Multimodality imaging of adult gastric emergencies: A pictorial review

Abhijit Sunnapwar, Vijayanadh Ojili, Rashmi Katre, Hardik Shah, Arpit Nagar

Department of Body Imaging, University of Texas Health Science Center, San Antonio, Texas, *Department of Body Imaging, Ohio State University Wexner Medical Center, Ohio, USA

Correspondence: Dr. Abhijit Sunnapwar, Department of Body Imaging, University of Texas Health Science Center, 7703 Floyd Curl Drive, San Antonio, Texas ‑ 78229, USA. E‑mail: sunnapwar@uthscsa.edu

Abstract

Acute gastric emergencies require urgent surgical or nonsurgical intervention because they are associated with high morbidity and mortality. Imaging plays an important role in diagnosis since the clinical symptoms are often nonspecific and radiologist may be the first one to suggest a diagnosis as the imaging findings are often characteristic. The purpose of this article is to provide a comprehensive review of multimodality imaging (plain radiograph, fluoroscopy, and computed tomography) of various life threatening gastric emergencies.

Key words: CT; emergencies; fluoroscopy; gastric

Introduction

Emergent gastric pathologic conditions can be catastrophic because they can cause perforation leading to spillage of gastric contents into the peritoneal cavity with associated high morbidity and mortality. Radiologists must be familiar with their imaging findings as the clinical presentation is varied. Gastric emergencies result from different pathophysiologic mechanisms such as distension, ischemia, perforation, obstruction, and infection/inflammation. Although computed tomography (CT) is the mainstay of diagnosis, conventional imaging modalities that include plain films and barium studies combined with fluoroscopy may provide clues to the diagnosis. In this article, we discuss gastric anatomy and clinical features along with a comprehensive review of multimodality imaging features of nontraumatic, noniatrogenic emergent conditions of the stomach.

Gastric Anatomy

Stomach can be divided into five segments namely the cardia, fundus, body, antrum, and pylorus [Figure 1A]. The cardia surrounds the gastroesophageal (GE) junction. Gastric fundus is the part of the stomach above a line drawn tangential to the GE junction. It is responsible for the "stomach bubble" seen on upright plain film and is the most dependent portion of the stomach in supine position. The body lies between the fundus and antrum and is the most distensible part of the stomach limited by the greater curvature, anteriorly and to the left, and lesser curvature posteriorly and to the right. The antrum is the thickest and most peristaltic part of the stomach. Incisura angularis is the transition point between the body and antrum and is seen as an indentation along the lesser curvature on fluoroscopic studies [Figure 1]. It is the most common site for gastric perforation.

Cite this article as: Sunnapwar A, Ojili V, Katre R, Shah H, Nagar A. Multimodality imaging of adult gastric emergencies: A pictorial review. Indian J Radiol Imaging 2017;27:13‑22.
ulcers. The pylorus is the gateway of stomach to the duodenum and contains the pyloric sphincter. The pylorus lies anteroinferior to the GE junction, is the narrowest part of the stomach, and is most commonly involved in gastric outlet obstruction (GOO).

The stomach is a highly distensible structure with maximum holding capacity as high as 1.5 liters. On contrast-enhanced CT, the wall of the stomach has a trilaminar appearance owing to the presence of low attenuation submucosa sandwiched between enhancing mucosa and high attenuation muscularis propria plus serosa. This trilaminar appearance is also termed mural stratification. In an adequately distended stomach, the normal upper limit for wall thickness of the gastric body is 5 mm and that of antrum is 12 mm [Figure 1B]. An underdistended stomach not only leads to false positive appearance of thickening of gastric wall but may also obscure some pathologies.

