Acute gastric dilatation: a potentially life-threatening entity

Poster No.: C-1561
Congress: ECR 2012
Type: Educational Exhibit
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Keywords: Ischemia / Infarction, Dilatation, Acute, Education, CT, Conventional radiography, Gastrointestinal tract, Emergency, Abdomen
DOI: 10.1594/ecr2012/C-1561

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Learning objectives

Describe the main causes and radiological findings of acute gastric dilatation (AGD) as well as its complications.

Background

Acute gastric dilatation was first described by Duplay in 1833.

It is a potentially life-threatening event with relatively few references in the literature.

It is encountered most often as a postoperative complication, volvulus, superior mesenteric artery syndrome (SMAS) and in a multitude of disorders, such as anorexia and bulimia nervosa, psychogenic polyphagia, trauma, diabetes mellitus, medications, electrolyte abnormalities, …

The pathogenesis of acute gastric dilatation is still unclear, with different theories being postulated.

Morris et al. claimed that anesthesia and debilitation may be predisposing factors. In 1859 the atonic theory was introduced by Brinton. In patients with eating disorders, the stomach undergoes atony and muscular atrophy during a period of starvation, so that a sudden ingestion of foods overtaxes an already weakened stomach. A mechanical theory, in relation with SMAS was proposed by Rokitansky in 1861.

Other authors suggest that a acute gastric dilatation may be a functional entity secondary to regional diseases. However, the pathophysiology is probably multifactorial.

Gastric infarction as a complication of acute gastric dilatation is an unusual associated circumstance.

The gastric reservoir is well known for its rich vascular network which generally protects it from ischemia when significant gastric distension occurs, so gastric necrosis is a very rare event. When intragastric pressure from gastric distension exceeds 20 cmH2O, intramural blood flow is impaired and results in gastric ischemia and necrosis. In cases of AMGD (acute massie gastric dilatation), intragastric pressure usually exceeds 30 cmH2O and produces a dramatic decrease of intramural blood flow, with necrosis and perforation usually following. In the majority of the cases, the necrosis that occurs along the greater curvature and gastric fundus is significant and requires emergent treatment.
This event is more common in young women (67%) and usually occurs along the greater curvature (63%).

Clinically, emesis is present in more than 90%, and significant diffuse abdominal distension and abdominal pain is common. If perforation results, symptoms become much more marked due to peritoneal irritation, that can lead early on to a profound vagal response, resulting in neurogenic shock and later to septic shock.

On physical examination, a diffuse tympany on palpation, a splash on percussion, and a distended Douglas pouch may be found.

Most frequently acute gastric dilatation requires surgical intervention to prevent gastric necrosis. An early diagnosis with a prompt gastric decompression in the phase of parietal ischemia and mucosal necrosis may avoid an unnecessary surgery.

MDCT is the most useful diagnostic tool, since it shows gastric distension, presence of ischemia and also can reveal the cause.

Over the last five years three cases of AGD associated to ischemia were diagnosed in our department. The three cases had different cause: SMAS, eating disorder and volvulus, and a different outcome: healing with conservative treatment or surgery, and death.

**Imaging findings OR Procedure details**

Imaging is a key factor in diagnosis.

Radiographic recognition of acute gastric dilatation is not difficult.

An enormous left upper quadrant soft tissue mass in the region of the stomach with a displacement of adjacent viscera laterally and inferiorly is seen in the supine film, and an air-fluid level within the stomach on upright or decubitus examination.

Abdominal radiograph also can shows streaks of air in the stomach wall or branching lucencies in right upper quadrant.
MDCT is the most useful diagnostic examination. It is a relatively simple, non-invasive and accurate imaging technique.

MDCT reveals massive gastric dilatation, often occupying the abdominal cavity from the diaphragm to pelvis, and fluid level, and allows visualization of small amounts of intraabdominal air that could not be seen at conventional abdominal radiography. Gastric wall pneumatosis and portal venous gas are ominous signs that are often associated with bowel ischemia and high mortality rates.

However, findings of portomesenteric vein gas at CT should be carefully evaluated in the context of clinical findings.

Gastric distention can produce minimal mucosal disruption that allows intraluminal gas to become intravascular. Portomesenteric vein gas secondary to gastric distention can occur in association with iatrogenic gastric and bowel dilatation (gastrostomy, sclerotherapy for gastric varices, endoscopic retrograde cholangiopancreatography), mechanical obstruction, and acute gastric dilatation.

Nevertheless, when CT demonstrates portomesenteric vein gas and clinical findings suggest the presence of gastric ischemia, surgery is mandatory.

Pneumoperitoneum, peritoneal free fluid and discontinuity in the gastric wall are imaging findings that we can detect in cases of perforation.

On the other hand, compression by the distended stomach and traction of the vascular pedicle by the small intestine and mesentery could cause duodenal obstruction, reflux into the pancreatic ducts and secondary pancreatitis.

MDCT may determinate the cause such as SMAS (superior mesenteric artery syndrome) or gastric volvulus.

MDCT evaluation of SAMS reveals duodenal obstruction causing proximal distension and its relationships to the aorta and the superior mesenteric artery. It allows the evaluation
of aorto-mesenteric distance (<8mm) and the aortomesenteric angle (<15º) and the visualization of retroperitoneal and mesenteric fat.

The association between SMAS and fast weight loss was emphasized by many authors. A SMAS may be precipitated by a binge-eating episode leading to an AGD.

Gastric volvulus, which is defined as abnormal torsion of the stomach, usually of more than 180°, also can be confirmed.

CT findings of gastric volvulus may vary depending on the site of torsion, and the final position of the stomach. In organoaxial volvulus, CT depicts an "upside-down" stomach, with the lesser curvature located below the greater curvature. The gastroesophageal junction also is lower than normal and the duodenum appears distorted. In mesenteroaxial volvulus, the pylorus rotates from right to left so that the stomach is "right-side up" at CT.

