Computed Tomography (CT) in Gastric Urgencies: What radiologists need to know.

Poster No.: C-3223
Congress: ECR 2018
Type: Educational Exhibit
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Keywords: Volvulus, Trauma, Hemorrhage, eLearning, Contrast agent-oral, Contrast agent-intravenous, PACS, CT-Angiography, CT, Gastrointestinal tract, Anatomy, Abdomen
DOI: 10.1594/ecr2018/C-3223

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Learning objectives

Review and illustrate the CT findings of urgent gastric pathology.

Describe the key points for diagnostic imaging of gastric emergencies.
Background

Abdominal pain, nausea and vomiting are very frequent symptoms in patients who go to the emergency service. These symptoms can be secondary to pathologies such as gastritis, carcinoma, lymphoma, carcinoid, metastasis, bezoar or corrosive damage of the stomach.

Clinical and laboratory tests are frequently nonspecific, so when faced with the suspicion of urgent gastric pathology, CT will be the initial test of choice for diagnosis and treatment planning. Endoscopy remains an important tool for assessing acute gastric disease, although it is more invasive and not as readily available as CT.

The CT can evaluate the mural and extramural extension of the diseases, having limitations when evaluating the mucosa. Although the evaluation of the mucosa is poor in CT compared to endoscopy, CT is very useful to identify inflammatory pathology (gastritis, ulcers) and complications of gastric pathology (perforation, obstruction and hemorrhage).

Advances in CT technology and three-dimensional postprocessing (3D) software have allowed endoscopic images based on CT (virtual endoscopy), as well as accurate staging of neoplastic diseases of the stomach.

The acquisition of appropriate CT images requires patient fasting, adequate gastric distension and a negative intraluminal contrast agent.
Findings and procedure details

The stomach is divided into five segments: the cardia, the fundus, the body, the antrum and the pylorus.

The arterial supply to the stomach comes from the three branches of the celiac axis: the left gastric, common hepatic and splenic arteries, forming two arterial arches. Numerous anastomoses make the stomach relatively resistant to ischemia.

The gastric wall is composed of mucosa, submucosa, muscularis propria and serosa. It presents a trilaminar appearance after the administration of contrast (can increase up to 120 HU) that is altered in gastric disease.

In a relaxed stomach, the thickness of the wall at the level of the body is less than or equal to 5 mm that can measure up to 12 mm at the level of the antrum.

To assess the stomach, proper distension of the gastric chamber is essential, this prevents the disease from going unnoticed or that the collapsed gastric wall can simulate a disease.

The protocol of the CT will vary depending on the patient's clinic, being able to obtain images with axial multidetector CT with sections of 3 mm thickness at intervals of 2 mm, generally after the administration of intravenous iodinated contrast, at a speed of 3cc/sec and with a delay of 70 seconds.

Intravenous contrast is used to evaluate neoplastic, inflammatory conditions and gastric ischemic involvement.

If the patient has a history of gastric disease, water (1000-1500ml) is administered orally as a negative contrast, 15 minutes before the examination, as this allows a better assessment of the gastric wall and a better detection of subtle disease. Positive contrast may mask the presence of intraluminal hemorrhage.

Multiphasic contrast tests are not routinely performed in patients with suspected gastric disease, but an angiographic CT protocol is used that includes the non-contrast, arterial and portal venous phases for patients with suspected acute gastrointestinal bleeding.
The pathologies to be studied have been classified into 5 groups: **Fig. 1**

1. **Inflammatory** (includes gastritis, emphysematous gastritis and ulcer).

2. **Obstructive** (includes secondary to gastric volvulus, peptic ulcer, malignant process, bezoar syndrome, Bouveret syndrome, gastric band slippage, vertical band gastroplasty).

3. **Gastric perforation** (secondary to peptic ulcer, malignant process, gastric band and penetrating trauma).

4. **Hemorrhage**.

5. **Ischemia**.

