Bowel Perforations

Patrice Taourel, Joseph Pujol, and Emma Pages-Bouic

Contents

P. Taourel (✉) · J. Pujol · E. Pages-Bouic
Département of Imaging, Hôpital Lapeyronie,
371, avenue du Doyen Gaston Giraud,
34295 Montpellier Cedex 5, France
e-mail: p-taourel@chu-montpellier.fr

Abstract

Gastrointestinal tract perforation is an emergent condition that requires prompt surgery. Diagnosis largely depends on imaging examinations, and correct diagnosis of the presence, level, and cause of perforation is essential for appropriate management and surgical planning. Although plain radiography classically remains the first imaging modality, the high clinical efficacy of computed tomographic examination in this field has been well recognized. CT semiology is based on direct findings, including the identification of the bowel wall interruption, and indirect findings, including a pneumoperitoneum, a peridigestive infiltration, and a bowel wall thickening. These findings have different patterns according to the cause and the site of the bowel perforation. This chapter deals with the CT findings, CT pitfalls, and CT impact in the diagnosis and management of bowel perforation.

1 Introduction

Gastrointestinal (GI) tract perforations are a common cause of acute abdominal pain syndrome. They account for 1–3% of cases acute abdomen syndrome. Diagnosing intestinal perforation is a surgical emergency.

As for suspicion of obstruction and colic pain, suspected perforation remains one of the indications for abdominal plain film (APF). Nevertheless, the development of computed tomography (CT) in clinical management of acute abdominal pain along

P. Taourel (ed.), *CT of the Acute Abdomen,* Medical Radiology. Diagnostic Imaging,
DOI: 10.1007/174_2010_86, © Springer-Verlag Berlin Heidelberg 2011

with the setting up of an accurate and efficient semiology allowed improved diagnosis of GI tract perforations with respect to the presence, localization site, and cause.

Early diagnosis of GI tract perforation together with identification of the site and cause improves the prognosis of GI tract perforation and has a great impact on the therapeutic choice, including the type of surgery and the means of access.

2 Pathology

2.1 Definition of a Bowel Perforation

GI perforations are discontinuities of the GI wall which allow intestine lumen and peritoneal cavity or subperitoneal or retroperitoneal spaces to communicate.

The peritoneal irritation may be caused by a purulent fluid, as well as by an aseptic fluid (at least at the beginning of the evolution), in particular in the case of perforation of a gastroduodenal ulcer. Peritonitis may be generalized or localized. When the fluid bathes the entire peritoneal cavity, it is a generalized peritonitis. When only part of the abdominal cavity is involved, it is a localized peritonitis. Some localized peritonitis clinically manifest themselves as plastic peritonitis, also called plastron, due to an inflammatory reaction of the surroundings organs (epiploic fat in particular). On palpation, the plastron makes up a resisting mass, a "boardlike" abdominal wall.

2.2 Causes of GI Tract Perforations

GI tract perforations can affect any segment of the digestive tract, and complicate every digestive disease, whether tumoral, inflammatory, ischemic, postradiation, or ulcerous. Nevertheless, in order of frequency, perforations are most often the complication of a gastroduodenal ulcer or a sigmoid diverticulitis.

2.2.1 Esophageal Perforation

Spontaneous perforations—not caused by a trauma (mostly from iatrogenic or endoscopic causes)—of the esophagus are commonly known as Boerhaave syndrome. It differs from Mallory–Weiss syndrome, in which lesions, often linear, occur within the mucosa and the submucosa, and are characterized by an upper hemorrhage (Rubesin and Levine 2003). Similarly to Mallory–Weiss syndrome, it is postulated to result from a sudden rise in intraluminal esophageal pressure, commonly associated with vomiting (typically with alcoholism), which can induce a dilatation of the lower esophagus, up to 5 times its normal diameter. Perforation typically occurs at the weakest point in the esophagus, at the left posterolateral wall of the lower third of the esophagus (Korn et al. 2007). Mediatinitis frequently is seen late in the course of illness. The contiguous pleura is often ruptured.

2.2.2 Gastroduodenal Perforation

Perforations of a gastroduodenal ulcer represent the leading cause of GI tract perforations. As an example, in the largest series assessing multidetector CT in GI perforations, more than half of the perforations were related to a gastroduodenal ulcer (Imuta et al. 2007). On the anatomopathologic plan, chronic ulcer is a round or oval-shaped loss of deep substance, with sharp margins, covered with a fake yellowish membrane, amputating the muscularis, which becomes sclerous. Depending on the depth of the parietal lesion, true ulcer must be distinguished from abrasion, erosion, and ulceration. Perforations complicate 2–10% of peptic ulcers (Ramakrishinan and Salinas 2007; Behrman 2005). They more frequently affect the first part of the duodenum and its anterior face, and more rarely the gastric antrum and the lesser curvature. The prevalence of perforations is the same for both gastric and duodenal ulcers. However, duodenal ulcers are 3 times more frequent than gastric ulcers. The causes of perforations are dependent on multiple factors, and are commonly associated with *Helicobacter pylori* infections. Intake of nonsteroidal anti-inflammatory drugs is found in about half of cases of perforated ulcers. Cocaine was shown as an important causative factor in perforated juxtapyloric ulcers in a series of patients from an urban hospital (Feliciano et al. 1999). Other rarer causes are associated with an increased prevalence of digestive ulcer, such as Zollinger–Ellison syndrome, in which ulcers are more likely jejunum ulcers, and Crohn disease.

Gastric tumor perforations are rarer than ulcer perforations and can be associated with adenocarcinoma, leiomyosarcoma, or lymphoma. The perforation is

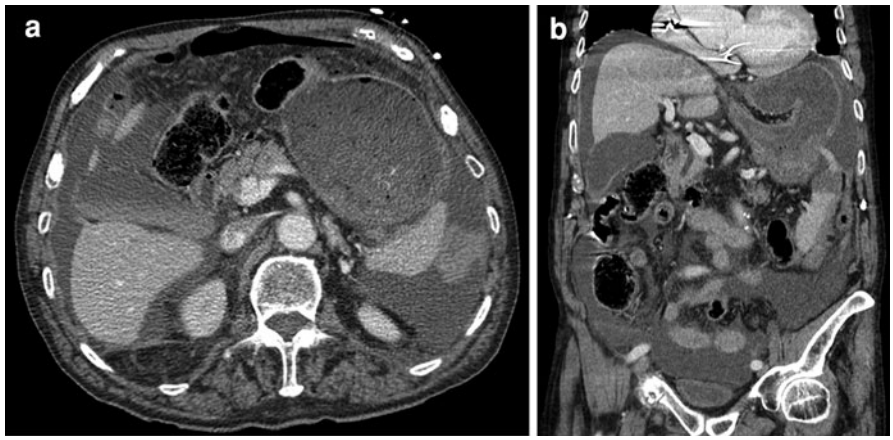


Fig. 1 Gastric volvulus. Axial (**a**) and coronal (**b**) views show a distended stomach with a hyperdense content, a thickened gastric wall, and a pneumoperitoneum

seldom the reason for the discovery of the gastric tumor. Gastric penetrations associated with a lymphoma are often due to a lymphoma of the mucosa-associated lymphoid tissue type. The perforation may either be related to the evolution of the disease itself or secondary to chemotherapy.

Gastric perforation may complicate gastric volvulus (Fig. 1), which is a disease with a high rate of perforation.

At the duodenal level, except from ulcer and tumor perforations, duodenal diverticulum is a typical but rare cause of digestive perforation. The most frequent duodenal diverticula are juxtapapillary diverticula created by herniation of the mucosa or submucosa through the muscularis mucosae at a weak spot known as the duodenal window. Perforation of those diverticula is a rare complication, and is most commonly of a retroperitoneal site.

2.2.3 Small Bowel Perforations

Perforations of the small bowel are rare, besides those related to ischemic causes. Perforations of ischemic origin occur either in a context of mechanical obstruction as a complication of ischemic digestive strangulation or in a context of primary ischemia as a severity factor, most frequently associated with arterial ischemia or low-debit ischemia, rather than venous ischemia. Other causes are rare and account for one case per year and for every 350,000 inhabitants (Kimchi et al. 2002). They include Crohn disease, tumor, infection (tuberculosis, opportunist infection), and jejunal or Meckel diverticulitis. The pathophysiological changes, the frequency, and the site of the perforation differ according to the cause.

Small bowel perforation complicating mechanical occlusion Obstruction is due to a band or internal or external hernia. Strangulation complicates a mechanism of incarceration with a severe proximal distension which induces a venous stasis, followed by an arterial stasis, with pneumatosis and/or perforation.

Small bowel perforation complicating primary small bowel ischemia This is a major complication of digestive ischemia.

Crohn disease Besides ischemia, this is a leading cause of small bowel perforation. Nevertheless, perforations represent a rare complication of Crohn disease, with an incidence of 1%. Other Crohn disease perforating complications exist, such as loop–loop bowel fistula and vesicular fistula.

Tuberculosis Abdominal tuberculosis can affect any organ, although it is often secondary to pulmonary tuberculosis. The ileocecal region is the most frequently affected area in digestive tuberculosis (Ha et al. 1999). Perforations observed in digestive tuberculosis are rare, accounting for 1–10% of all cases.