**Role of Imaging**

When a patient presents to the emergency department (ED) with epigastric pain, abdominal radiograph is done as part of the initial work-up and can provide important clues such as free intraperitoneal air or acute gastric distension. Multidetector computed tomography (MDCT) is invaluable in evaluation of the stomach and surrounding structures with excellent spatial resolution. It also helps in the evaluation of vascular anatomy and associated vascular pathologies such as arteriovenous malformation and varices. On table oral contrast, if feasible, can achieve gastric distension and can be helpful in evaluation of gastric perforation. Water is an excellent negative oral contrast and does not interfere with evaluation of mucosal enhancement. Ideally, 750 ml of water should be administered before 15 minutes and 250 ml just before scanning. CT is limited by suboptimal mucosal detail as well as nondynamic evaluation, which can be achieved with fluoroscopic imaging. Upper gastrointestinal (GI) fluoroscopy with water-soluble contrast is very useful in suspected postoperative leaks and fistulas and evaluation of gastric bands. Gastric emergencies are summarized in Table 1.

**Gastric perforation**

Gastric perforation is a surgical emergency with high mortality rates ranging 10–40%. Lesser curvature is the most common site in cases of peptic ulcer as well as in acute gastric dilatation or necrosis. Extraluminal air with predominant upper abdomen distribution around the stomach and liver is the most common (97%) CT finding, however, it may not be detected in cases of very small or contained perforation. Gastric wall defect and/or ulcer is the second most common CT feature (84%) followed by extraluminal oral contrast (0–27%). Other CT features include wall thickening, adjacent fat stranding, and fluid.

**Gastric perforation from peptic ulcer disease**

Peptic ulcer disease (PUD) is the most common cause of nontraumatic gastric perforation. Ulcers along the anterior wall and curvatures perforate into the peritoneal cavity, whereas those along the posterior wall may lead to a contained perforation in lesser sac [Figure 2]. The most common presentation is severe upper abdominal pain. Signs

---

**Table 1: Imaging findings in adult gastric emergencies**

| Emergent conditions       | Imaging features                                      |
|---------------------------|------------------------------------------------------|
| Perforation               | Intraperitoneal free fluid or oral contrast material leakage. |
| Peptic ulcer disease      | Wall defect-ulcer, wall thickening, fat stranding     |
| Malignancy                | Nodular, eccentric or mass like wall thickening, lymphadenopathy, omental cake formation and distant metastasis. |
| Gastric band erosion      | Contrast material surrounding the band, intraluminal band, subphrenic abscess |
| Acute gastric dilatation  | Massively distended stomach with air fluid level      |
| Obstruction               | Dilated stomach                                      |
| Malignancy                | Nodular, eccentric or mass like wall thickening; lymphadenopathy, omental cake formation and distant metastasis. |
| Gastric Volvulus          | Greater curvature superior to lesser curvature in OAV, antrum superior to GE junction in MAV, wall thickening, diaphragmatic hernia. |
| Obstructed diaphragmatic hernia | Paracardiac mass with air fluid level, herniated stomach |
| Bouveret syndrome         | Dilated stomach with gall stone in the region of pylorus or duodenum, pneumobilia, gall bladder wall thickening. |
| Slipped gastric band      | Abnormal horizontal position of band with increased angle of φ > 58°, eccentric dilated pouch more than 4 cm. |
| Bezozar                   | Mottled density material within distended stomach.    |
| Foreign body              | Foreign body in the region of stomach                |
| Infection/Inflammation    | Wall thickening and edema, intramural gas, venous gas |
| Emphysematous gastritis (EG) | Wall thickening and edema, intramural gas, venous gas |
| Acute gastric necrosis    | Same as in EG                                        |
| Hemorrhage                |                                                     |
| Spontaneous gastric hematoma | Heterogeneous hyperdense or mass like thickening of stomach wall on CT |

---

**Figure 1 (A and B):** Normal gastric anatomy: Upper GI fluoroscopy image (A) showing the normal gastric anatomy including air filled fundus, body, incisura angularis (arrow), antrum, and pyloric canal. Axial CT image (B) showing normal wall thickness (<5 mm) of the gastric body.
of peritonitis including rigidity and guarding, and rebound tenderness may also be present depending on the location, extent, and duration of perforation. CT features include focal gastric wall thickening, ulcer defect, extraluminal air bubbles in contact with stomach or free intraperitoneal air, free fluid, adjacent fat stranding, oral contrast leakage, and wall discontinuity.\[10\] Free air around the liver and stomach and in the lesser sac are reliable signs of gastroduodenal perforation [Figure 2].\[11\]