**GASTRITIS:**

Gastritis is a common benign disease of the stomach that is characterized by the presence of inflammatory cells in the stomach wall. Gastritis can be broadly classified as erosive (related to non-steroidal anti-inflammatory drugs, alcohol, stress, systemic diseases, viral or fungal infections), non-erosive related to infection by Helicobacter pylori, atrophic gastritis and hypertrophic gastritis (related to Zollinger-Syndrome Ellison and Menetrier’s disease) and noninfectious (granulomatous and oesinophilic gastritis).

**The most common CT finding** in patients with gastritis is the diffuse thickening of the gastric folds and the wall. In severe cases, the gastric wall will demonstrate a low attenuation compatible with edema and inflammation of the submucosa. If the inflammation is severe, there may be stratification or "halo" of the gastric wall that is better seen in arterial phase images. Neoplasms do not create this striated appearance. **Fig. 2**

*H. pylori* gastritis can simulate a gastric neoplasm because it often causes a focal thickening along the greater curvature of the stomach or circumferential wall of the antral wall. Pyloric hypertrophic stenosis in adults can also manifest as segmental thickening of the wall. Because CT findings of gastritis and tumors can overlap, endoscopy is often necessary for the definitive diagnosis.

In patients with hypertrophic gastritis and elevated gastrin levels, CT is useful for the diagnosis of gastrinoma, location and its relationship with adjacent structures. Also, a nonspecific finding such as ascites may be useful in diagnosing the serous form of eosinophilic gastritis in the appropriate clinical setting.

**GASTRIC ULCER**
Between the gastric body and the antrum is the angularis incisura, which is the place of transition to the mucosa. Due to this transition, the incisura is the most common site for gastric ulcers.

**On CT**, most gastric ulcers are not visible, because they affect only the superficial layers of the gastric wall. However, deep ulcers that have penetrated or perforated the gastric wall can be detected. The perforation secondary to the ulcer appears as inflammatory changes, thickening of the gastric wall, as well as extraluminal air bubbles or pneumoperitoneum. Fig. 3

Marginal ulcers are observed in the suture line in postoperative patients, and specifically in patients who have undergone gastrojejunostomy and Roux-en-Y gastric bypass. The ulcer usually occurs on the jejunal side of the gastrojejunal anastomosis. Complications of marginal ulcers include stoma stenosis, hemorrhage, and perforation. Marginal ulceration is the most frequent cause of early and late upper gastrointestinal bleeding after Roux-en-Y gastric bypass.

**ENFISEMATOSA GASTRITIS**

Emphysematous gastritis is an infrequent entity that is usually caused by the invasion of the gastric wall by aerobic and anaerobic bacteria, as well as some fungal species (Escherichia coli, Klebsiella pneumoniae, Enterobacter species, Pseudomonas aeruginosa and Candida species). It is a life-threatening disease whose cardinal symptom is the intense abdominal pain that can occur with sepsis and shock.

**On CT** there is thickening of the wall and edema, with air inside the layers of the wall that can dissect in the gastric veins and the portal venous system. You can also observe, although rarely, similar image characteristics in gastric ischemia or caustic ingestion.

There is a benign condition called gastric emphysema that can also lead to air inside the gastric wall in the context of a recent procedure. In fact, gastric emphysema is seen more often than emphysematous gastritis.

**GASTRIC OBSTRUCTION:**

Gastric obstruction is a physiopathological and clinical consequence secondary to any pathological process that prevents gastric emptying. The usual clinical symptomatology is usually early satiety, vomiting, gastric distention and abdominal pain.
They can be of benign or malignant cause, being from 50 to 80% of the cases of high occlusion attributed to malignant pathology (10-15% of pancreatic cancer in advanced stages and up to 5-10% of gastric neoplasms). As for the benign causes, the most frequent is usually peptic ulcer (5%), corrosion secondary to caustics, inflammatory processes (acute and chronic pancreatitis, inflammatory polyp, eosinophilic gastroenteritis), stenosis induced by NSAIDs, iatrogenic (post-vagotomy), endoscopic mucosal post-resection), benign tumors (adenoma, lipoma or stromal tumors) and other causes (Bouveret syndrome, annular pancreas, amyloidosis, duplication cyst, hypertrophic pyloric stenosis in the adult, bezoar or foreign body).