Lymphoma Roughly all lymphomas affecting the digestive wall are non-Hodgkin lymphomas, mostly of B type. The small bowel is, besides the stomach, the mots common lymphoma digestive affection. Lymphoma represents the most frequent cause of small bowel tumor perforation. Such perforations are more frequent in digestive affections associated with a B-type lymphoma and can be chemotherapy-induced (Ghai et al. 2007). Lymphomatous affections can be either secondary, with digestive localizations associated with splenic or hepatic lymph nodes, or primary,

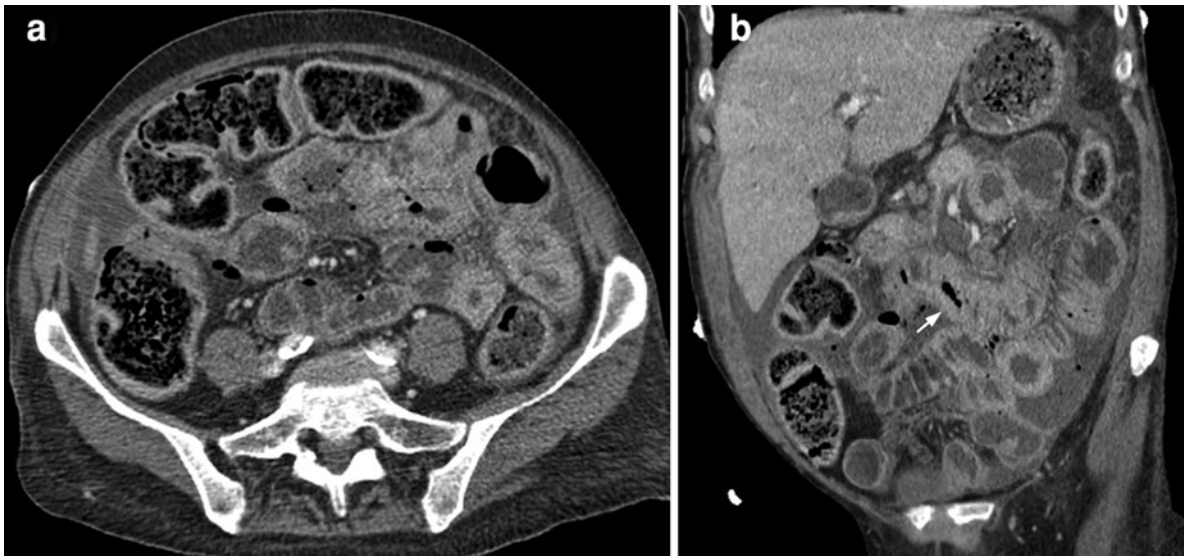


Fig. 2 Small bowel tumor perforation under chemotherapy. Axial (**a**) and coronal (**b**) views show a thickening of the bowel wall involving a large part of the small bowel because of peritonitis but which is more pronounced at the site of the tumor (*arrow*). Close to the tumor there is nonluminal fluid and air, which show the site of the perforation. A malignant tumor of the small bowel treated by chemotherapy was known in this patient

with digestive tract localization only. Other malignant small bowel tumors may be perforated and so may be revealed by acute abdomen. Otherwise, in a patient with a known small bowel tumor, chemotherapy may favor perforation (Fig. 2).

Small bowel diverticulitis The small bowel diverticulum can be either of a jejunal site, typically localized on the mesenteric border of the intestine, or of an ileal site, associated with a congenital Meckel diverticulum. Jejunal diverticula are acquired, mostly occurring between the sixth and the seventh decade of life. They do not have muscular lining. Up to 10% of patients presenting with jejunal diverticulitis will have a small bowel perforation (Park et al. 2005) and hemorrhage; perforations being the most frequent complications. Complications secondary to Meckel diverticulitis are rare, occurring in 2% of patients presenting with Meckel diverticulum (Park et al. 2005) (Fig. 3). Meckel diverticulum is a congenital abnormality, occurring in 1–3% of the population.

Jejunal ulcer Jejunal ulcers can complicate Zollinger–Ellison syndrome. Zollinger–Ellison syndrome is characterized by hypersecretion of gastrin produced by either pancreatic or duodenal endocrine tumors. It is associated with ulcerations and jejunal ulcers which can be perforated.

Vasculitis (aka angiitis) Numerous vasculitides can lead to intestinal perforation, which typically occur during attacks of the disease, and exceptionally are the only manifestations. An anatomoclinical mismatch often exists, and lack of abdominal contracture is sometimes seen, especially in corticoid-treated patients. Small bowel involvement predominates at the ileal level, and perforations are often multiple. Vasculitides are defined by an inflammation associated with necrosis of the vascular wall. They are classified by anatomical and histological criteria, as well as by the caliber of the affected blood vessels, and the presence or absence of vascular granuloma and parietal necrosis (Pagnoux et al. 2003). Vasculitides mainly leading to intestinal perforation include polyarteritis nodulosa, Wegener syndrome, Churg–Strauss syndrome, Behçet syndrome, and rheumatoid-arthritis-associated vasculitides, with polyarteritis nodulosa and Behçet syndrome being the most frequent. In Behçet syndrome, digestive involvement occurs in 10–50% of patients with relatively ubiquitous localization, predominating in the terminal ileus and cecum. Predominant lesions are generally large

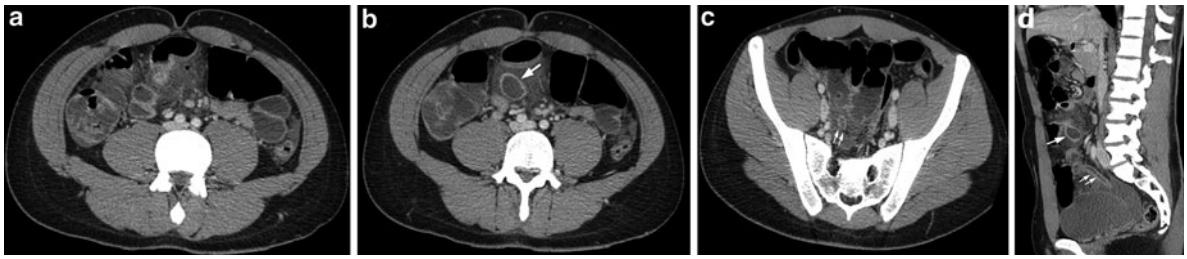


Fig. 3 Perforated Meckel diverticulum. The axial slice (**a**) shows extraluminal fluid and air within the mesentery suggestive of a small bowel perforation. On a slightly lower slice (**b**), distended Meckel diverticula with a very enhanced wall are individualized (*arrow*). On a pelvic slice (**c**), the appendix is well seen (*double arrows*) with abnormally enhanced wall because of reaction to peritonitis. Sagittal reformatting (**d**) shows both the Meckel diverticula (*arrow*) and the appendix (*double arrows*), both having abnormally enhanced walls. Surgery confirmed the perforation of a Meckel diverticulum

and deep ulcers. Ulcers mostly affect the ileocecal region, being more diffuse and less deep at the colic level (Chung et al. 2001).

Foreign body The most frequent sites of intestinal perforation due to a foreign body are the esophagus and the ileal, ileocecal, and rectosigmoid regions. As little as 1% of ingested foreign bodies is responsible for an intestinal perforation, even though this complication is systematically looked for. Fish bones are the leading cause of intestinal perforation due to a foreign body.

2.2.4 Appendiceal Perforation

Appendiceal perforations complicate about 25% of cases of acute appendicitis and represent the natural evolution pattern of ulcerated appendicitis.

2.2.5 Colic Perforation

Sigmoid diverticulitis Colic diverticulosis is a condition commonly occurring in developed countries. It affects up to two thirds of the population beyond 65 years, and can lead to clinical symptoms in about a quarter of patients. Nevertheless, among affected patients, only a few will develop a diverticular perforation. It is considered that as few as 10% of patients hospitalized for a sigmoid diverticulitis will require surgery due to a generalized peritonitis by perforation. Peritonitis in the context of acute diverticulitis is mostly localized peritonitis which corresponds to a poorly limited pelvic abscess, with a risk of secondary rupture leading to a two-step generalized peritonitis. Generalized peritonitis is most often the consequence of a ruptured diverticular abscess in the peritoneum. Depending on whether or not there is a communication between the initial abscess and the colic lumen, peritonitis will be stercoral or purulent (Loiseau et al. 2005).

More rarely, diverticular peritonitis can occur without a preliminary abscess, during perforation of the colic wall from vascular origin (Tagliacozzo and Tocchi 1997).

Lastly, a pseudotumoral sigmoiditis can lead to a mechanical obstruction of the colon complicated with a diastatic perforation of the cecum. This phenomenon more often complicates colon obstructions from neoplasic origin.

Tumoral colic perforation Colic tumors can cause perforations following two mechanisms:

1. By a direct perforation from tumoral origin
2. By a diastatic perforation of the cecum, upstream from a tumor, most often sigmoid, complicated with a mechanical obstruction

In a retrospective study including 1,650 patients with a colorectal adenocarcinoma, a 3% prevalence of perforations was shown (Chen and Sheen-Chen 2000). An association with a colic mechanical obstruction was shown in all 48 cases of perforation. In 35 of the cases, the perforation was located at the tumor site, whereas it was proximal in the remaining 13 cases. As a comparison, a mechanical obstruction existed in 10% of patients. According to other authors, the incidence of colic perforations would be higher in colon cancer patients treated with an anti-angiogenic such as Avastin® (Saif et al. 2007;

Heinzerling and Huerta 2006). Such antiangiogenic treatment could even explain the rise of colic perforation complicating a neoplasia with no mechanical obstruction.