**Perforated gastric malignancy**

Necrotic or ulcerated malignancies are the second most common cause of gastric perforation after peptic ulcers.\[12\] Adenocarcinoma, lymphoma, and gastrointestinal stromal tumor (GIST) can lead to gastric perforation. Perforation in gastric adenocarcinoma is rare and seen in advanced stages but may also occur in early stages due to deep ulceration.\[13\] The risk factors associated with spontaneous rupture or perforation of GIST include an exophytic mass with areas of necrosis or cystic degeneration and rapid growth on follow-up imaging.\[13\] Hollow viscous perforations associated with lymphoma are more common in small bowel than in stomach.\[14\]

CT findings may include the features of gastric perforation, as discussed above, in combination with features of malignancy including nodular, focal, eccentric, or mass-like wall thickening and loss of trilaminar appearance due to replacement of submucosa with tumor cells [Figure 3]. Signs of secondary spread including lymphadenopathy, omental cake formation, and distant metastasis may also be detected on CT.

**Post-bariatric surgery perforation**

Laparoscopic roux-en-Y gastric bypass and laparoscopic adjustable gastric banding (LAGB) are the two most widely performed bariatric surgical procedures.\[15\] LAGB is a popular procedure involving placement of adjustable silicone gastric band 2 cm distal to the GE junction with formation of a small proximal pouch with capacity 15 mL.\[15\] The band diameter can be adjusted through a subcutaneous port in anterior abdominal wall.

Perforations associated with gastric banding can be seen soon after surgery or can be delayed for a few weeks. Early postsurgical gastric perforation is rare and is seen in 0.1–0.8% of cases.\[16\] Clinical presentation is extremely variable ranging from fever, abdominal pain, to signs of sepsis. Extraluminal air and contrast material and/or abscess may be seen on CT. Delayed perforation is seen at least after 3 weeks following the surgery and may be seen even after several months.\[16\] It usually results from gastric band erosion and is reported in up to 3% of the cases.\[16\] It may be a result of continuous pressure of band against the gastric wall, inflammatory foreign body reaction, infection, or use of nonsteroidal anti-inflammatory drugs.\[15,16\] The clinical features include hematemesis, weight gain, and diffuse abdominal tenderness. Fluoroscopy with water soluble contrast revealing contrast material surrounding the band is a definite sign of intragastric band erosion [Figure 4A and B].\[16\] Other signs associated with band erosion are an open band and a change in band position.\[16\] CT findings include intraluminal position of the band, contrast material surrounding the band, and subphrenic abscess [Figure 4C and D].

Although laparoscopic roux-en-Y gastric bypass is considered a gold standard among surgical options for bariatric surgeries, laparoscopic sleeve gastrectomy (LSG) is also gaining popularity as it is technically less demanding with lower surgical risks.\[17\] Staple site leak is one of the most severe complications of LSG with an incidence of 0–7%.\[18\] Fluoroscopy with water-soluble contrast may reveal intraperitoneal leakage of contrast at gastrectomy site [Figure 5]. If there are surgical drains, it is prudent to evaluate them for contrast opacification at fluoroscopy.
Acute gastric dilatation (AGD) is a rare occurrence compared to other gastric pathologies and was first described by Duplay in 1833.\(^{[19]}\) It is most common in postoperative patients; other causes include eating disorders such as anorexia or bulimia nervosa, psychogenic polyphagia, trauma, diabetes mellitus, or gastric dysmotility in critically ill patients.\(^{[9,20]}\) Delay in treatment can result in gastric necrosis, perforation, abdominal compartment syndrome, cardiorespiratory failure, and severe sepsis.

