Case Reports in Gastroenterology

Case Rep Gastroenterol 2024;18:439–448
DOI: 10.1159/000541516
Received: February 9, 2024
Accepted: August 16, 2024
Published online: October 15, 2024

Case Series

Acute Gastric Dilatation: A Retrospective Case Series from a Single Institution

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Keywords

Acute gastric dilatation · Ischemia · Necrosis · Gastric outlet obstruction · Nasogastric tube decompression

Abstract

Introduction: Acute gastric dilatation (AGD) is a massive distension of the stomach caused by the accumulation of gas, gastric secretions, or food material. AGD is a radiological diagnosis with no clear etiopathogenesis and is often misdiagnosed owing to a lack of clear diagnostic criteria and physician awareness. ***Case Presentation:*** In this case series, we describe the clinical presentations and outcomes of 4 patients with AGD. Three (75%) of the patients were female, and one (25%) was male. The patients' ages ranged from 53 to 84 years, with an average age of 73.5 years. Abdominal pain, nausea, and vomiting were the most frequently reported complaints. Two (50%) patients had cancer, one (25%) had an acquired duodenal stenosis, and the fourth patient experienced an ileus. ***Conclusion:*** AGD is a surgical emergency with a 50–100% mortality rate; thus, prompt diagnosis and management are crucial. Herein, we describe a case series of AGDs that were diagnosed and managed at our institution. We aim to raise awareness about this fatal yet underrecognized clinical entity.

Introduction

The French surgeon Simon-Emmanuel Duplay first described acute gastric dilatation (AGD) in 1833 as massive distension of the stomach as seen on imaging [1–5]. Since then, a few cases have also been reported in the literature, the majority of which were diagnosed after

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complications. AGD occurs due to the accumulation of gas, gastric secretions, or food material in the stomach, along with loss of gastric wall tension [1, 2, 6, 7]. AGD is a rare event with no implicit definition; therefore, it is often misdiagnosed due to a lack of physician awareness [1, 8, 9]. Various pathophysiologic theories have been proposed, but none have been confirmed [8, 10]. In anorexia nervosa, it is postulated that erratic eating habits lead to gastric dysmotility, thus predisposing patients to AGD [11].

AGD causes and risk factors include binge/purging behavior in anorexia nervosa, polyphagia, psychogenetic polyphagia, pathological aerophagia, gastroparesis, electrolyte imbalances, abdominal surgery, and general anesthesia [1, 7, 9–15]. Mechanical causes of AGD include gastric volvulus, trauma, acquired pyloric stenosis, and malignancy. AGD can lead to nonspecific symptoms such as severe diffuse abdominal pain, abdominal distension, nausea, and vomiting [11, 16]. Prompt diagnosis is crucial to rule out gastric ischemia, necrosis, obstruction, perforation, and initiation of appropriate treatment [11]. In addition to a detailed history and focused physical examination, a computerized tomography (CT) scan of the abdomen and pelvis can lead to a timely diagnosis of AGD. An abdominal X-ray can help rule out gastric perforation, and an upper endoscopy can detect an obstruction or early signs of gastric ischemia [11]. Gastric scintigraphy study helps diagnose gastroparesis in patients with advanced diabetes mellitus and other eating disorders [11].

Nasogastric decompression and fluid resuscitation are the first-line interventions for most of the patients [7, 9–11, 17]. Laxatives and promotility agents help with symptomatic management. Surgery is employed in patients with gastric necrosis, malignant distension, hemodynamic instability, or signs of gastric perforation [10–12]. AGD has a high mortality of up to 75% and can progress to gastric emphysema, gangrene, shock, or perforation [18, 19]. Thus, early diagnosis and prompt surgical treatment are essential to prevent mortality. We present a case series of AGDs that were diagnosed and treated at our hospital in New Jersey. We aim to raise awareness about this fatal yet underrecognized clinical entity. Further research is required to study the etiopathogenesis of AGD and to create guidelines for its diagnosis and management. The CARE checklist has been completed and attached as an online supplementary file (for all online suppl. material, see https://doi.org/10.1159/000541516).