Other causes of colic perforation Many inflammatory colitises (often of right development) or ischemic colitises can cause a colic perforation. The cecum is predisposed to perforation as the intraluminal pressure increases, such as in colic mechanical obstruction as discussed above, or in idiopathic cecal distension. Stercoral colitis can lead to highly serious stercoral peritonitis. Stercoral peritonitis mostly affects elderly as well as laid-up patients. The perforation is typically located on the antimesenteric border of the intestine, which is less vascularized than the mesenteric border and more sensitive to mechanical constraint (Facy et al. 2007). Stercoral peritonitis is typically located in the sigmoid colon or upper rectum as a fecaloma more frequently develops in the distal colon. The diagnosis is made upon the presence of colic distension by fecal material and the presence of intraperitoneal material. The prognosis of stercoral peritonitis is very severe.

2.2.6 Rectal Perforation

Rectal perforations are mostly related to a foreign body, or from iatrogenic origin complicating a surgical or endoscopic gesture. They can also occur following a cleansing enema or maneuver to extract fecaloma with the constitution of a stercoral peritonitis (Fig. 4).

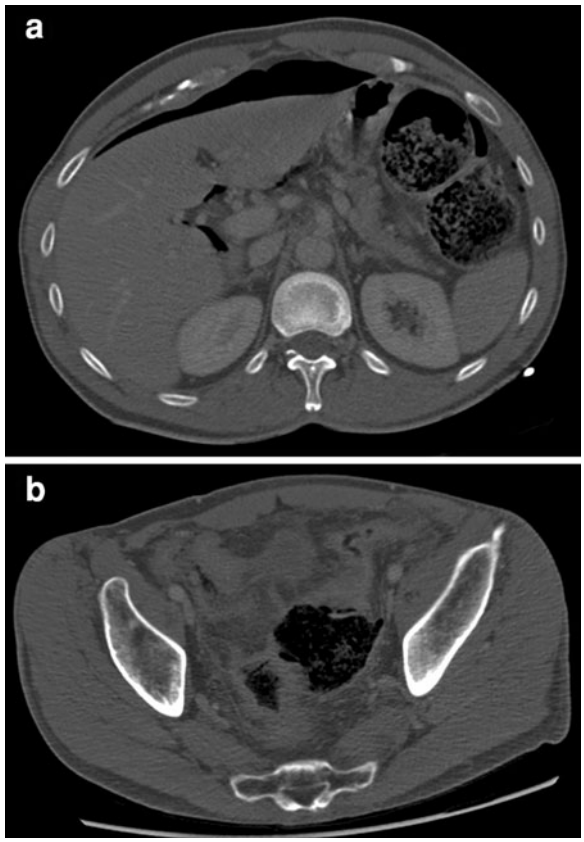


Fig. 4 Iatrogenic perforation located at the junction between the sigmoid colon and the rectum. The upper axial slice (**a**) shows a free pneumoperitoneum, and the lower axial slice (**b**) shows stool outside the colic tract. At surgery, the perforation complicating a cleansing enema was located at the junction between the sigmoid colon and the rectum

3 Clinical Findings

3.1 Peritoneal Syndrome

The onset is most often characterized by a syndrome of acute abdominal pain, sometimes "stab"-like. Pain, initially localized, quickly becomes generalized and increased during early and abundant vomiting episodes. Transit modifications (subocclusion) occur later.

General signs depend on the cause of the perforation and time elapsed from the perforation. They are typically dependent on the time of onset, and include fever, tachycardia, dyspnea, and oliguria. Septic shock with multiple organ failure can occur, especially with elderly and immunosuppressed patients. Patients may present with a grayish complexion, and hollow eyes (peritoneal face).

On examination, the pathognomonic sign is the contracture of the abdominal wall. In a lean and fit patient, muscles are visible, and the abdomen stops moving with respiration. The contracture is spontaneous, permanent, painful, and invincible. It is typically generalized to the entire abdomen, sometimes predominant at the site of the causative lesion. It must be appreciated on the basis of the musculature and the age of the subject. It can therefore be very discrete, even absent in elderly patients. It can be masked by analgesic treatment such as morphine. Pelvic touch is very painful, because of the peritoneal irritation.

3.2 Clinical Presentation According to the Cause

Several clinical and anamnestic signs can indicate the site of intestinal perforation. Ulcerous perforations affect men more than women, with the same incidence with regard to age, whereas small bowel perforation, appendiceal perforation, and colic perforations mostly affect the elderly.

In esophageal perforations, pain is localized to the thorax base or the epigastrium. Vomiting, cervical subcutaneous emphysema, and respiratory distress are late presentations. Two signs with a strong orientation value are the rise of pain in a context of violent vomiting and the patient's background, including ulcer history, neurologic history, and alcohol intake.

In gastroduodenal ulcer perforations, the site of pain—epigastric at first, which can radiate to the iliac fossa or to the shoulders—the epidemiologic context—intake of drugs with gastric wall toxicity, such as anti-inflammatory drugs, which are responsible for about half of ulcer perforations (Ramakrishinan and Salinas 2007), a great stress (recent surgery), and a young age orientate one toward diagnosis. In the case of frequent ulcerous attacks, adherences with neighboring organs can form around the first duodenum (liver, gallbladder, and colon). In this case, the perforation does not manifest itself with acute-generalized peritonitis symptoms but rather as acute pain localized in the right upper quadrant.

In small bowel perforations, the peritoneal syndrome is in general less obvious, the rise of the pain is less sudden, a fever is found in half of cases, and symptoms of obstruction can lead to a mechanical obstruction of the small bowel, in a context of infection.

Appendiceal perforations complicate from 18 to 35% of cases of appendicitis, appendicitis being the most frequent cause of abdominal emergency in a surgery department. Differentiating between appendicitis and complicated appendicitis with perforation is challenging when the clinical features are compatible with appendicitis. Flamant's criteria (Flamant 1995), used to postpone an eventual hospitalization (namely, lack of defense, lack of fever beyond 38°C, and lack of hyperleucocytosis), typically allow a perforated appendicitis to be excluded.

Colic perforations are in general complications of diverticular sigmoiditis and colic tumors. They thus occur in the elderly, with less sudden clinical presentation and symptoms of abdominal infection in case of a diverticulitis, or episodes of subocclusion with altered general state in case of a colon cancer.

4 CT Findings

4.1 Direct Findings

Direct visualization of a localized rupture of the digestive tract wall and of a communication between the intestinal content (air or liquid) and the perintestinal atmosphere is pathognomonic of the site of intestinal perforation (Fig. 5). It is a CT finding whose visualization can be favored by using fine sections and multiplanar reconstructions.

4.2 Indirect Findings

4.2.1 Pneumoperitoneum

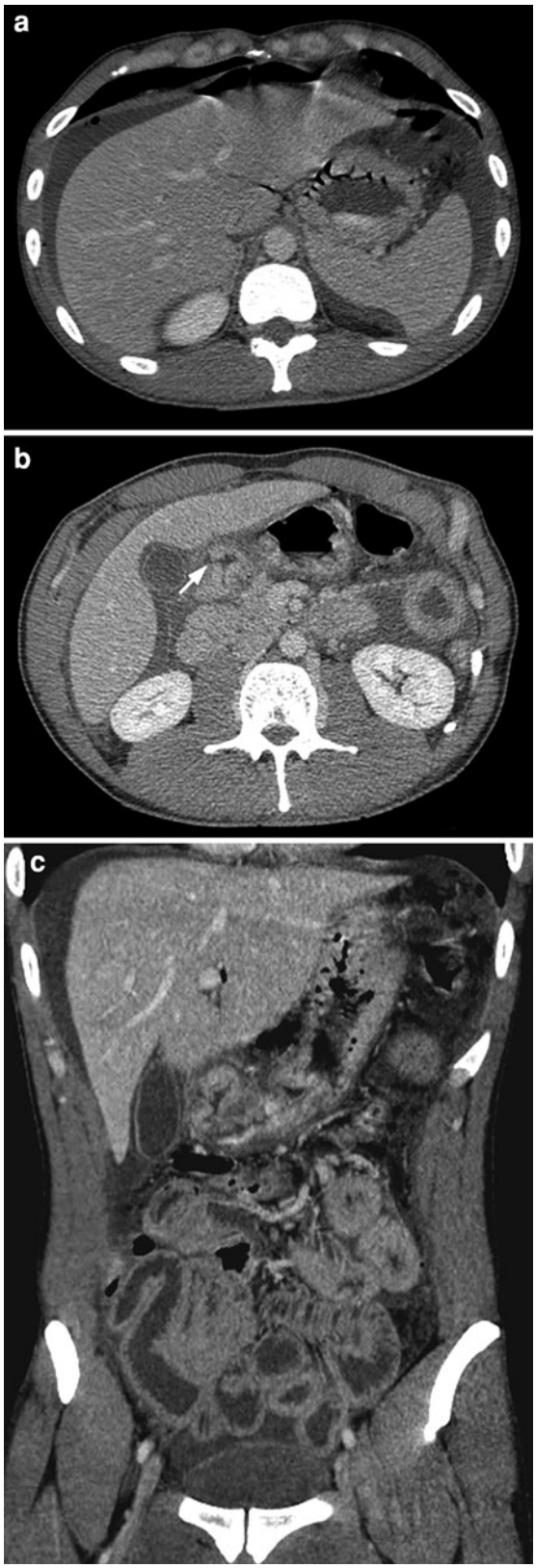
A pneumoperitoneum is the cardinal sign of digestive perforation (Cho and Baker 1994). On CT, the pneumoperitoneum is easily visible as large windows and fine slices are used (Rubesin and Levine 2003; Grassi et al. 2004). The presence and the amount of free air in the peritoneum depend on the anatomical site and extension of the perforation, on preexisting intestinal distension, and moreover, on the time elapsed from the perforation to the scan. The site of a pneumoperitoneum, whether localized or not localized, points toward a perforation of the adjacent bowel, particularly when extraluminal free air is in little abundance (Kim et al. 2009).