**CT shows** a general dilated stomach and findings based on its etiology, such as: peptic ulcer (edema of the gastric wall or stenosis), malignancy (mass or nodular thickening that enhances contrast, replacement of the submucosa with soft tissue, lymphadenopathy or metastasis), bezoar (mottled material), Bouveret's syndrome (obstruction due to ectopic gallstone in the distal stomach or duodenum, with air in the gallbladder or biliary tree), gastric band slipped: increased angle (normal 4° -58°) and vertical band gastroplasty (distended gastric remnant with fibrosis or food impaction in the stoma). **Fig. 4 and Fig. 5.**

**GASTRIC VOLVULUS:**

Gastric volvulus is the torsion of more than 180° of the stomach that produces an obstruction of the gastric outlet and can cause ischemia or perforation. Patients present classically with sudden epigastric pain, intractable arcades, and inability to pass a nasogastric tube (the Borchardt triad).

It is common in elderly patients with a hiatal hernia and in most cases it is secondary to defects in the diaphragm, with paraesophageal hernia being the most frequent cause.

Because of the potential for ischemia and perforation, acute gastric volvulus has a high morbidity and mortality if it is not treated quickly with stomach decompression, reduction of volvulus and correction of the underlying cause.

Organoaxial volvulus is the obstruction of the stomach due to rotation around the longitudinal axis of the stomach, which causes the antrum to move anteriorly and the fundus to rotate posteroinferiorly, so that the greater curvature is greater than the lesser curvature. Gastric volvation can be classified into two major subtypes according to the axis of rotation: organic (59%), mesenteric (29%) and the rest are mixed.

**On CT,** certain characteristics of the volvulus are observed, including: obstruction of the gastric outlet associated with the abnormal location of the gastric outlet, lack of passage
of the enteric contrast material, thickening of the wall, presence of adjacent fluid and involvement of fat.

The organ-axial volvulus occurs when there is a rotation of the stomach in its longitudinal axis, producing a cranial displacement of the greater gastric curvature, thus leaving the lower gastric curvature located caudally in the abdomen (the antrum rotates anterosuperiorly and the fundus posteroinferiorly). In adults it is usually secondary to para-esophageal hernias or post-traumatic cases and in children secondary to hernias of Bochdaleck. The volvulation is severe when there is a rotation superior to 180°, observing an abdominal distension with retention of oral contrast and predisposing to ischemia and gastric perforation. Fig. 6

In the mesenteroaxial volvulus the stomach rotates around its short axis, so that the antrum moves above the gastroesophageal junction, twisting its vascular supply. The rotation is usually partial (less than 180°) and is not associated with underlying diaphragmatic defects.

**GASTRIC PERFORATION:**

Gastric ulcers are the main cause of perforation of the gastroduodenal tract followed by malignant necrotic or ulcerated neoplasms. They can also be secondary to trauma or iatrogenesis.

According to its location, we speak of ulcers of the anterior surface of the lesser curvature and ulcers of the posterior wall. In the first case, they tend to associate more frequently with free perforation and the diagnosis is based on the demonstration of the pneumoperitoneum. If the ulcer is located in the posterior gastric wall, it is mostly associated with contained or covert perforations, in which the pancreas is usually affected, producing inflammatory changes. The ulcer is penetrating when it contacts the neighboring organs (liver or pancreas).