Case Presentations

Case 1

An 84-year-old male with a medical history of hypertension, gastroesophageal reflux disease, and recent admission for acute gastric distention presented to the emergency department (ED) with worsening abdominal distention and pain. Esophagogastroduodenoscopy (EGD) with biopsy during that admission revealed gastric dilation, gastritis, and acute duodenitis with a duodenal stricture. A stent was placed in the duodenum, and the gastric distention improved with nasogastric tube (NGT) decompression. The biopsies were negative for malignancy and *Helicobacter pylori* infection. Once the patient tolerated a regular diet, he was discharged to a rehabilitation facility for medical optimization.

On readmission, the patient was alert, awake, and in mild distress due to pain. He had a well-healed scar in the right lower quadrant and a grossly distended abdomen that was tender on palpation. The patient had hypoactive bowel sounds throughout but without peritoneal signs. The remainder of the physical examination was unremarkable. In the ED, the patient was afebrile and saturated well on room air. Vital signs were significant for elevated blood pressure but without tachycardia. Triage blood tests were significant for hyperglycemia, hypokalemia (3.1 mEq/L), and hypomagnesemia (1.2 mEq/L). CT scan of the abdomen and pelvis showed a massive AGD with food content and wall thickening in the first part of the

duodenum (shown in Fig. 1). An NGT was inserted for gastric decompression, and intravenous fluids were started for hydration. The NGT drained brownish output on intermittent suction. Abdominal X-ray and upper gastrointestinal series revealed a high-grade duodenal obstruction with proximal dilatation of the stomach. The obstruction was high-grade but incomplete.

The patient underwent a second EGD with dilatation and duodenal stent placement. Post-procedure, the patient did not tolerate diet advancement and the NGT was reinserted. The hospital course was complicated by elevated liver function tests and jaundice, likely secondary to ampullary obstruction. A repeat EGD showed duodenal stricture to be malignant-appearing, stented lumen appeared narrowed and ampulla could not be visualized. Multiple biopsies returned as acute duodenitis with no evidence of malignancy. As the patient's condition continued to worsen, his family decided on palliative surgery. He underwent an exploratory laparotomy with gastrojejunostomy, cholecystectomy for gallbladder decompression, and stone extraction. A tumor infiltrating the pylorus, first and second portion of the duodenum, with infiltration of the distal common bile duct and cystic duct was noted. Omental and mesenteric nodules were found on further inspection, and the biopsies showed metastatic carcinoma on frozen section. An intraoperative cholangiogram revealed cystic duct obstruction, and interventional radiology was consulted for percutaneous transhepatic cholangiography. After the procedure, an upper gastrointestinal series showed patent gastrojejunal anastomoses without leaks or obstructions. The patient was successfully tapered off of the total parenteral nutrition and started on a mechanical soft diet, which was advanced as tolerated. The patient was discharged to a nursing home for further care.

Case 2

A 53-year-old female with a history of hypertension, diabetes mellitus, and iron deficiency anemia was referred to the ED from an outpatient surgical center due to an abnormal finding during an upper endoscopy. The EGD revealed a gastric mass with obstruction of the gastric outlet. The patient reported a 1-month history of epigastric pain, nausea, and vomiting. The abdominal pain was dull in nature, constant, and non-radiating. The vomitus was non-bloody, non-bilious, and usually post-prandial. The patient also reported decreased appetite and a twenty-pound weight loss over 2 months. She denied having fever, chills, acid reflux, dysphagia, melena, or recent changes in stool caliber.

In the ED, the patient's vital signs were within the normal ranges, and she appeared in no obvious distress. On physical examination, the patient's abdomen was soft and tender to palpation in the epigastrium without guarding, rigidity, or rebound tenderness. The remainder of the physical examination was unremarkable. Admission blood tests showed leukopenia (white blood cell count, $4.2 \times 10^3/mm^3$) and iron deficiency anemia. Lactic acid level, lipase level, and urinalysis were unremarkable. A contrast-enhanced CT scan of the abdomen and pelvis revealed gastric distension with an apparent irregular circumferential wall and probable narrowing involving the pylorus/proximal duodenum (shown in Fig. 2). A few prominent and mildly enlarged adjacent lymph nodes were also noted.