4.2.2 Fatty Infiltration

A localized fatty infiltration adjacent to an intestinal segment points, with a less valuable diagnostic value than for the pneumoperitoneum, toward the perforation of this particular intestinal segment.

4.2.3 Intestinal Wall Thickening

A localized thickening of the intestinal wall in acute abdomen syndrome with a pneumoperitoneum orientates one toward the cause (by analyzing the thickening features) and the site of the perforation. Inversely, an extended thickening of the intestinal



◀**Fig. 5** Small bowel thickening consecutive to a gastroduodenal ulcer perforation. The upper axial slice (**a**) shows a free pneumoperitoneum and peritoneal fluid, whereas the duodenal discontinuity (*arrow*) is well seen on the slice in (**b**). Note the thickening of the small bowel wall seen on the axial view (**b**) and coronal reformatting (**c**)

wall has a poor diagnostic value and can simply be the consequence of peritonitis (Fig. 5).

4.3 CT Findings According to the Cause

4.3.1 Esophageal Perforation

CT is used to look for indirect signs: a mediastinal posterior collection of liquid revealing a mediastinitis, a posterior pneumomediastin that can diffuse to the retroperitoneum, parietal signs as a localized thickening of the esophageal wall. Exceptionally, it will identify parietal wall discontinuity, as a direct sign of the perforation.

4.3.2 Gastroduodenal Perforation

Identification of a rupture in the intestinal wall, most commonly in the duodenal bulb or prepyloric region, is a direct sign of a perforated gastroduodenal ulcer (Fig. 6).

Indirect CT findings of an ulcer perforation typically are the presence of an abundant pneumoperitoneum, free air around the falciform ligament, perigastroduodenal fatty infiltration, interduodenopancreatic fluid, and gastroduodenal thickening (Chen et al. 2001; Ongolo-Zogo et al. 1999).

Considering the predominant bulbar localization of duodenal ulcer, the perforation is intraperitoneal. A retroperitoneal localization, which is visualized by imaging as leaking of extraintestinal gas, in the right anterior pararenal space indicates a duodenal perforation beyond the bulbar segment, often related to a duodenal diverticulum or a perforation from GI endoscopy (Yagan et al. 2009).

The perforation is not always related to an ulcer. It can originate from a tumor. Such a diagnosis must be suspected in patients with an important irregular thickening of the gastric wall.

The perforation can be limited to the gastric wall, but can be extended to the colic wall with formation of a gastrocolic fistula. This event is rare. It can be found in tumoral gastric perforations, in Crohn diseases with gastric and colic involvement, and

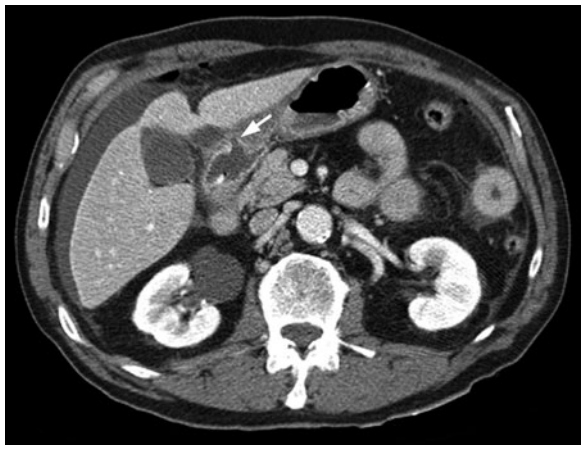


Fig. 6 Perforation due to an antropyloric ulcer. The discontinuity of the anterior wall of the gastric wall is well identified (*arrow*) on these 3-mm axial reconstructions

exceptionally in certain ulcers affecting patients treated with nonsteroidal anti-inflammatory drugs (Ramakrishinan and Salinas 2007).

As described above, perforations of a duodenal diverticulum are mostly retroperitoneal. In a small series including eight cases, both a retroperitoneum and retroperitoneal fatty infiltration were found in all cases, with an associated pneumoperitoneum in three of the cases (Fig. 7). A second or third duodenum localized diverticulum was identified in seven of the cases (Ames et al. 2009).

4.3.3 Small Bowel Perforation

CT findings of small bowel perforation are findings of air bubbles within the mesentery fluid collection with a pneumoperitoneum (Kimchi et al. 2002), localized fatty infiltration in the mesentery, or a localized. Thickening of the small bowel wall often exists, with little prognostic value with respect to the site, as the thickening could result from peritonitis, hence indicating either a colic or a gastroduodenal perforation.

Small bowel perforation complicating a bowel obstruction CT (Catel et al. 2003) will show signs of mechanical obstruction, signs of incarceration and strangulation with a localized edema, a venous congestion, or and mostly, an intestinal wall thinning or an intestinal pneumatosis revealing an infarcted intestine.

Acute ischemia of the mesentery CT will show parietal findings with a localized or not localized defect enhancement of the intestine wall revealing parietal distress, and vascular signs, thrombus, most often proximal, in the superior mesenteric artery, or embolus sited on arterial bifurcation.

Crohn disease CT shows a thickening of the intestinal wall, a marked fatty infiltration (sclerolipomatosis), and an increased parietal vascularization. Perforation in a Crohn disease setting complicates an evolved Crohn disease, with common fistula and abscess formation (Furukawa et al. 2004) (Fig. 8).

Digestive lymphoma In addition to a small bowel parietal thickening, criteria favoring a lymphoma are findings of an ectatic digestive lumen, of adenomegaly, or the extent of thickening. However, in type T lymphoma, moderate thickening and missing lymph nodes can make a differential diagnosis with inflammatory involvement difficult (Byun et al. 2003).

Small bowel diverticulitis CT will show signs of complicated jejunal diverticulitis, or signs of Meckel diverticulitis, by individualizing thickened diverticula with peridiverticular fatty infiltration, and the presence of small pneumoperitoneum bubbles more often localized around the diverticulum than free, and close to the anterior parietal peritoneum (Hibben et al. 1995). A Meckel diverticulum containing air, liquid, or fecal material is sometimes visualized. The diverticulum wall can be thickened (Fig. 3) or with no enhancement, and even interrupted, revealing a perforated diverticulum.

Digestive tuberculosis Tuberculous perforations affect the terminal ileus, with a thickened ileocecal valvule and a thickened ileal and cecal wall. A small bowel obstruction is often associated, as a consequence of the stenosis of the terminal ileus. Enlarged lymph nodes with a hypodense center may be seen. The main differential diagnoses are Crohn disease and ileocecal lymphoma.

Jejunal ulcer In jejunal ulcer perforations occurring in the setting of Zollinger–Elisson syndrome, scan findings include a localized defect of the digestive wall, sited at a proximal jejunal loop, with associated extraluminal air bubbles, and with a localized infiltration of fat close to the pathologic loop, as seen in gastroduodenal ulcer perforations.

Foreign body In addition to indirect signs of perforation, such as localized extradigestive collection,

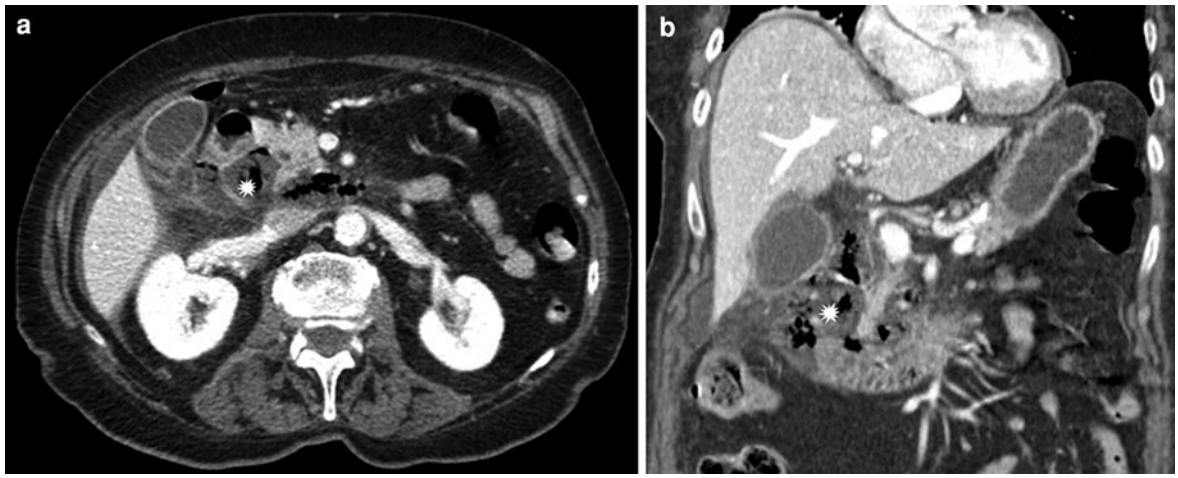


Fig. 7 Perforation of a duodenal diverticulum. Axial (**a**) and coronal (**b**) views show the diverticulum (*star*) adjacent and posterior to the second part of the duodenum on the axial slice. There is a rounded collection with air and fluid along the lateral surface of the diverticulum. Note also the presence of both a retropneumoperitoneum and a pneumoperitoneum

