myriad in spectrum presentation the triggers continue to be the same a history of psychiatric disorders like anorexia nervosa, bulimia, and psychogenic polyphagia (1), (2) or following surgical procedure like fundoplication, pyloric stenosis. A masquerader in abdomen emergencies with abdominal pain and vomiting in the initial stages to shock & multiorgan collapse in later presentation. If not picked early the consequences disastrous. The mortality and morbidity from gastric dilatation is reported at 80% to 100% (1), (2), (21).

Keywords: Acute Gastric Dilatation, Malnutrition, Covid19, Perforation

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Introduction

The craving of man for food is an old and intimate one. It can be traced back to the womb and follows birth with breast feeding. It was death due to binge eating that led E.Revilloid in 1885 to demonstrate in cadavers and by Key Abergs in 1929 on the autopsy following death in individuals that the capacity and threshold for a full stomach following a meal was four litres or about four quarts. Volumes greater than four liters equivalent to intragastric pressures of 20mmg impair intramural blood flow leading to focal necrosis of the inner mucosal lining with tears that if prolonged to ischemia necrosis and serosal perforation [14], [23], [38]. Individuals with pathologic eating disorders like bulimia nervosa, and psychogenic polyphagia are the high risk group and have larger baseline gastric volume due to repeated wolf eating habits to produce larger baseline volume capacity of the stomach by gastric hypertrophy. A combination of binge eating in a patient with compromised gastric ischemia perfusion serves a trigger for Acute Gastric dilatation. Acute massive gastric dilation is an extreme full spectrum gastric dilatation. Gastric rupture is seen with an intra gastric pressure of 120 to 150 mm Hg [24], [39].

Case review

A 55yr old male in Intensive care was referred for Abdominal pain, vomiting and progressive distension abdomen. An initial abdominal Xray showed a dilated stomach that over the period of a few hours as seen on second Xray revealed an impending perforation of the stomach. A cranio-caudal length extension of the stomach measured 43 cm. There was no evidence for perforation.

Laboratory investigations revealed the following results: White blood cell count: 13.100/uL; mean platelet volume: 10.5 fL; platelet count: 320 × 10^3/uL; hemoglobin: 10.1 g/dL; hematocrit: 35.2%; serum proteins: 4.7 g/dL; serum albumin: 3 g/dL; serum globulin: 2.8 g/dL; aspartate aminotransferase: 25 IU/L; alanine amino-transferase: 23 IU/l; creatinine: 0.9 mg/dl; serum sodium: 135 mEq/L; serum potassium: 4 mEq/L; serum chloride: 101 mEq/L; C-reactive protein: 10 mg/L; serum lactate dehydrogenase: 100 IU/L; serum creatine kinase: 4,086 IU/L; serum glucose: 101 mg/dL; arterial blood pH: 7.37 nmol/L; $PaCO_2$: 4.93 kPa; pO_2: 6 kPa; HCO_3: 19.5 mmol/L; base excess: 8.8 mEq/L.

A timely insertion of a nasogastric tube decom-pressed the stomach ultimately averting a crisis of ischemia necrotic perforation. The initial drain was over 500 mL of gastric content and a repeat abdominal Xray hours later revealed resolution of gastric dilatation.