The patient was kept nil per os (NPO), and an NGT was inserted and placed on intermittent suctioning for gastric decompression. She was maintained on dextrose 5% in 0.45% normal saline fluids and later transitioned to peripheral parenteral nutrition pending further workup. The gastroenterologist shared that the EGD biopsy results were significant for moderately to poorly differentiated gastric adenocarcinoma. The patient underwent an endoscopic ultrasound with duodenal stent placement and was able to tolerate oral intake after the procedure. She had a port-a-cath placed and completed the first cycle of pre-operative FLOT (fluorouracil, leucovorin, oxaliplatin, and docetaxel) chemotherapy while inpatient. The patient completed the FLOT regimen outpatient and underwent distal

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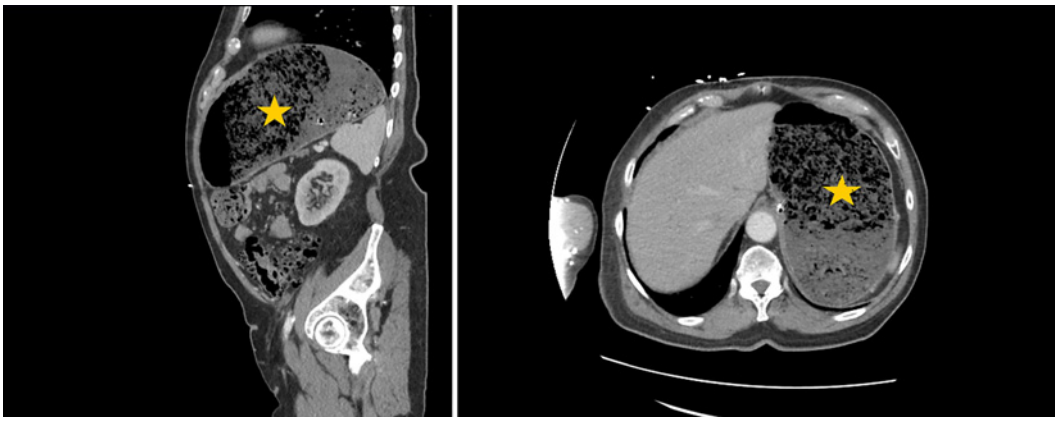

Fig. 1. A contrast-enhanced CT scan of the abdomen and pelvis (sagittal and axial planes) showing significant distension of the stomach containing food debris.

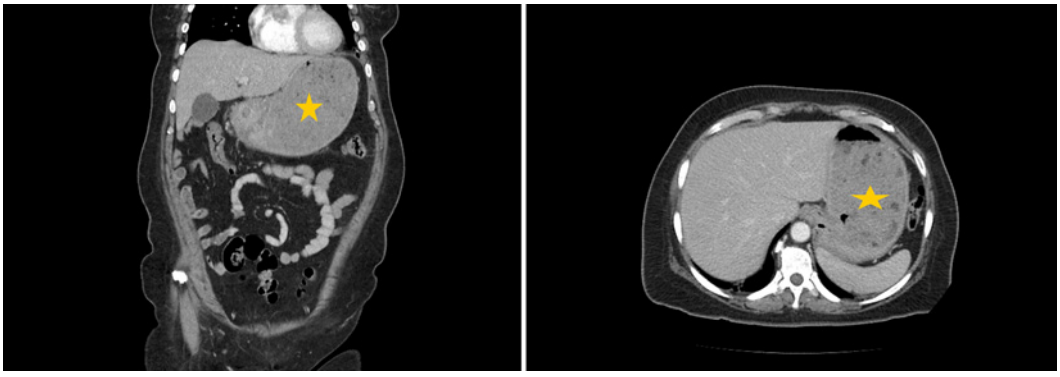

Fig. 2. A contrast-enhanced CT scan of the abdomen and pelvis (coronal and axial planes) showing gastric distension with probable luminal narrowing involving the pylorus/proximal duodenum.

gastrectomy with Roux-en-Y reconstruction and omentectomy. She has since completed the postoperative FLOT regimen and continues to follow up with the oncology clinic for continued care.