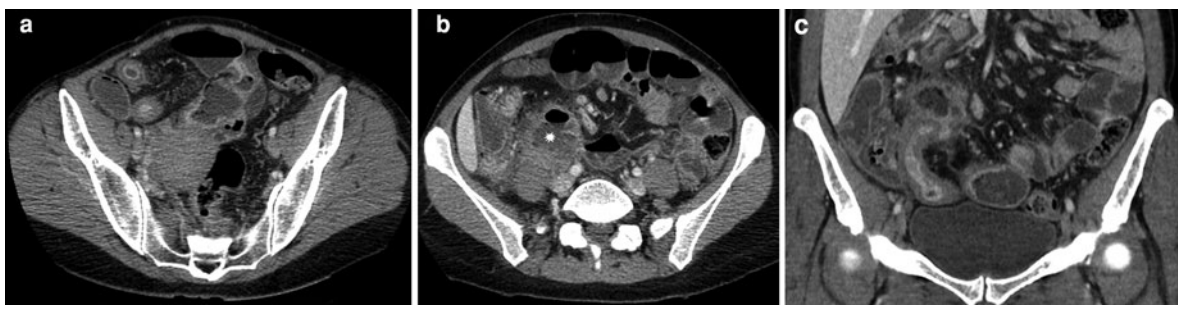


Fig. 8 Small bowel perforation complicating a Crohn disease. The lower axial slice (**a**) and the coronal view (**c**) show Crohn disease findings with wall thickening of the terminal ileum, target sign on the distal ileum, and sclerolipomatosis. The upper axial slice (**b**) shows an abscess (*star*) located in front of the right psoas muscle

fatty infiltration, and thickening of the involved digestive wall, direct visualization of a foreign body, either calcified or metallic, through the digestive wall allows a diagnosis to be made with certainty (Goh et al. 2006) (Fig. 9).

Vascularitis In polyarteritis nodulosa, the scan will show digestive wall thickening related to ischemia, imaging of thrombosis of the portal venous system, and signs of hepatic, splenic, or renal infarction. It will also detect signs of pancreatitis, or cholecystis. In Behcet syndrome, ulcers are reliably displayed by digestive opacification. On CT, digestive wall thickening is important, sometimes being circumferential and mass-shaped, and sometimes being voluminous, which can be mistaken for digestive tumors (Pagnoux et al. 2003). As compared with Crohn disease, ulcerations are wider and deeper, and peridigestive inflammatory infiltration is less important, with little or no sclerolipomatosis.

4.3.4 Appendiceal Perforation

Semiology, which allows complicated and uncomplicated appendicitis to be distinguished, is based on the presence of abscesses, phlegmon, extraluminal air, extraappendiceal stercolith, and a defect in enhancement of the appendiceal wall and ileus. All such signs are in favor of a perforated appendicitis. A free pneumoperitoneum is very rare in appendiceal perforations (Fig. 10).

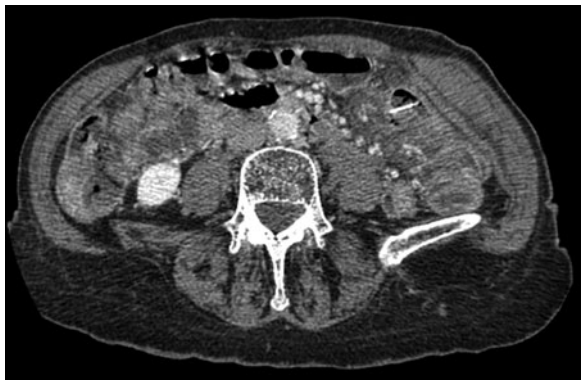


Fig. 9 Foreign body going through the small bowel wall. The foreign body is clearly seen and was related to a rabbit bone

4.3.5 Colic Perforation

CT is now the reference standard for evaluating complications of colic diverticular disease (Loiseau et al. 2005; Zins et al. 2007). It individualizes signs of diverticulitis, with thickening of the colic wall commonly involving the sigmoid, perisigmoid fatty infiltration, and diverticula. It shows signs of perforation, such as extraluminal air, localized in the mesosigmoid region, or at a distance in the pelvis, or as a free subdiapharagmatic pneumoperitoneum which may be very limited (Fig. 11) because the perforation is closed by the adjacent inflamed mesosigmoid. It shows pericolic or pelvic abscesses, further evaluating their size, as well as extradigestive materials, revealing a stercoral peritonitis.

CT findings (Lorhmann et al. 2005) correlate well with surgical findings; hence, complications can be graded according to the Hinchey grading system (Hinchey et al. 1978).

- Stage I, in which there is a phlegmon or a pericolic abcess
- Stage II, in which either the phlegmon or the pericolic abscess is voluminous, or there are pelvic or retroperitoneal or abdominal abscesses at a distance
- Stage III, with a purulent generalized peritonitis
- Stage IV, with a stercoral peritonitis

The diagnosis of tumoral colic perforation has to be proposed when extraluminal air is adjacent to the thickened colic wall, revealing a tumor. Common findings are major peritumoral fatty infiltration and small amounts of extraluminal air. In contrast, in cecal diastatic perforations, with accompanying CT signs of large bowel obstruction and a distended colon, the pneumoperitoneum is often highly abundant. A cecal parietal pneumatosis is sometimes associated with such an abundant pneumoperitoneum, pointing toward the site and mechanism of perforation, although not specific to cecal transmural distress in the setting of colic mechanical obstruction (Taourel et al. 2004).

In stercoral perforations, diagnosis is made upon a coexisting free peritoneum and a very important colic distension by materials. The colon wall is distended and sometimes thickened, and pericolic fat is infiltrated (Heffernan et al. 2005).

In perforation complicating an ischemic colitis, CT frequently shows a lack of enhancement of the bowel wall (Fig. 12)

4.3.6 Rectal Perforation

On CT, extraluminal air in the perirectal fat is the most frequent finding. Additional findings include presacral fluid, extraluminal feces, and rectal wall thickening (Zissin et al. 2008).

5 CT Pitfalls

The main potential error in diagnosing a pneumoperitoneum by CT is confusion between intraluminal and extraluminal air, and more particularly, doubt between intraperitoneal and preperitoneal air in the case of a small anterior pneumoperitoneum, the latter diagnosing issue especially arising in abdomen trauma.

Even when optimum imaging examination (CT with fine sections and a wide window) is used, a pneumoperitoneum may not be present in a true digestive perforation (false negative) (Grassi et al. 2004). In contrast, a pneumoperitoneum may be present in the absence of a digestive perforation (false positive).

A false negative can be related to digestive perforation without pneumoperitoneum formation. This has been well demonstrated in a study (Grassi et al. 2004) using a series of gastroduodenal perforations. A pneumoperitoneum could not be found by CT in some cases, but was detected in a few of the cases by delayed scans (beyond 6 h). Nevertheless, a free pneumoperitoneum can be delayed as well. A localized pneumoperitoneum adjacent to the perforation is found in roughly all nontraumatic perforations of

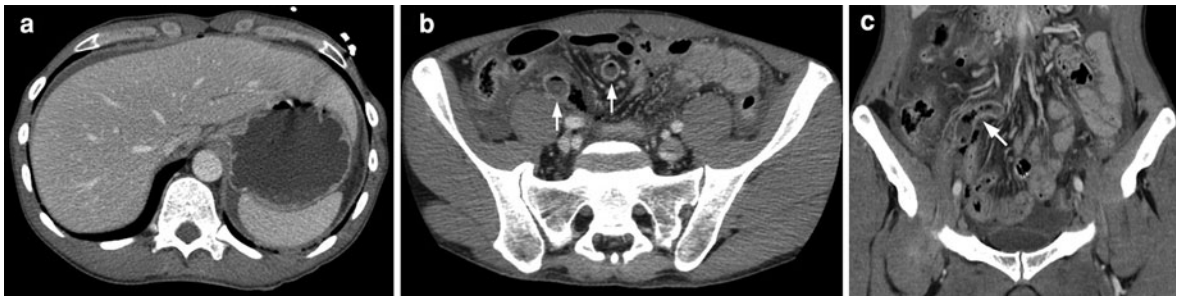


Fig. 10 Peritonitis consecutive to an appendiceal perforation. The upper axial slice (**a**) shows a free pneumoperitoneum. The lower axial slice (**b**) shows a collection in the right lower quadrant with dilatation of the appendix (*arrow*) and important enhancement of the appendix wall, which is thickened. The coronal view (**c**) shows the appendicitis in its length

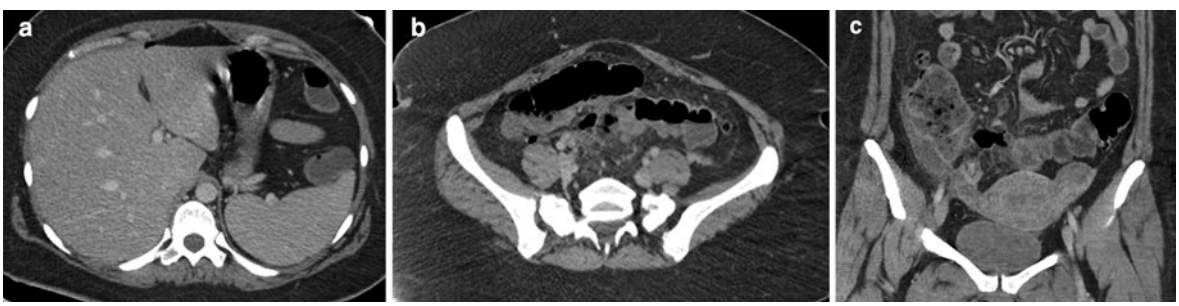


Fig. 11 Perforation of sigmoid diverticulitis. The upper axial slice (**a**) shows a free pneumoperitoneum, whereas the lower slice (**b**) and coronal reformatting (**c**) show a thickening of the sigmoid wall with fat stranding. Note also the presence of air bubbles outside the bowel

nonappendiceal origin (Imuta et al. 2007) involving intraperitoneal segments of bowel.