We could also find gastric dilatation with stenosis of the lower esophagus and/or gastroduodenal junction.

Fig. 4,5,6: A 36-year-old female with history of bulimia nervosa, who presented to the emergency room with the chief complaint of abdominal pain, vomiting and abdominal distension.

Axial (Fig. 4 on page 9) coronal (Fig. 5 on page 10) and sagittal (Fig. 6 on page 12) contrast enhanced CT images show a massively dilated, air-filled stomach with streaks of air in the stomach wall (arrow-head in 4).

Tubular areas of decreased attenuation in the liver, findings that are consistent with gas in the intrahepatic portal veins, are also observed (arrows in 5).

No cause was visualized.

The ischemia was confirmed at surgery and a total gastrectomy was performed.

Fig. 7,8,9,10: A 32-year-old female studied 2 years ago by dysphagia, who came to the emergency department, 3 weeks after a spontaneous child birth, with total food intolerance, abdominal pain and abdominal distension. An abdominal radiograph showed a massive distended stomach and duodenum (Fig. 3 on page 8).

Nasogastric tube was placed; five litres of fluid were removed from the stomach, and the pain decreased.

Axial (Fig. 7 on page 14 Fig. 8 on page 14) and oblique (Fig. 9 on page 15) contrast enhanced CT images show a marked thickened gastric wall, gastric
pneumatosis, a minimal amount of gas in the portal veins (arrow in 7) and duodenal distension with a stop at midline (arrow in 8).

Sagittal contrast enhanced CT image (Fig. 10 on page 16) demonstrates a relatively small distance between the aorta and the superior mesenteric vessels (0.6cm) and a narrow aorto-mesenteric angle (15º).

Conservative treatment was decided. The patient regained weight and was discharged.

**Fig. 11,12,13,14,15:** A 6-year-old girl with cerebral palsy, who arrived in emergency department in acute abdominal pain, massive abdominal distension and peritonitic signs.

Axial (Fig. 11 on page 17, Fig. 12 on page 18), coronal (Fig. 13 on page 19, Fig. 14 on page 21) and sagittal (Fig. 15 on page 23) contrast-enhanced CT images with lung window show a gastric distension with air-fluid level, gastric, portal and mesenteric pneumatosis (arrows in 14) associated to intrabdominal free fluid and a large pneumoperitoneum and pneumomediastinum.

CT images show also distal esophageal stenosis with prestenotic dilatation (asterisk) and stenosis at the gastroduodenal junction.

A volvulus was confirmed at surgery with fatal postoperative outcome.

**Images for this section:**
Fig. 1: An abdominal radiograph showing a mass effect which is displacing the bowel gas to the right and inferior aspects of the abdomen
Fig. 2: A supine abdominal radiograph showing a mass effect which is displacing the bowel gas to the right and inferior aspects of the abdomen, and an air-fluid level in the stomach
Fig. 3: An abdominal radiograph showing a massive distended stomach and duodenum
Fig. 4: Axial contrast enhanced CT image shows a massively dilated, air-filled stomach with streaks of air in the stomach wall (arrow-head)
Fig. 5: Coronal contrast enhanced CT image shows a massively dilated, air-filled stomach with streaks of air in the stomach wall. Tubular areas of decreased attenuation
in the liver, findings that are consistent with gas in the intrahepatic portal veins, are also observed (arrows)
Fig. 6: Sagital contrast enhanced CT image shows a massively dilated, air-filled stomach with streaks of air in the stomach wall

Fig. 7: Axial contrast enhanced CT image shows a marked thickened gastric wall, gastric pneumatosis and a minimal amount of gas in the portal veins (arrow)
Fig. 8: Axial contrast enhanced CT image shows duodenal distension with a stop at midline (arrow)
Fig. 9: Oblique contrast enhanced CT image shows a marked thickened gastric wall and gastric pneumatosis
Fig. 10: Sagital contrast enhanced CT image demonstrates a relatively small distance between the aorta and the superior mesenteric vessels (0.6cm) and a narrow aortomesenteric angle (15°)
Fig. 11: Axial contrast-enhanced CT images with lung window show a gastric distension with air-fluid level, gastric, portal and mesenteric pneumatosis associated to intrabdominal free fluid and a large pneumoperitoneum and pneumomediastinum. CT images show also distal esophageal stenosis with prestenotic dilatation (asterisk) and stenosis at the gastroduodenal junction.
**Fig. 12:** Axial contrast-enhanced CT image with lung window shows a gastric distension with air-fluid level, gastric, portal and mesenteric pneumatosis associated to intrabdominal free fluid and a large pneumoperitoneum
**Fig. 13:** Coronal contrast-enhanced CT image with lung window shows a gastric distension, gastric and portal pneumatosis associated to a large pneumoperitoneum and pneumomediastinum
**Fig. 14:** Coronal contrast-enhanced CT image with lung window shows portal and mesenteric pneumatosis (arrows) associated to intrabdominal free fluid and pneumoperitoneum CT images show also distal esophageal stenosis with prestenotic dilatation (asterisk) and pneumomediastinum.
Fig. 15: Sagital contrast-enhanced CT image with lung window shows a gastric distension with air-fluid level a large pneumoperitoneum and pneumomediastinum CT image shows also distal esophageal stenosis with prestenotic dilatation (asterisk)
Conclusion

Acute gastric dilatation is known as a rare but life-threatening emergency.

Because a delay in diagnosis can have devastating consequences, including gastric ischemia and infarction, prompt diagnosis is essential.

With prompt recognition, acute gastric dilatation can be treated conservatively.

Radiologists should familiarizes with this entity and know its principal radiological findings.

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