Case 3

An 81-year-old female with a medical history of chronic obstructive pulmonary disease, urge incontinence, and Barrett's esophagus without dysplasia presented to the ED for evaluation of acute-onset abdominal pain. Surgical history was significant for lysis of adhesions surgeries due to small bowel obstructions in 2013, 2015, and 2017. The abdominal pain was described as diffuse in nature, constant, and associated with multiple bouts of brown emesis. The patient's last bowel movement occurred a few days prior to the ED visit, but she passed flatus. She denied having fever, chills, acid reflux, dysphagia, diarrhea, abdominal trauma, melena, hematochezia, dysuria, or gross hematuria.

In the ED, the patient's vital signs were normal, but she appeared uncomfortable due to pain. A focused physical examination revealed a soft and distended abdomen that was diffusely tender to palpation. No peritoneal signs or palpable organomegaly were appreciated on examination. The patient had hypoactive bowel sounds throughout. Blood tests were notable for hypercalcemia (10.9 mg/dL) and elevated serum bicarbonate (34 mEq/L). Due to

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concern for bowel obstruction, a contrast-enhanced CT of the abdomen and pelvis was performed, which showed a markedly distended stomach without duodenal distension, with some mild inflammation around the antroduodenal junction suggesting possible peptic ulcer disease (shown in Fig. 3). The CT scan also showed mildly hyperemic small bowel loops, mainly in the pelvis, suggestive of enteritis.

An NGT was placed on low intermittent suction for gastric decompression. The patient also received symptomatic treatment with intravenous fluids, antiemetics, acetaminophen, pantoprazole, and electrolytes. An EGD ruled out peptic ulcer disease, gastric outlet obstruction, and duodenitis. It was, however, notable for mild esophagitis likely from NGT trauma. A repeat CT scan and abdominal X-ray showed a resolution of the ileus, and the patient was discharged with plans for outpatient follow-up with the surgical clinic.

Case 4

A 76-year-old female with a medical history of morbid obesity, dyslipidemia, atrial fibrillation on digoxin/dabigatran, and with a pacemaker presented to the ED with abdominal pain, nausea, and vomiting. The abdominal pain was described as sharp in nature, central, and non-radiating. The pain improved with pain medications and worsened with oral intake. She denied having fever, chills, dysphagia, diarrhea, constipation, or melena.

The patient was in her usual state of health until the year prior, when she was brought to the ED for evaluation of acute-onset epigastric pain. At that time, a CT scan of the abdomen and pelvis showed marked distention of the stomach and proximal duodenum. The patient underwent NGT decompression, but remained symptomatic. An EGD showed one non-bleeding gastric ulcer on the lesser curvature of the stomach, likely from NGT trauma. It also revealed moderate stenosis of the third portion of the duodenum. The mucosa within the stenosis was ulcerated and erythematous. A 22-mm WallFlex™ stent was placed with 2 clips in the duodenum at the stenotic site. The histopathology of the duodenum was significant for intraepithelial lymphocytes and focal degenerative changes. The patient was eventually discharged after that admission.

At this visit, her hemodynamics were stable, and a focused physical examination was significant for epigastric tenderness without signs of peritonism. Triage blood work was significant for hypokalemia (3.2 mEq/L) and hyponatremia (132 mEq/L). The rest of the chemistry was within normal ranges. CT scan of the abdomen and pelvis demonstrated a distal duodenal stent with worsened distention of the stomach and proximal duodenum (shown in Fig. 4). An EGD revealed an acquired malignant-appearing intrinsic severe stenosis in the third portion of the duodenum.

A previously placed metal stent was seen in the second portion of the duodenum. Ingrowth had occurred in the stent, obstructing its internal lumen. This was stented with a 22 mm × 12 cm WallFlex™ stent under fluoroscopic guidance. Pathology showed nonspecific mild acute and chronic duodenitis. It was negative for parasites, dysplastic, or neoplastic processes. After discharge, the patient experienced a 40-pound weight loss and remained symptomatic. She was eventually referred to the surgical service where she underwent a diagnostic laparoscopy, extensive lysis of adhesions, and laparoscopic gastrojejunostomy. It has been 2 years since the patient's surgery, and she remains asymptomatic.