Fig.1

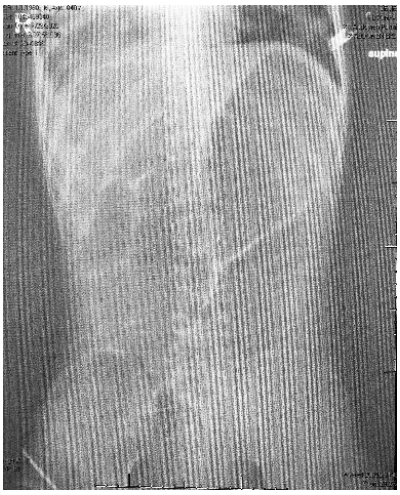

Fig.2 Casualty

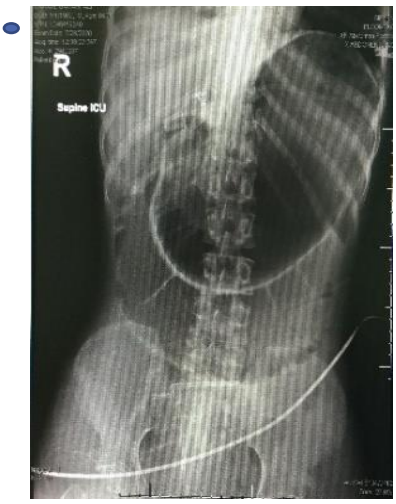

Fig. 3 ICU

Discussion

Acute Gastric dilatation is a surgical emergency. Acute gastric dilatation was first described by SE Duplay in 1833 [1]. The common triad of abdominal pain, attempted non bilious vomiting with progressive abdominal distension must alert the treating physician to the diagnosis. A bedside positive succussion splash test with an evident distended abdomen is diagnostic of Acute Gastric Dilatation. The predisposed age group are the elderly with atherosclerosis and females [13]. Those in the young age report a history of recreational drug abuse and finally those with psychiatric illness like Anorexia nervosa or Bulimia, psychogenic polyphagia.

A history of Abdominal pain but repeated attempts and inability to vomit is a feature of a distended fundus occluding of the gastroesophageal junction seen in the initial stages of acute gastric dilatation. The reason cited for this inability to vomit is the angulation of the fundus of the stomach with oesophagus against the right crus of the diaphragm thereby producing a one way valve effect [17]. Another bedside sign is the inability to pass an NGT [19]. A history of consumption of poisons and corrosive substances, even bicarbonate ingestion need to be considered when evaluating a patient with Acute gastric dilatation [16]. A neglected abdominal distension over hours or days causes necrosis [22] with perforation that is localized with guarding that becomes generalized with the onset of overt peritonitis. The development of shock and organ failure are terminal events. The site of gastric perforation is generally the greater curvature and fundus, the lesser curvature less likely to perforate [3], [13]. Anterior perforations of the gastric wall are more common than posterior ones. The site of perforation is dependent on the underlying disease state like peptic ulcer location, atherosclerosis, uremia, and the use of corticosteroids and immunosuppressive drugs [27], [30]. The underlying anatomical variations of arterial arcade to the stomach also predispose to ischemic and necrosis [26], [29]. In young patients with anorexia nervosa there is a history of starvation followed by binge eating [8].The atonic and atrophic gastric mucosal lining with underlying muscular atrophy coupled with slow gastric emptying [39] tears up this mucosal barrier after binge eating thereby setting the stage for Acute gastric dilatation. Russells sign is an important bedside clue seen in young anorexic individuals. A massive gastric dilatation is a distention of the stomach that extends from the diaphragm to the pelvis.

In 1859, Brinton introduced the atonic theory [13]. The stomach undergoes atony and muscular atrophy during a period of starvation, so that wolf eating or binge of food consumption in patients with eating disorders, is a predisposer to Acute gastric dilatation. The Mechanical theory proposed by Von Rokitansky in 1861, states that there is a vascular compression of the third segment of the duodenum, between superior mesenteric artery, aorta, and vertebral column that results in acute gastric dilatation [12]. Congenital mechanical causes include a diaphragmatic hernia that causes intrathoracic herniation of the stomach predisposing it to gastric ischemia, infarct, necrosis and dilatation. A Congenital elongated hepatogastric ligaments can theoretically predispose a previously dilated stomach to volvulus. Intrinsic motor pacemaker theory [40] states that it is the inhibition of the pacemaker cells of Cajal by segmental ileus that trigger Acute gastric dilation. The iatrogenic theory states that history of Invasive procedures like surgery [28] or embolization [31], [32], or systemic vasopressin infusion are precursors to Acute gastric dilatation. A history of Radiofrequency ablation for ventricular tachycardia is reported to cause Acute gastric dilatation the most plausible explanation for the latter being the RFA induced interference of the vagus nerve [20]. Likewise Infective causes like regional hepatic Porto thrombophlebitis also predispose to acute gastric dilatation [13], [39]. Systemic diseases like diabetes induce gastroparesis by autonomic neuropathy and dysfunction of pacemaker cells of Cajal [3], [4], [8],

[15], [18]. Old age, dehydration and shock predispose to venous thrombosis especially if the gastric arterial blood arcade is compromised by diseases like atherosclerosis [13]. In the pediatric age group aerophagia a functional acquired gastrointestinal disorder causes gastric ischemia by abdominal distension, this inappropriate swallowing of air resulting in relaxation of the upper oesophageal leading to gastric distention [13]. The prevalence of aerophagia in children is estimated to be eight to ten percent [7].