Excluding a pseudopneumoperitoneum notably related to preperitoneal air, with air confined to the inner layer of the abdominal wall and external to the parietal peritoneum, a false positive correspond to the presence of a documented pneumoperitoneum, with no digestive perforation. A false positive can originate from the thorax, digestive organs, or gynecologic organs (Table 1) (Catel et al. 2003). Finally, a retropneumoperitoneum can be related to a diffuse pneumomediastinum.

For the diagnosis of the perforation's cause, in a patient with a pneumoperitoneum, the identification of a thickening of the bowel wall pointing toward the site of the perforation may be misleading: a thickening of the bowel may be the consequence of the peritonitis due to the perforation of another part of the GI tract. This specifically involves the small bowel with thickening of the small bowel wall, which may make differential diagnosis between small and large bowel perforation difficult (Fig. 13). In the same way, the site of the pneumoperitoneum does not point toward the site of the perforation when the pneumoperitoneum is abundant, and this may correspond to a gastroduodenal or a colic perforation (Fig. 14).

6 CT Accuracy

CT is very accurate for pneumoperitoneum identification, even if it is poorly abundant or localized. Reading requires the use of wide windows. A 1-mm section can be useful for identification of a small pneumoperitoneum. CT is likely to very accurately diagnose a pneumoperitoneum, as long as it exists.

Classic indirect CT signs pointing toward an ulcer perforation are the presence of an abundant pneumoperitoneum, air located around the round ligament, outlining of the falciform ligament, infiltration of perigastroduodenal fat, and the finding of a gastroduodenal thickening. In a retrospective study

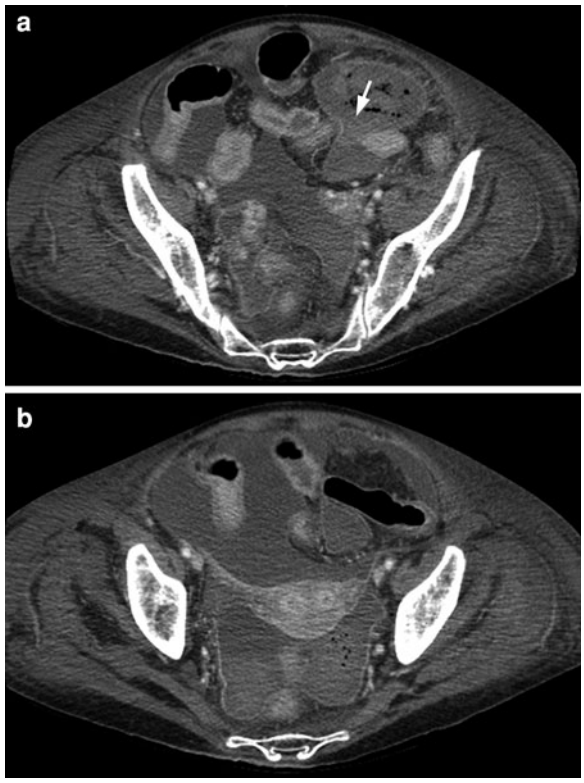


Fig. 12 Ischemic colitis complicated by perforation. The upper axial slice (**a**) shows a collection in the peritoneum with the peritoneal membrane, which is enhanced. The descending colon is thickened without enhancement of its posterior wall (*arrow*). The lower axial slice (**b**) shows some air bubbles within the collection located in the Douglas pouch. Note also the enhanced peritoneum

Table 1 Causes of pneumoperitoneum without bowel perforation

Pseudopneumoperitoneum on abdominal plain film
Lucency under the diaphragm
Fatty area under the diaphragm (more common in obese patients or in patients with corticoid)
Nonperitoneal gas
Properitoneal gas
Gas within lung trapped under a lung collapse
Air within the bowel
Chilaiditi syndrome
Stomach closed to the diaphragm
Emphysematous cholecystitis or pyelonephritis
True pneumoperitoneum
Thoracic causes
Diffusion of pneumothorax or pneumomediastinum
Abdominal causes
Abdominal surgery
Perioneal dialysis
Endoscopy: pneumoperitoneum is due to the diffusion of intraluminal air
Gastroparesy
Pneumatosis cystoides intestinalis
Female genital tract causes
Hysterectomy
Sexual intercourse or cunnilingus
Pelvic examination
Athletic activities such as waterskiing

(Ghekiere et al. 2007a) including 81 digestive perforations, about half of which related to a gastroduodenal perforation, we demonstrated the lack of specificity of indirect signs. The presence of air around the ligamentum teres or visualization of the falciform ligament outlined by some air could be related to a perforation from any other origin when the pneumoperitoneum is abundant. A reactive gastroduodenal thickening could also indicate peritonitis from another cause. In our series, the best indirect sign of a gastroduodenal perforation was the presence of fluid localized between the duodenum and the head of the pancreas. This sign had a positive predictive value greater than 90% for diagnosing a perforated ulcer. In contrast, the presence of an abundant pneumoperitoneum or a pneumoperitoneum outlining the falciform ligament and also a pneumoperitoneum within the lesser sac had a positive predictive value ranging from 40 to 52% for the diagnosis of a gastroduodenal ulcer perforation. More recently, a Korean study has confirmed that evaluation of the site of the pneumoperitoneum, and more specifically the presence of periportal free air around the round or falciform ligament were only ancillary findings in the diagnosis of a gastroduodenal perforation (Choi et al. 2009). In contrast, a pneumoperitoneum only localized within the mesentery points toward a small bowel perforation, whereas a free pneumoperitoneum localized within the submesocolic space points toward a colic perforation.

Direct visualization of the site of perforation is a major advantage of multidetector CT, owing to thin sections (Fig. 15) and multiplanar reconstructions. In a study by Chen et al. (2001), analyzing ulcer

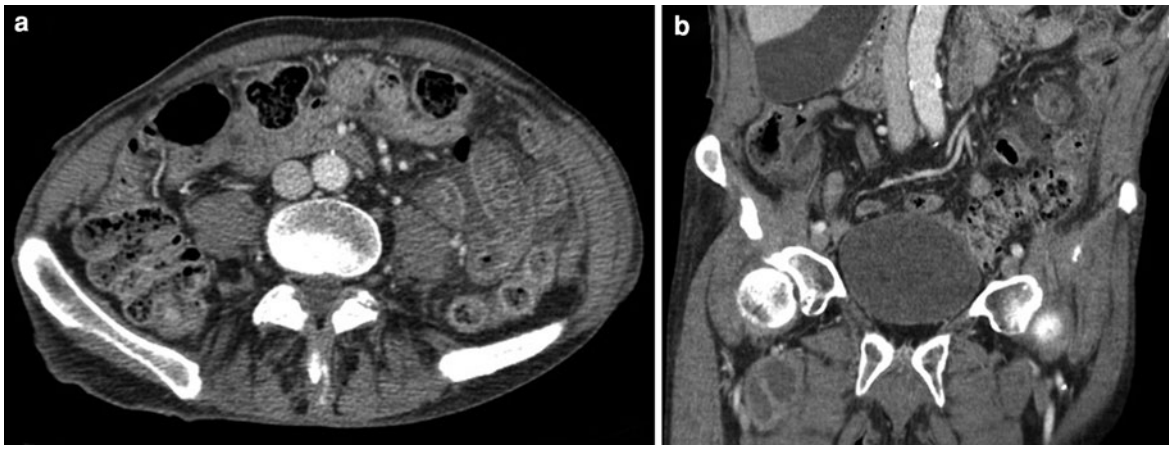


Fig. 13 Sigmoid perforation with reactive thickening of the small bowel. Axial slices (**a**) show thickening of the small bowel wall, peritoneal fluid, and some extraluminal air bubbles. On the coronal reformatting (**b**), sigmoid diverticula are well identified with some slight thickening of the sigmoid wall. Surgery showed perforated sigmoid diverticulitis