Discussion

In this case series, we share our experience with diagnosing and managing AGD to avoid further complications. AGD is a rare and potentially life-threatening condition characterized by rapid stomach distension due to gas and/or fluid accumulation. AGD is an

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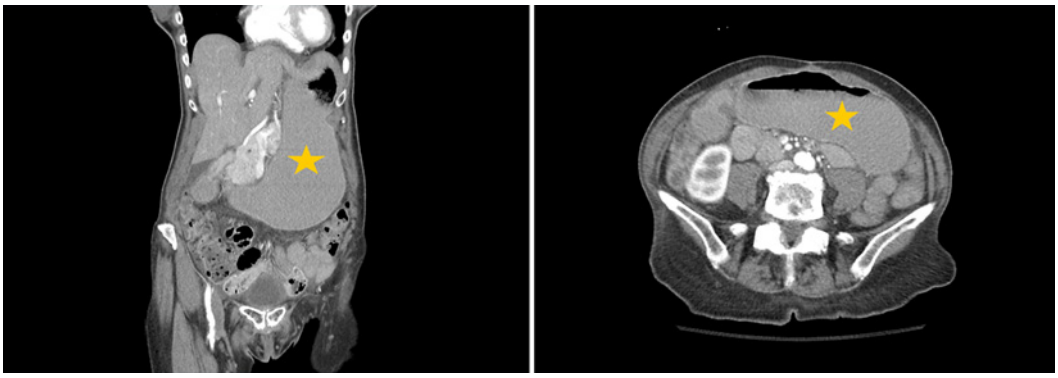

Fig. 3. A contrast-enhanced CT scan of the abdomen and pelvis (coronal and axial views) showing marked gastric distension with some mild inflammation around the antroduodenal junction.

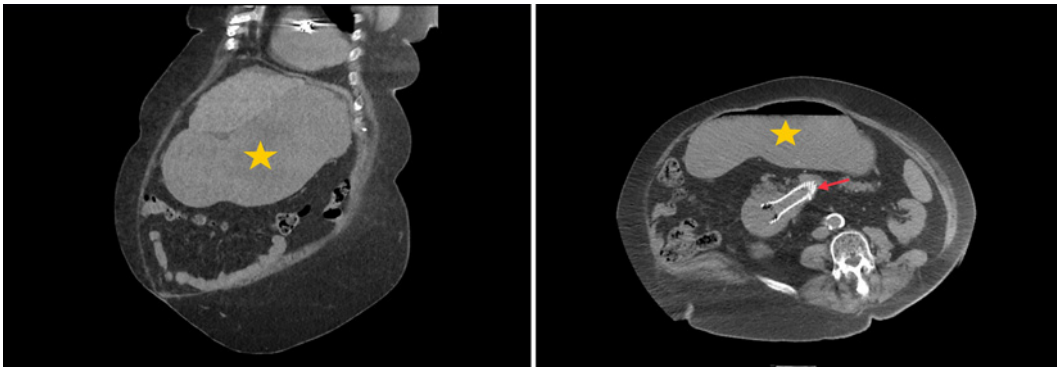

Fig. 4. A CT scan of the abdomen and pelvis (coronal and axial views) showing significant distension of the stomach and proximal duodenum. A duodenal stent (red arrow) can be seen in the horizontal portion of the duodenum.

alarming radiological finding, and an early diagnosis and intervention are key to improving survival rates [4, 5, 11, 13]. Although its true etiopathogenesis remains unclear, AGD is associated with eating disorders, gastric volvulus, gastroparesis, electrolyte imbalances, polyphagia, psychogenetic polyphagia, pathological aerophagia, abdominal surgery, trauma, and pyloric stenosis [1, 9–11, 13–15]. AGD has also been described as a rare postoperative complication after esophageal or abdominal surgeries [16, 20]. Lau et al. [16] shared a case of acute massive gastric dilatation as a rare complication of open fundoplication in a pediatric patient with a history of congenital diaphragmatic hernia. In our case series, AGD causes include cancer, gastric ileus, and acquired duodenal stenosis (Table 1).