Gastric perforation following AGD occurs due to ischemic necrosis secondary to overdistension. The stomach can distend to approximately 4 liters before it perforates.\(^{[20]}\) Stomach has a rich blood supply and is relatively resistant to perforation, however, intragastric pressure >20 cm of H\(_2\)O can impair venous drainage subsequently leading to ischemia, necrosis, followed by perforation.\(^{[20]}\) It is more commonly seen in women (F:M = 2:1) and occurs along the lesser curvature in 63% of the cases.\(^{[9]}\) Emesis is the most common symptom (90%) followed by abdominal pain and distension.\(^{[9,20]}\) Delay in treatment can result in gastric necrosis, perforation, abdominal compartment syndrome, cardiorespiratory failure, and severe sepsis.

Gastric obstruction

Malignancy

With the advent of advanced antiulcer therapy in the form of H\(_2\) blockers and proton pump inhibitors, malignancy is now the most common cause of gastric obstruction.\(^{[21]}\) Adenocarcinoma is the most common malignancy of the stomach and may present with an annular, constricting pyloric mass resulting in GOO.\(^{[4]}\) Arterial phase imaging with negative oral contrast increases its conspicuity on CT, which may reveal focal, irregular nodular, or mass-like thickening of the wall with heterogeneous enhancement.\(^{[7]}\) Primary gastric lymphoma is less likely to cause GOO than adenocarcinoma, as it is considered a “soft” tumor.\(^{[4]}\) Apart from primary gastric tumors, tumors arising from surrounding structures including the duodenum, pancreas, and biliary tree may also cause GOO.\(^{[3]}\)

Gastric volvulus

Gastric volvulus is defined as abnormal rotation of the stomach along its long or short axis leading to GOO. It is an emergency condition as delay in treatment can result in gastric ischemia and perforation. The predisposing factors are congenital or acquired diaphragmatic hernia, gastric neoplasm, and adhesions. Borchardt’s triad consisting of sudden onset abdominal pain, intractable retching, and inability to pass a nasogastric tube is classic for gastric volvulus.\(^{[22]}\)

Organoaxial volvulus (OAV) and mesentericoaxial volvulus (MAV) are two types of gastric volvulus depending on the axis of rotation.\(^{[23]}\) OAV is more common that MAV, whereas some cases have features of both.\(^{[24]}\) OAV involves rotation of stomach along its long axis with greater curvature lying superiorly and to the right in relation to the lesser curvature.\(^{[22]}\) The fundus is displaced posterosuperiorly whereas the antrum lies in an anterosuperior position.\(^{[21]}\) Paraesophageal hernia
and Bochdalek hernia are major predisposing factors for OAV in adults and children, respectively.\cite{23,26} MAV has a lesser association with diaphragmatic hernia and is associated with increased risk of complications. It involves rotation of the stomach along short axis, so that the pylorus lies in a superior position than the GE junction with twisting of vascular supply of stomach [Figure 9].\cite{23}

Upper GI tract barium fluoroscopy may reveal features of GOO in a herniated stomach and abnormal axis of the stomach [Figures 8 and 9]. CT with coronal and sagittal reformation is helpful in diagnosing both types of volvulus and associated complications. Additional findings noted on CT are gastric wall thickening, perigastric fluid or fat stranding, and intraperitoneal air in cases of perforation [Figure 9].

**Obstructed Diaphragmatic hernia**

Diaphragmatic hernia can be congenital or acquired. Bochdalek and Morgagni hernia are the two main types of congenital hernia. Herniation of stomach is more common in Bochdalek hernia, whereas Morgagni hernia mainly contains part of the liver.\cite{27} Morgagni hernia involves the herniation of intra-abdominal contents into the chest through a defect between the sternal and costal attachments of the diaphragm (space of Larrey) situated anteromedially. Bochdalek hernia occurs in the left posterolateral chest and is due to the malformation of pleuroperitoneal membranes. While obstructed Bochdalek hernia containing stomach has been reported
in a few case series, only 3 cases of obstructed Morgagni hernia have been reported. In adults, paraesophageal hernia is a predisposing factor for gastric obstruction and strangulation. Imaging features include visualization of a paracardiac mass obscuring the diaphragm on chest radiograph [Figure 10]. Air-fluid level may also be seen in the lower chest. CT with coronal reformation and upper GI barium fluoroscopy show features of gastric outlet obstruction including esophageal and gastric dilatation, herniated stomach in the chest, and site of obstruction [Figure 10].