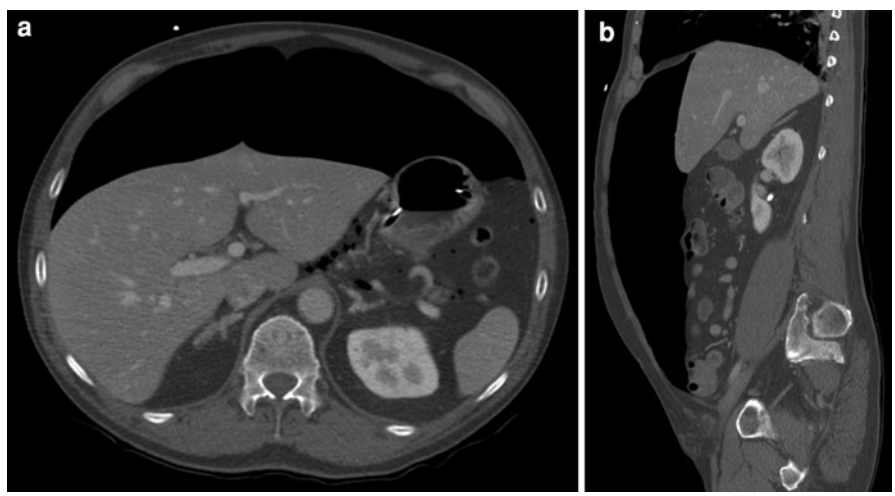


Fig. 14 Big pneumoperitoneum without any value for the perforation localization. The axial view (**a**) and sagittal reformatting (**b**) show a big pneumoperitoneum in a patient hospitalized in an intensive care unit. There is no argument for a gastric versus a colic perforation. At surgery, there was a tear in the lesser curvature of the stomach complicating cardiac massagea

perforations, discontinuity of the digestive wall was never visualized, as CT was performed using centimeter sections. In another study, performed using 3-mm sections, discontinuity of the digestive wall was visualized in 60% of patients with a perforated ulcer (Ongolo-Zogo et al. 1999). In a retrospective series of 15 perforated gastroduodenal ulcers, Cazejust et al. (2007) directly individualized the parietal rupture in eight cases by using a 16-detector CT scanner and millimeter sections.

In our experience (Ghekiere et al. 2007b), in a retrospective study including 40 patients with nontraumatic digestive perforations from various sites and causes, the utility of thin sections and reconstruction significantly allowed direct identification of the route of perforation. Identification was possible in 5–20% of the cases, depending on the readers, using 5-mm sections in the axial plane, in 28–48% of the cases using both 1- and 5-mm sections, and in 43–53% of the cases using both thin slices and sagittal coronal and axial planes. Coronal plane sections proved useful in detecting perforations of both the superior and the inferior wall of the antrum and the bulb or first part of the duodenum. In a study by Hainaux et al. (2006) including 85 patients with various causes of both trauma and nontrauma perforations, the route of perforation was visualized in 29 cases out of 85 (34.1%), using a four-detector CT

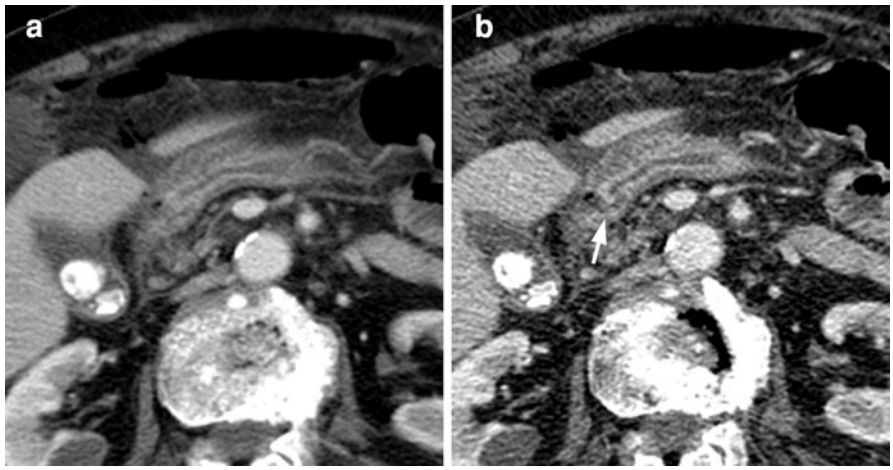


Fig. 15 Impact of slice thickness on the identification of a perforated gastrointestinal ulcer. The antral lumen and the defect in the antral wall are better seen on the 1-mm-thick slice (**b**) than on the 5-mm-thick slice (**a**)

scanner and 2.5-mm sections. These authors also illustrated the potential utility of multiplanar reformatting for identifying the parietal rupture.

CT semiology in diagnosing appendiceal perforation has been validated by three retrospective studies following the same method. The results are summarized in Table 2. All three studies found specific signs of appendicitis, although with a relatively low sensitivity, especially for one specific case of extra-appendiceal stercolith. A localized defect in enhanced appendiceal wall was reported as the most sensitive sign of appendicitis (Bixby et al. 2006). However, thin sections, which are more sensitive to artifacts related to surrounding bone structures or digestive peristaltic movement, could be responsible for a false positive, owing to visualization of a localized digestive wall defect, with no true perforation. Findings of peritoneal fluid, either free or localized, are not a specific sign of appendiceal perforation.

7 CT Impact

The advantages in identifying the site of perforation in patients presenting with digestive perforation deserve to be discussed, as it can be advocated that a peritonitis from digestive perforation may require, whatever its site, emergency surgery. Nevertheless, the type of surgery depends on the site and cause of perforation.

In patients with gastroduodenal ulcer perforation, laparoscopy is an interesting choice, as shown in a number of randomized studies comparing laparoscopic with laparotomic repair of perforated ulcer (Lau et al. 1996; Druart et al. 1997; Siu et al. 2002). A laparoscopic intervention is often recommended in gastroduodenal ulcer perforations of the anterior or lateral site, whereas perforations of the posterior face of the stomach or duodenum could be better managed by regular open abdomen surgery owing to difficult access to the posterior wall of the antropylorobulbar region in peritoneoscopy (Mabrut et al. 2007).

Unlike gastroduodenal ulcer perforations, small bowel perforations are best treated by laparotomy.

For colic perforation, perforations from tumor will be repaired by open abdomen surgery. In contrast, management of perforations from sigmoid diverticulitis depends on the evolution of the disease, as well as on the surgery team practice. The French Society of Gastroenterology and Surgery has given recommendations for management of diverticulitis based on the morphologic assessment of complications by means of a CT scan and Hinchey classification:

- Hinchey grading stage I (phlegmon or colic abscess): After medical treatment and/or CT-guided drainage, resection and anastomosis is the recommended surgical procedure, eventually associated with a protective colostomy. A laparoscopic procedure may be used.
- Hinchey grading stage II: CT-guided drainage and sampling for further microbiology analysis is recommended for abscesses greater than 5 cm. In the case of efficient drainage, primary resection followed by anastomosis is recommended. In the case of a failed drainage or if drainage is not possible, surgical treatment with anastomosis,

Table 2 Accuracy of computed tomography for the diagnosis of perforation in appendicitis

	Horrow et al. (2003)	Bixby et al. (2006)	Tsuboi et al. (2008)
Number of patients	94	244	102
Appendiceal perforation (%)	41.4	25.4	39.2
Slice thickness (mm)	5–10	3.2	2–3
Ileus			
Sensitivity (%)		53	
Specificity (%)		93	
Extraintestinal gas			
Sensitivity (%)	36	35	22.5
Specificity (%)	100	98	100
Abscess			
Sensitivity (%)	36	34	37.5
Specificity (%)	100	99	100
Localized defect of enhancement			
Sensitivity (%)	64	64	95
Specificity (%)	100	80	96.8
Extraluminal appendicoloth			
Sensitivity (%)	20		32.5
Specificity (%)	100		100
Phlegmon			
Sensitivity (%)	46.5		40
Specificity (%)	94		95

associated or not associated with protective colostomy is recommended.

- Hinchey grading stage III (generalized purulent peritonitis): A sigmoid resection is recommended. Depending on both the local and the general state of the patient, either resection and anastomosis (associated or not associated with protective ostomy) or the Hartmann procedure will be chosen.
- Hinchey grading stage IV (stercoral peritonitis): The Hartmann procedure is the reference intervention.

A dedicated CT assessment of a complicated diverticulitis not only allows for a modified emergency management, but is also required for later management, as a prophylactic surgery is recommended in the case of a diverticulitis attack with serious signs on the CT scan (namely, abscess or extraluminal leaking of air or contrast liquid).

Similarly, preoperative diagnosis of appendiceal perforation is valuable as it can affect the prognosis of appendicitis, as well as the therapeutic management. Preoperative findings of a localized appendiceal perforation with peritonitis is a reason for switching from an appendectomy by celioscopy to a laparotomic procedure (Liu et al. 2002). Furthermore, medical therapy with intravenous antibiotics combined or not combined with percutaneous drainage may not only limit the extent of surgery, but may also allow, at least, one-step surgery to be undertaken. Finally, medical treatment of uncomplicated appendicitis represents a therapeutic option likely to develop in the future. Indeed a recent analysis of the disconnect between the incidence of nonperforated and perforated appendicitis with a decrease of nonperforated appendicitis and an increase of perforated appendicitis has some implications for pathophysiology and potentially for management of an appendicitis. This may advocate individualization of two different conditions, a perforated appendicitis not necessarily evolving from an appendicitis that has not been surgically treated (Livingston et al. 2007).