AGD has a varied clinical spectrum due to a large pool of possible etiologies and risk factors. Patients commonly present with acute-onset abdominal pain, progressive abdominal distension, bloating, post-prandial fullness, nausea, vomiting, dysphagia, odynophagia, skin mottling, or a succession splash [1, 4, 7, 9–11, 13–15, 21]. Vomiting is a significant symptom and is seen in more than 90% of AGD cases [5]. Patients with an AGD due to gastric volvulus may be unable to vomit due to occlusion of the gastroesophageal junction [4, 5]. Despite the stomach's rich vascular supply, an AGD can lead to venous congestion of the stomach wall, which increases intragastric pressure [1, 4, 5, 15].

Table 1. Summary of patient demographics, presentations, clinical diagnoses, and interventions

Patient	Age, years	Gender	Presenting symptoms	Etiology	Intervention	Outcome
Case 1	84	Male	Abdominal pain, abdominal distension, nausea, vomiting, jaundice	Metastatic cholangiocarcinoma	Palliative surgery (ex-lap with gastrojejunostomy), percutaneous transhepatic cholangiography	Hospice care
Case 2	53	Female	Epigastric pain, nausea, vomiting, low appetite, weight loss	Gastric adenocarcinoma with gastric outlet obstruction	Gastroduodenal stenting	ECOG 1
					Distal gastrectomy with reconstruction and omentectomy	
					FLOT chemotherapy	Currently on chemotherapy
Case 3	81	Female	Abdominal pain, nausea, vomiting	Gastric ileus	NGT decompression	Clinically improved and discharged
Case 4	76	Female	Abdominal pain, nausea, vomiting, low appetite, weight loss	Acquired duodenal stenosis	Stent-in-stent placement Laparoscopic gastrojejunostomy	Clinically improved and discharged

Intragastric pressures in the range of 14–30 cm H_2O are associated with gastric ischemia and a grave prognosis [4, 15, 19]. In some cases, AGD with ischemia may evolve into gastric perforation, emphysematous gastritis, or abdominal compartment syndrome. Other complications of AGD include arrhythmias, aortic occlusion, superior mesenteric artery syndrome, and mesenteric ischemia [8, 15, 19, 21–23]. Park and Nho [6] described a unique case of aortic compression and abdominal compartment syndrome as a consequence of severe gastric dilatation. Enzmann et al. [24] described an extremely rare case of fatal aortic occlusion due to compression from self-induced AGD. The case involved a young woman with bulimia disorder who ingested 15 L of water to present with sufficient weight to her psychiatrist.

Early diagnosis of AGD is crucial to prevent serious sequelae such as gastric ischemia or necrosis. Abdominal radiographs are ordered as part of the initial work-up, which reveals massive gastric dilation with a fluid level [4, 5]. Additional imaging can further exclude the possibility of gastric perforation or hemorrhage in which immediate treatment would be required [11]. CT scan of the abdomen and pelvis is another tool that can be employed to diagnose AGD and assess the extent of gastric involvement [3, 11]. Endoscopy has been shown to help decipher the etiology of obstruction and assess the mucosa for ischemic changes [11]. It can function as both diagnostic and therapeutic, suctioning the trapped gases while assessing for any pathological changes [4, 19].

The management of AGD varies greatly, from conservative management to invasive surgery. NGT decompression is the first-line modality in patients who are hemodynamically stable [1, 4, 6, 21]. After decompression, endoscopy is typically performed both to visualize the mucosa, as well as to aspirate any remaining gastric contents [19]. An upper endoscopy can be used for gastric detorsion and decompression if an AGD is recurrent or due to an acute gastric volvulus [4]. Surgeons at Kings Mill Hospital in the United Kingdom employed a

venting percutaneous endoscopic gastrostomy tube with a jejunal extension to manage a case of recurrent AGD in a male patient with Prader-Willi syndrome [12]. In most cases, further management is dictated by the cause of the AGD and the patient's clinical status. In our case series, for example, cases 1, 2, and 4 required surgeries, while case 3 was resolved with an NGT decompression (Table 1).