The common site of perforation in Acute gastric dilatation is the gastric curvature [13] the lesser curvature and pyloric regions of the stomach tend to be spared [11]. Once perforation sets in the abdomen passes through stages of reaction to the inflammatory or chemical infiltrate followed by stage of peritoneal lining exudation and finally from a localized to generalized peritonitis [33]. This Irritation of the peritoneal cavity can momentarily produce a profound vagal response before manifesting with features of true septic shock [23]. This massively distended stomach is even reported to produce an abdominal compartment syndrome with compromise of the blood flow in the abdominal aorta and leading to absent pulses in the lower limbs [6] This Ischemia is presumably caused by venous insufficiency by the massively dilated gastric content [9], [10], [25].

An abdominal Xray Imaging is diagnostic of gastric dilatation. While the presence of free gas along the diaphragm is suggestive of perforation, their presence along the lesser sac are indicative of perforation of the posterior gastric wall [34] [37]. It is a relatively rare presentation of acute gastric dilatation.

In haemodynamically stable patients with a history of previous surgery an Abdominal CT scan is a more accurate method of identifying the primary cause that led to acute gastric dilation [35], [36].Occasionally Acute gastric dilatation can lead to gastric emphysema and emphysematous gastritis. Gastric emphysema results from a mucosal breach followed by forceful entry of air between the gastric layers also known as non infectious gastric emphysema [41],[42]. The most commonly involved microorganisms are streptococci, Clostridium perfringens, E coli, Pseudomonas aeruginosa [43]. In contrast to non infectious gastric emphysema, the patients with infectious gastric emphysema present with fever, chills, and abdominal pain [43],[5]. Abdominal Compartment syndrome is another complication of AGD, where the sudden increase in pressure within a closed anatomic space threatens the viability of surrounding tissue and organs by the compression of Inferior vena cava.

Management

The most important step in the initial management of this patient is to promptly decompress the stomach with a nasogastric tube [NGT]. This will halt venous congestion and potential ischemic areas and prevent aspiration [14]. The administration of fluids for resuscitation, base line investigations blood cultures, IV antibiotics, and abdominal Xrays. Delayed and missed perforations haunt back with severe dehydration, a hypochloraemic metabolic alkalosis, signs of sepsis and single organ or multi-organ. Endoscopic aspiration of thick coffee ground aspirates, will decompress the over inflated stomach in a setup where the facility is available. Once peritonitis sets in, the surgical options for treatment depend on the duration of ischemia and extent of necrosis the longer the delay the more likely an extensive procedure like subtotal or a total gastrectomy will be required. This translates to extended duration in ICU with older age people with systemic diseases therefore an increase the morbidity and mortality.

For localized necrotic areas a Cellen jones pedicled omental patch closure or a Grahams patch traditional or modified would suffice. The operative procedure can be performed as single stage procedure for haemodynamically stable patients or as a two staged procedure if haemodynamically unstable damage control mode. The options include a hemigastrectomy, a subtotal or total gastrectomy with either

oesphagojejunostomy reconstruction, or if a two staged procedure, an initial esophagostomy with insertion of a feeding jejunostomy [1], [16] with subsequent reconstruction once stable. There is a role for successful nonoperative therapy and has been advocated by some surgeons [4], [11].

Conclusion

Gluttony or not, the key is an open mind to the symptoms an NG gastric decompression goes a long way in everting a disaster.

Declaration of Consent

Appropriate patient consent forms were obtained.

Conflict of Interest

Non.

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