**CT** helps to predict the site of perforation through signs such as the presence of extraluminal air, discontinuity of the gastric wall, leakage of intraluminal contrast (in the case of oral contrast administration), inflammation of the surrounding mesenteric fat, thickening of the affected wall and presence of adjacent fluid collections. The location of the air is usually seen in the vicinity of the perforation. The presence of air bubbles in the periportal space, around the falciform ligament and air trapped in the fissure of the falciform and round ligament, are findings suggestive of perforation of the upper gastrointestinal tract. Fig. 7
The presence of intra- or retroperitoneal air is not always associated with perforation, since it can be found in cases of mechanical ventilation, pulmonary barotrauma, peritoneal lavage or pneumothorax.

Reconstructions with MIP can help visualize the wall defect. The use of positive oral contrast helps to identify extraluminal contrast leakage.

**GASTRIC TUMOR:**

Gastric perforation may be secondary to a gastric malignancy, particularly in ulcerated masses such as those seen with adenocarcinoma, lymphoma, and large gastrointestinal stromal tumors (GIST). Perforation of gastric adenocarcinoma usually occurs in patients older than 65 years with advanced disease. Ulcerated gastric lymphoma can also cause gastric perforation, although perforation of the small intestine is more common.

**CT shows** the mass or focal thickening of the gastric wall and replacement with soft tissue of the submucosa. Extragastric signs of malignancy, such as lymphadenopathy or metastasis, may also be present.

Any lesion that produces a mass effect on the gastric wall is considered a gastric tumor. 90% of gastric cancers are adenocarcinomas, with the remaining 10% corresponding to non-Hodgkin's lymphomas, stromal and carcinoid tumors. Benign gastric tumors are rare and their main clinical interest lies in ruling out their possible evolution towards malignancy. Fig. 8, Fig. 9, Fig. 10, Fig. 11 and Fig. 12.

**GASTRIC PATHOLOGY POST PROCEDURE OR POST SURGICAL:**

Gastric bands can cause gastric perforation either acutely as a postoperative complication or in the chronic context secondary to the erosion of the transmural band. Acute perforation is a rare complication, but chronic erosion is slightly more common. Erosion of the gastric band may be due to surgical trauma, inflammatory reaction to the foreign body or use of NSAIDs.

The clinical presentation varies, from asymptomatic to acute abdominal emergency.

**On CT,** in the perforation of patients with gastric band, free extraluminal gas or loculated or subphrenic abscess can be seen. You can see oral contrast material or air that outlines the band. Patients with band erosion may be presented alternatively with infection of the site or its route, with involvement of the adjacent fat. Fig. 13, Fig. 14 and Fig. 15.
GASTRIC HEMORRHAGE:

Acute gastric bleeding presents different etiologies among which are erosions or ulcers, bleeding varicose veins, vascular lesions and neoplasms. The bleeding can be of venous or arterial cause. The most frequent etiology of venous bleeding are gastric or esophageal varices secondary to portal hypertension, although up to 30% of patients with portal hypertension may have arterial bleeding. The clinical presentation depends on the amount of blood lost, so those with a loss of less than 100 ml or presenting in a state of systemic shock can be asymptomatic if they have a loss greater than 15% of the total blood volume.

Direct signs of hemorrhage include hematemesis, vomiting in coffee grounds, hair or, in the context of rapid bleeding, hematochezia. Although endoscopy is the preferred method to diagnose and treat upper gastrointestinal bleeding, CT is useful in cases where endoscopy is not clinically feasible or is not diagnostic.

Unconstant CT shows intraluminal hyperdense areas that allow us to locate a sentinel hematoma. In the arterial phase extravasation of intraluminal contrast is observed (diagnosis of acute arterial gastric bleeding). The portal phase will help confirm the diagnosis of arterial bleeding, evidencing an increase in the amount of intraluminal contrast (contrast accumulation). If the intraluminal extravasation occurs in this phase and not in the arterial one, it will be suggestive of bleeding of venous origin.

The residual contrast ingested in the stomach or medications, surgical material or foreign bodies, can potentially result in both false-positive and false-negative studies by mimicking or obscuring bleeding. Fig. 16

VASCULAR ISCHEMIA:

Gastric ischemia is an uncommon condition caused by diffuse or focal vascular insufficiency. Despite the abundant blood supply to the stomach, systemic hypotension (as seen in sepsis or shock) can cause gastric ischemia. Other reported causes of gastric ischemia include celiac and mesenteric stenosis, vasculitis, and disseminated thromboembolism.