Patients with signs of gastric necrosis, rupture, abdominal compartment syndrome, or shock require partial or complete gastrectomy with esophagojejunostomy and tube feeding [2, 4]. Huang and Tang [23] reported an interesting case of superior mesenteric artery syndrome with AGD in an adolescent patient. An emergency operation revealed gastric necrosis, warranting proximal subtotal gastrectomy [23]. Similarly, Islam et al. [4] described a case of AGD with ischemia and gastric perforation that required emergent total gastrectomy after NGT decompression failed. The patient had a medical history of Crohn's disease but was non-compliant with his medications. He died within 24 h of the surgery due to disseminated intravascular coagulation.

Conclusion

AGD is a profound distension of the stomach that results from the accumulation of gas, gastric secretions, or food material. The etiology of AGD remains unclear, and it is frequently either underdiagnosed or misdiagnosed due to the absence of explicit diagnostic criteria. In this case series, we describe the clinical presentations and outcomes of 4 patients with AGD diagnosed and managed at our institution. Three of the cases were due to mechanical factors (cancer and duodenal stenosis), whereas one was due to gastric ileus. Three cases required endoscopic and surgical intervention, whereas the gastric ileus case was resolved with NGT decompression. In summary, we aim to raise awareness about this fatal yet underrecognized clinical entity.

Acknowledgments

The authors thank Dr. Idris Ali, Dr. Mohamed Elagami, Dr. Gabriel Melki, and Ms. Jessica Escobar for participating in these patients' care and assisting in the writing of this manuscript.

Statement of Ethics

This retrospective review of patient data did not require ethical approval in accordance with local/national guidelines. Written informed consent was obtained from each patient for publication of the details of their medical case and any accompanying images.

Conflict of Interest Statement

No conflicts of interest to declare.

Funding Sources

No funding was obtained for the writing or submission of this case series.

Author Contributions

L.B. and R.B. conceptualized the idea of this case series. S.V., T.W., and R.Y. wrote some sections of the case series. Y.C. and W.B. edited and proofread the final manuscript.

Data Availability Statement

All the data used to support the findings of this case series are available as part of the article and references. Further inquiries can be directed to the corresponding author.