**Bouveret syndrome**
Gallstone ileus is a rare complication of cholelithiasis and an unusual etiology for GI obstruction. Less than 3% of eroding gallstones impact in the duodenum or pylorus resulting in gastric outlet obstruction, an entity first described by Leon Bouveret in 1896. It occurs most commonly in elderly women with chronic biliary diseases. The presentation is variable but most patients present with nausea, vomiting, and pain in the epigastric region. Plain radiograph may show gallstone in the region of stomach or duodenum with pneumobilia. Upper GI series may demonstrate dilated stomach with filling defect in the region of pylorus or duodenum. CT is the modality of choice demonstrating an ectopic gallstone in pylorus or duodenum causing GOO, pneumobilia, and gall bladder wall thickening [Figure 11]. Early diagnosis and treatment are of paramount importance as mortality may be as high as 30%. [33]

**Post-gastric banding obstruction**
Post-gastric banding complications including stomal stenosis and band slippage leading to acute obstruction. Acute stomal stenosis is the most common complication post LAGB and can occur due to stomal edema or obstruction by food particles. Patient may present with nausea, vomiting, and upper abdominal discomfort. Upper GI barium fluoroscopy reveals stomal narrowing with delayed passage of contrast [Figure 12]. Treatment involves port decompression to relieve stomal obstruction. Gastric band slippage is seen in 4–13% of patients and can also lead to stomal stenosis and obstruction. Recurrent vomiting and faulty surgical techniques are predisposing factors. It occurs due to upward herniation of stomach through the band and can be anterior or posterior. Anterior slippage is more common than posterior slippage. Patients may present with cessation of weight loss, reflux, and vomiting. Anterior band slippage with eccentric pouch formation leading to acute stomal obstruction may be associated with gastric volvulus, ischemia, or perforation. Emergent band decompression is required to relieve symptoms followed by band removal or repositioning. Plain radiograph and CT with coronal reformation reveal abnormal horizontal position of band with increased angle of phi >58 degrees (angle between longitudinal axis of gastric band and spinal column, normal range: 4–58 degrees) [Figure 13A and D]. Upper GI barium fluoroscopy and CT may reveal abnormal position of band with eccentric dilated pouch more than 4 cm [Figure 13B and C]. Fluoroscopy may show stomal narrowing with delayed passage of contrast.

**Bezoar and foreign bodies**
Bezoars are concretions of nondigestible ingested matter acting as foreign bodies. They are a potential cause of GOO. Trichobezoar and phytozeoars are composed of hair and vegetable matter, respectively. Trichobezoars are most commonly found in females and patients with psychiatric disorders. The risk factors for phytozeoar include gastric dysmotility including postoperative gastroparesis, pancreatic exocrine dysfunction, cystic fibrosis, and cyclosporine use. Bezoars can also extend into the small bowel causing obstruction. Mottled density within a distended stomach can be seen on plain radiograph and barium fluoroscopy [Figure 14]. CT depicts inhomogeneous, round, or ovoid mass containing areas of soft tissue density intermixed with gas and oral contrast material.
Ingested foreign bodies greater than 6 cm in size can also cause GOO. This condition is more commonly seen in children, patients with psychiatric illness, prisoners, and alcoholics. Prompt endoscopic removal is the treatment of choice in these cases due to the risk of perforation. Plain radiograph showing the foreign body in the region of stomach may be the only imaging study required for the diagnosis. However, CT may be useful in cases of radiolucent foreign bodies and to assess for complications including perforation [Figure 15].