On CT, focal ulceration can be seen until thickening of the gastric wall and intramural gas (pneumatosis). Ischemic ulcerations occur more frequently along the anterior and posterior gastric walls near the anastomoses between the two arterial arches over the lesser and greater curvatures. Gastric dilation can also be observed and is believed to be due to ischemic gastroparesis. Fig. 17
Images for this section:

Fig. 1

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Fig. 2: Axial images of the abdomen in two different patients. Thickening of the antral wall, mucosal enhancement (arrows) and submucosal edema compatible with gastritis are observed.

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**Fig. 3:** Penetrating ulcer in the gastric antrum. The continuity solution (arrow), inflammatory changes, thickening of the gastric wall and extraluminal air bubbles or pneumoperitoneum are seen.

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Fig. 4: Coronal and sagittal section showing a mass at the level of the cardia (arrows), which interrupts the passage of food to the gastric chamber. The final diagnosis was a neoformative process of the cardia.

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**Fig. 5:** Axial section of the same patient from the previous figure. You see the mass that covers the entire cardia causing obstruction to the passage of food.

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**Fig. 6:** Gastric volvulus: obstruction of the stomach due to rotation around the longitudinal axis of the stomach (arrows), which causes the antrum to move anteriorly and the fundus rotates posteroinferiorly.

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Fig. 7: Gastric perforation: Extraluminal air (arrow) is seen adjacent to the perforation. The discontinuity of the gastric wall is seen with leakage of gastric contents, inflammation of the surrounding mesenteric fat, thickening of the affected wall and presence of adjacent fluid collections.

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Fig. 8: Gastric adenocarcinoma: Axial and coronal sections. The mass is seen in the lesser curvature of the stomach that produces focal thickening of the gastric wall and soft tissue tissue.

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**Fig. 9:** Gastric linitis: Axial cuts. Diffuse thickening of the gastric wall (arrows) with decreased gastric lumen in a patient presenting with dyspepsia and insidious abdominal pain.

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Fig. 10: Gastric lipoma: The lipoma of the gastric wall (arrows) is seen. In our patient it was a casual diagnosis.

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Fig. 11: Gastric lymphoma: Mass of homogeneous attenuation that produces a marked thickening of the wall (arrow) of the stomach with a large lateral extension of the tumor (along the wall of the stomach), that is, dissemination by the submucosa.

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Fig. 12: GIST: They are frequently exophytic. Typically, the mass is of soft tissue density with central areas of lower density when there is necrosis (arrow), usually in large tumors.

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Fig. 13: Gastric pathology post procedure: The gastrostomy material is objectified in a neurological patient. He goes for severe abdominal pain that does not give up with analgesics and fever. Diagnosis is abscess around the tube (arrow)

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Fig. 14: Gastric pathology post procedure: An important amount of extraluminal air (arrow) is observed in a patient, one week after having undergone gastrostomy placement.

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Fig. 15: Post-surgical gastric pathology: A defect of the anterior abdominal wall is seen where the gastric chamber protrudes. It is compatible with gastric eventration.

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**Fig. 16:** Digestive hemorrhage in a patient with pancreatitis. High density content (arrow) is seen inside the gastric chamber at the pyloric antrum level after contrast administration.

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Fig. 17: Vascular Ischemia: Stenosis is seen at the origin of the artery of the celiac trunk.

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Conclusion

With the development of new and better software techniques, the use of CT is becoming more frequent in urgent abdominal pathology. Although endoscopy plays an important role in the evaluation of acute gastric pathology, CT is often the first imaging technique performed in the emergency department and offers a quick and accurate diagnosis that helps plan treatment.

It is important to be familiar with the characteristics of CT in various gastric disorders for proper diagnosis and treatment.
References