References

1 Shaikh DH, Jyala A, Mehershahi S, Sinha C, Chilimuri S. Acute gastric dilatation: a cause for concern. Case Rep Gastroenterol. 2021;15(1):171–7. https://doi.org/10.1159/000512401
2 Shaikh OH, Reddy N, Kumbhar US, Suresh C. Acute massive gastric dilatation as a result of closed- loop obstruction of stomach: an unusual and rare phenomenon. BMJ Case Rep. 2020;13(9):e235943. https://doi.org/10.1136/bcr-2020-235943
3 Jano F, Behr C, Almajali F, Moran V. Acute gastric dilatation complicated by necrosis and perforation following a binge eating episode. Cureus. 2022;14(11):e31727. https://doi.org/10.7759/cureus.31727
4 Islam S, Shah A, Naraynsingh V, Harnarayan P. Acute gastric dilatation with ischemia and perforation requiring emergency total gastrectomy: a case of suspected abdominal compartment syndrome. Cureus. 2023;15(7):e41265. https://doi.org/10.7759/cureus.41265
5 Sahoo MR, Kumar AT, Jaiswal S, Bhujabal SN. Acute dilatation, ischemia, and necrosis of stomach without perforation. Case Rep Surg. 2013;2013:984594. https://doi.org/10.1155/2013/984594
6 Park KB, Nho WY. Abdominal compartment syndrome caused by severe acute gastric distension in a patient with COVID-19. Medicine. 2023;102(28):e34326. https://doi.org/10.1097/MD.0000000000034326
7 Yorke J, Gyamfi FE, Awoonor-williams R, Osei-Akoto E, Acheampong E, Acheampong EN, et al. Acute gastric necrosis in a teenager. Case Rep Surg. 2020;2020:8882179. https://doi.org/10.1155/2020/8882179
8 Tuero C, Docio G, Artajona A, Arin B, Cires M, Monton S. Acute massive gastric distention with emphysematous gastritis: a case report and literature review. Cir Cir. 2022;90(6):838–41. https://doi.org/10.24875/CIRU.21000614
9 Azevedo F, Canhoto C, Costa B, Carvalho H. Pneumoperitoneum from acute gastric dilation and perforation. BMJ Case Rep. 2019;12(11):e232392. https://doi.org/10.1136/bcr-2019-232392
10 Ashouri M, Vezvaei P, Kazemeini A, Sherafati A, Mirfazaelian H. Acute gastric dilation following trauma: a case report. Adv J Emerg Med. 2020;4:e13. https://doi.org/10.22114/ajem.v0i0.192
11 Pitre T, Mah J, Vertes J, Tugwell B. Acute gastric dilatation in a patient with severe anorexia nervosa: a case report. J Med Case Rep. 2021;15:61–4. https://doi.org/10.1186/s13256-020-02575-7
12 Mohammed AMA, Dennis RJ. Use of a venting PEG tube in the management of recurrent acute gastric dilatation associated with Prader-Willi syndrome. J Surg Case Rep. 2016;2016:rjv174–2. https://doi.org/10.1093/jscr/rjv174
13 Chandra N, Karisma B, Sampada K, Shrivastava B, Shakya N, et al. Gastric volvulus: an uncommon and life threatening cause of acute gastric dilatation in a young male: a case report. Clin Case Rep. 2022;10(11):e6537. https://doi.org/10.1002/ccr3.6537
14 Nam K, Shin HD, Shin JE. Acute gastric dilatation and ischemia associated with portal vein gas caused by binge eating. Korean J Intern Med. 2019;34(1):231–2. https://doi.org/10.3904/kjim.2017.050
15 Loi C, Chen K. Total gastric necrosis following massive gastric dilatation due to superior mesenteric artery syndrome. Asian J Surg. 2023;46(6):2363–4. https://doi.org/10.1016/j.asjsur.2022.12.008
16 Lau SE, Boam T, Parsons S, Motiwale S. Acute massive gastric dilatation: a rare, forgotten complication of fundoplication. BMJ Case Rep. 2020;13(5):e232479. https://doi.org/10.1136/bcr-2019-232479
17 Shaikh O, Chilaka S, Reddy N, Vijayakumar C, Kumbhar U. Acute massive gastric dilatation and gastric perforation as a result of closed-loop obstruction of the stomach. Cureus. 2021;13(2):e13365. https://doi.org/10.7759/cureus.13365
18 Ito H, Ogawa R. Extreme acute gastric dilation due to anorexia nervosa. Rev Gastroenterol Mex. 2022;87(3):382–3. https://doi.org/10.1016/j.rgmxen.2022.07.004
19 Núñez J, García-angarita FJ, Puerta A, Muñoz P, Sanjuanbenito A. Sleeve gastrectomy for idiopathic acute gastric dilatation with transmural necrosis. Ann R Coll Surg Engl. 2021;103(9):275–7. https://doi.org/10.1308/rcsann.2020.7121

Case Rep Gastroenterol 2024;18:439–448
DOI: 10.1159/000541516

20 Sohrabi F, Dimaggio F, Alasadi A, Mukherjee D. Massive gastric dilatation leading to acute respiratory distress 1 year after a Nissen fundoplication. BMJ Case Rep. 2018:2018:bcr2018225927. https://doi.org/10.1136/bcr-2018-225927
21 Tominaga Y, Hirayama I, Yano T, Kurihara K, Ishii M. Localized skin mottling associated with acute gastric dilatation. Clin Case Rep. 2023;11:e6856–3. https://doi.org/10.1002/ccr3.6856
22 Gurushankari B, Sureshkumar S, Anandhi A, Rajesh BS, Naik D, Saurabh K, et al. Acute gastric dilatation leading to ischemic necrosis: a rare complication following sigmoid volvulus. Sultan Qaboos Univ Med J. 2021;21(3): 504–6. https://doi.org/10.18295/squmj.4.2021.026
23 Huang Z, Li C, Tang G. Superior mesenteric artery syndrome with acute gastric dilatation caused by binge eating in an adolescent. Korean J Intern Med. 2022;38(4):572–3. https://doi.org/10.3904/kjim.2022.344
24 Enzmann F, Guggenbichler S. Fatal aortic occlusion due to compression from self induced acute gastric dilatation. Eur J Vasc Endovasc Surg. 2019;58(2):281. https://doi.org/10.1016/j.ejvs.2019.02.025