Infection/inflammation

Emphysematous gastritis

Emphysematous gastritis (EG) is a life threatening condition caused by infection of the stomach with gas forming organisms. Mucosal disruption of the wall of stomach with subsequent superinfection with gas forming organisms is the basic pathophysiology. Common underlying conditions are corrosive ingestion, alcohol abuse, abdominal surgery. Diabetes mellitus and immunosuppression are predisposing factors. Clinical presentation may be dramatic and include acute sepsis and shock. Plain radiograph reveals gas bubbles outlining the stomach wall in a mottled distribution [Figure 16A]. CT imaging features include gastric wall thickening and edema, intramural gas, perigastric fat stranding, gas in gastric veins, and portal venous system [Figure 16B and C].

Gastric emphysema is a benign condition with intramural air and may be confused with EG. However, patient is asymptomatic with absence of gastric wall edema and inflammation. In addition, intramural gas in gastric emphysema is linear and parallel to the stomach wall on plain radiograph and CT. Predisposing factors include violent coughing, vomiting, and recent endoscopic procedure. Spontaneous resolution is the usual outcome.

Acute gastric necrosis

It occurs due to ischemic injury to the wall of stomach. Risk factors include acute gastric dilatation, gastric volvulus, systemic hypotension, celiac axis occlusion, disseminated thromboembolism, and vasculitis. Ischemic necrosis in AGD occurs due to venous insufficiency, as described previously. Imaging features are similar as seen in EG and include gastric wall thickening with intramural and portal venous gas [Figure 17]. However, there is absence of associated bacterial infection of stomach wall.
Hemorrhage

Spontaneous gastric hematoma

Anticoagulant therapy induced spontaneous intramural hematoma involving the GI tract is a rare complication with an incidence of 1/2500/year. The most common site is duodenum followed by jejunum and ileum, whereas intramural hematoma involving the esophagus and stomach are rare. Clinical presentation includes abdominal pain, intestinal obstruction, hematemesis, and melena. Spontaneous gastric hematoma can also result in GOO. Early diagnosis with aggressive correction of coagulation is required as there is high risk of hematoma rupture with hemorrhage and hypovolemic shock. Imaging features on CT include heterogeneous hyperdense or mass like thickening of stomach wall in a patient on anticoagulant therapy. Magnetic resonance imaging may be helpful in excluding underlying mass [Figure 18].
Conclusion

Gastric emergencies are being reported with increasing frequency due to advent and exponential rise of bariatric surgeries, although an untouched stomach also can develop complications. These complications are truly life threatening and prompt diagnosis is of paramount importance, with the radiologist playing a key role in it. Not all these emergencies are treated surgically, with some needing endoscopy or conservative approach. Radiologist should be familiar with these conditions in order to guide the emergency department physician.

Acknowledgement

Dr. Christine Menias for extensive help in preparation of this manuscript.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Rubesin SE, Furth EE, Levine MS. Gastritis from NSAIDS to Helicobacter pylori. Abdom Imaging 2005;30:142-59.
2. Pickhardt PJ, Asher DB. Wall thickening of the gastric antrum as a normal finding: Multidetector CT with cadaveric comparison. AJR Am J Roentgenol 2003;181:973-9.
3. Guniganti P, Bradenham CH, Raptis C, Menias CO, Mellnick VM. CT of Gastric Emergencies. Radiographics 2015;35:1909-21.
4. Horton KM, Fishman EK. Current role of CT in imaging of the stomach. Radiographics 2015;35:1909-21.
5. Bertleff MJ, Lange JF. Perforated peptic ulcer disease: A review of history and treatment. Dig Surg 2010;27:161-9.
6. Moller MH, Adamsen S, Wojdemann M, Moller AM. Perforated peptic ulcer: How to improve outcome? Scand J Gastroenterol 2009;44:15-22.
7. Svanes C. Trends in perforated peptic ulcer: Incidence, etiology, treatment, and prognosis. World J Surg 2000;24:277-83.
8. Thorsen K, Glomsaker TB, van Meer A, Soreide K, Soreide JA. Trends in diagnosis and surgical management of patients with perforated peptic ulcer. J Gastrointest Surg 2011;15:1329-35.
9. 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;33:302-4.
10. Lee D, Park MH, Shin BS, Jeon GS. Multidetector CT diagnosis of non-traumatic gastroduodenal perforation. J Med Imaging Radiat Oncol 2016;60:182-6.
11. Kim SH, Shin SS, Jeong YY, Hoo SH, Kim JW, Kang HK. Gastrointestinal tract perforation: MDCT findings according to the perforation sites. Korean J Radiol 2009;10:63-70.
12. Furukawa A, Sakoda M, Yamasaki M, Kono N, Tanaka T, Nitta N, et al. Gastrointestinal tract perforation: CT diagnosis of presence, site, and cause. Abdom Imaging 2005;30:524-34.
13. Kim SW, Kim HC, Yang DM. Perforated tumours in the gastrointestinal tract: CT findings and clinical implications. Br J Radiol 2012;85:1307-13.
14. Vaidya R, Habermann TM, Donohue JH, Ristow KM, Maurer MJ, Macon WR, et al. Bowel perforation in intestinal lymphoma: Incidence and clinical features. Ann Oncol 2013;24:2439-43.
15. Blachar A, Blank A, Gavert N, Metzer U, Fluser G, Abu-Abeid S. Laparoscopic adjustable gastric banding surgery for morbid obesity: Imaging of normal anatomic features and postoperative gastrointestinal complications. AJR Am J Roentgenol 2007;188:472-9.
16. Mehanna MJ, Birjawi G, Moukaddam HA, Khoury G, Hussein M, Al-Kutoubi A. Complications of adjustable gastric banding, a radiological pictorial review. AJR Am J Roentgenol 2006;186:522-34.
17. Park JY, Kim YJ. Laparoscopic gastric bypass vs sleeve gastrectomy in obese Korean patients. World J Gastroenterol 2015;21:12612-9.
18. Zanotti D, Elkalaawy M, Mohammadi B, Hashemi M, Jenkinson A, Adamo M. Gastro-cutaneous fistula 4 years after a fully resolved staple line leak in sleeve gastrectomy. J Surg Case Rep 2015;2015.
19. Todd SR, Marshall GT, Tyroch AH. Acute gastric dilatation revisited. Am Surg 2000;66:709-10.
20. Lunca S, Rikkers A, Stanescu A. Acute massive gastric dilatation: Severe ischemia and gastric necrosis without perforation. Rom J Gastroenterol 2005;14:279-83.
21. Shone DN, Nikoomonesh P, Smith-Meek MM, Bender JS. Malignancy is the most common cause of gastric outlet obstruction in the era of H2 blockers. Am J Gastroenterol 1995;90:1769-70.
22. Feldman MFL, Brandt LJ, Sleisenger and Fordtran’s gastrointestinal and liver disease: Pathophysiology/diagnosis/management. 9th ed. Philadelphia, PA: Saunders Elsevier; 2010.
23. Peterson CM, Anderson JS, Hara AK, Carenza JW, Menias CO. Volvulus of the gastrointestinal tract: Appearances at multimodality imaging. Radiographics 2009;29:1281-93.
24. Sonthalia N, Ray S, Kanha D, Saha A, Maitra S. Gastric volvulus through morgagni hernia: An easily overlooked emergency. J Emerg Med 2013;44:1092-6.
25. Kohn GP, Price RR, DeMeester SR, Zehetner J, Muensterer OJ, Awad Z, et al. Guidelines for the management of hiatal hernia. Surg Today 2003;33:409-28.
26. Godshall D, Mossallam U, Rosenbaum R. Gastric volvulus: Case report and review of the literature. J Emerg Med 1999;17(5):837-40.
27. Alamo L, Gudincher F, Meuli R. Imaging findings in fetal diaphragmatic abnormalities. Pediatr Radiol 2015;45(13):1887-900.
28. Gangopadhyay AN, Upadhyaya VD, Gupta DK, Sharma SP.
Obstructed Morgagni’s hernia. Indian J Pediatr 2007;74:1109-10.
29. Singh S, Wakhlu A, Pandey A, Kureel SN, Rawat JD. Delayed presentation of strangulated congenital diaphragmatic hernia: Learning from our experience. Hernia 2013;17:403-7.
30. Lukman MR, Sangar P, Sukumar N. Obstructed para-esophageal hernia in a nonagenarian treated by laparoscopic anterior gastropexy. The Medical journal of Malaysia. 2007;62 (1):83-4.
31. Zhao JC, Barrera E, Salabat M, Denham W, Leung D, Ujiki M. Endoscopic treatment for Bouveret syndrome. Surg Endosc 2013;27:655.
32. Brennan GB, Rosenberg RD, Arora S. Bouveret syndrome. Radiographics 2004;24:1171-5.
33. Malvaux P, Degolla R, De Saint-Hubert M, Farchakh E, Hauters P. Laparoscopic treatment of a gastric outlet obstruction caused by a gallstone (Bouveret’s syndrome). Surg Endosc 2002;16:1108-9.
34. Sonavane SK, Menias CO, Kantawala KP, Shanbhogue AK, Prasad SR, Eagon JC, et al. Laparoscopic adjustable gastric banding: What radiologists need to know. Radiographics 2012;32:1161-78.
35. Weiner R, Bockhorn H, Rosenthal R, Wagner D. A prospective randomized trial of different laparoscopic gastric banding techniques for morbid obesity. Surg Endosc 2001;15:63-8.
36. Gorter RR, Kneepkens CM, Mattens EC, Aronson DC, Heij HA. Management of trichobezoar: Case report and literature review. Pediatr Surg Int 2010;26:457-63.
37. Dellon ES, Morgan DR, Mohanty SP, Davis K, Aris RM. High incidence of gastric bezoars in cystic fibrosis patients after lung transplantation. Transplantation 2006;81:1141-6.
38. Ripolltes T, Garcia-Aguayo J, Martinez MJ, Gil P. Gastrointestinal bezoars: Sonographic and CT characteristics. AJR Am J Roentgenol 2001;177:65-9.
39. Velitchkov NG, Grigorov GI, Losanoff JE, Kjossev KT. Ingested foreign bodies of the gastrointestinal tract: Retrospective analysis of 452 cases. World J Surg. 1996;20:1001-5.
40. Grayson DE, Abbott RM, Levy AD, Sherman PM. Emphysematous infections of the abdomen and pelvis: A pictorial review. Radiographics 2002;22:543-61.
41. Kussin SZ, Henry C, Navarro C, Stenson W, Clain DJ. Gas within the wall of the stomach report of a case and review of the literature. Dig Dis Sci 1982;27:949-54.
42. Tang SJ, Daram SR, Wu R, Bhajee F. Pathogenesis, diagnosis, and management of gastric ischemia. Clin Gastroenterol Hepatol 2014;12:246-52.e1.
43. Herman J, Chavalitdhamrong D, Jensen DM, Cortina G, Manuyakorn A, Jutabha R. The significance of gastric and duodenal histological ischemia reported on endoscopic biopsy. Endoscopy 2011;43:365-8.
44. Veldt BJ, Haringsma J, Florijn KW, Kuipers EJ. Coumarin-induced intramural hematoma of the duodenum: Case report and review of the literature. Scand J Gastroenterol 2011;46:376-9.
45. Arain J, Al-Dabbagh A. Gastric outlet obstruction secondary to spontaneous intramural haematoma as a complication of warfarin treatment. J Surg Case Rep 2012;2012:13.