8 Diagnosis Strategy

In a patient with a suspected digestive perforation, the diagnostic strategy relies on the wide use of CT, which is the reference examination in management of acute abdomen. APF is only valuable when it shows a pneumoperitoneum. However, a digestive perforation can definitively not be excluded upon normal APF radiography. In practice, three clinical situations can be individualized:

1. There is no orientation element in the anamnesis.
2. An anamnestic context exists, allowing the diagnosis to be orientated.
3. Acute abdomen symptoms arise either early after surgery or after a coloscopy.

8.1 No Orientation Element in the Anamnesis

This is the most frequent clinical situation. The patient has no particular history, or the history is either unknown or not reported at the time of examination. Before abdominal symptomatology highly evoking a digestive perforation, APF radiography will confirm a pneumoperitoneum. Nevertheless, in most cases, a CT scan will be performed to identify the site and cause of the digestive perforation.

8.2 Diagnosis Oriented by Anamnestic Context

An anamnestic context exists, which can be a known bowel or colon disease, or intake of GI-toxic drugs such as anti-inflammatory drugs (corticoids, acetylsalicylic acid). When a gastroduodenal ulcer perforation is suspected because of an acute gastric pain combined with anti-inflammatory drug intake, APF detection of a pneunoperitoneum will suffice for further therapeutic management. In the case of a negative APF finding, endoscopy or a CT scan seeking a complicated digestive disease or differential diagnoses will be discussed.

Besides a clinical context of perforated ulcer, associated with an APF finding of a pneumoperitoneum, CT will be used to assess the known disease, for instance, Crohn disease, and its complications.

8.3 Postoperative or Postendoscopy Situations

In postoperative follow-up of abdominal surgery, abdominal pain may indicate a complication from surgery such as fistula formation. The presence of a pneumoperitoneum questions its origin: a residual postoperative pneumoperitoneum or a pneumoperitoneum revealing either an anastomotic fistula in the case of intestinal anastomosis or an iatrogenic perforation of the GI tract. The extent of the pneumoperitoneum and the presence or absence of localized inflammatory signs can orientate the diagnosis toward either a residual postoperative pneumoperitoneum or a GI tract perforation. Postoperative pneumoperitoneums are very frequent, with CT detection of a postoperative pneumoperitoneum reported in up to 44–87% of patients after abdominal surgery (Gayer et al. 2000; Earls et al. 1993). They are typically poorly abundant, less than 10 ml in most patients, with lean patients and men presenting with a more abundant pneumoperitoneum. However, they can be larger in case of surgical drainage (Gayer et al. 2000). The abundance of such a postoperative pneumoperitoneum decreases with time.

In clinical practice, if postoperative perforation is suspected for a patient presenting with a pneumoperitoneum, the diagnosis and therapeutic management will depend on the type of surgery and the risk of GI tract lesions, with the existence or no existence of intestinal anastomosis that may form a fistula, on the extent of the pneumoperitoneum related to the time since surgery and to a possible drainage, and finally on the presence of localized inflammatory signs, orienting the diagnosis toward GI tract lesions. In doubtful cases and if the patient's clinical condition allows it, CT may be repeated within 8–12 h, with an increase or a decrease of the pneumoperitoneum being strong arguments in favor or not in favor of a postoperative perforation.

In postcolonoscopy follow-up, GI tract perforation is a very rare complication (Anderson et al. 2000; Tulchinsky et al. 2006), complicating 0.005–0.002% of diagnostic colonoscopies and 0.2–0.4% of colonoscopies with polypectomy. Nevertheless, the development of mucosectomy has increased the risk of perforation. The mechanisms of perforations from colonoscopy are various and include hyperpressure either from the endoscope or from excessive

insufflations, and lesions from parietal electrocoagulation due to polypectomy. Perforations generally affect abnormal, inflammatory, and/or stenosed colon. The diagnosis of postendoscopy colic perforation will be made by the presence of a pneumoperitoneum. The therapeutic management, whether surgical or not, will depend on both the presence of peritoneal signs and colic preparation. Indications for surgical treatment include poor colic preparation, a blunt parietal rupture on colonoscopy or CT signs of peritonitis, or the presence of an obstruction downstream of the perforation, and, obviously, deterioration of clinical features when conservative treatment was chosen. Of note, such a pneumoperitoneum revealing a macroscopic perforation is only of value when there are acute symptoms after colonoscopy. Indeed, according to a few authors, a pneumoperitoneum can be related to up to 1% of colonoscopies. Although this percentage is likely to be overestimated (Pearl et al. 2006), a true benign pneumoperitoneum may form after colonoscopy, either from transparietal diffusion or from a microperforation favored by forced mobilization of the intestine and air insufflations, requiring no surgical treatment.

References

Ames JT, Federle MP, Pealer KM (2009) Perforated duodenal diverticulum: clinical and imaging findings in eight patients. Abdom Imaging 34:135–139

Anderson ML, Pasha TM, Leighton JA (2000) Endoscopie perforation of the colon: lessons from a 10-year study. Am J Gastroenterol 95:3418–3422

Behrman SW (2005) Management of complicated peptic ulcer disease. Arch Surg 140:201–208

Bixby SD, Lucey BC, Soto JA et al (2006) Perforated versus nonperforated acute appendicitis: accuracy of multidetector CT detection. Radiology 241:780–786

Byun HJ, Ah HK, Kim AY et al (2003) CT findings in peripheral T-cell lymphoma involving the gastrointestinal tract. Radiology 227:59–67

Catel L, Lefèvre F, Laurent V et al (2003) Occlusion du grêle sur bride: quels critères scanographiques de gravité rechercher? J Radiol 84:27–31

Cazejust J, Castaglioli B, Bessoud B et al (2007) Perforation gastro-duodénale: le rôle de la TDM multicoupe. J Radiol 88:53–57

Chen HS, Sheen-Chen SM (2000) Obtruction and perforation in colorectal adenocarcinoma: an analysis of prognosis and current trends. Surgery 127:370–376

Chen CC, Huang HS, Yang CC, Yeh YH (2001) The features of perforated peptic ulcers in conventional computed tomography. Hepatogastroenterology 49:1393–1396

Cho KC, Baker SR (1994) Extraluminal air diagnosis and significance. Radiol Clin North Am 32:829–844

Choi A, Jang KM, Kim MJ et al. (2009) What determines the periportal free air, and ligamentum teres and falciform ligament signs on CT: can these specific air distributions be valuable predictors of gastroduodenal perforation? Eur J Radiol. doi:10.1016/j.ejrad.2009.07.033

Chung SY, Ha HK, Kim JH et al (2001) Radiologic findings of Behçet syndrome involving the gastro-intestinal tract. Radiographics 21:911–926

Druart ML, Van Hee R, Etienne J et al (1997) Laparoscopic repair of perforated duodenal ulcer. A prospective multicenter clinical trial. Surg Endosc 11:1017–1020

Earls JP, Dachman AH, Colon E et al (1993) Prevalence and duration of postoperative pneumoperitoneum: sensitivity of CT vs left lateral decubitus radiography. AJR Am J Roentgenol 161:781–785

Facy O, Radais F, Chalumeau C (2007) Perforation stercorale du côlon. Physiopathologie et stratégie thérapeutique. Gastroenterol Clin Biol 31:1069–1070

Feliciano DV, Ojukwo JC, Rozycki GS et al (1999) The epidemic of cocaine-related juxtapyloric perforations. Ann Surg 229:801–806

Flamant Y (1995) Association de recherche en chirurgie et association universitaire de recherche en chirurgie. Douleurs abdominales aiguës de l'adulte. Encycl Med Chir (Paris), Gastro-entérologie, 9001-B-10

Furukawa A, Saotome T, Yamasaki M et al (2004) Cross-sectional imaging in Crohn disease. Radiographics 24:689–702

Gayer G, Jonas T, Apter S et al (2000) Postoperative pneumoperitoneum as detected by CT: prevalence, duration, and relevant factors affecting its possible significance. Abdom Imaging 25:301–305

Ghai S, Pattison J, Ghai S, O'Malley M, Khalili K, Stephens M (2007) Primary gastrointestinal lymphoma: spectrum of imaging findings with pathologic correlations. Radiographics 27:1371–1388

Ghekiere O, Lesnik A, Hoa D et al (2007a) Value of CT in the diagnosis of the cause of non-traumatic gastro-intestinal perforation. J Comput Assist Tomogr 31:169–176

Ghekiere O, Lesnik A, Millet I et al (2007b) Direct visualization of perforation sites in patients with a non-traumatic free pneumoperitoneum: added diagnostic value of thin transverse slices and coronal and sagittal reformations for multi-detector CT. Eur Radiol 17:2302–2309

Goh B, Tan YM, Lin SE et al (2006) CT in the preoperative diagnosis of fish bone perforation of the gastrointestinal tract. AJR Am J Roentgenol 187:710–714

Grassi R, Romano S, Pinto A, Romano L (2004) Gastroduodenal perforations: conventional plain film, US and CT findings in 166 consecutive patients. Eur J Radiol 50:30–36

Ha HK, Ko GY, Yu Es et al (1999) Intestinal tuberculosis with abdominal complications: radiologic and pathologic features. Abdom Imaging 24:32–38

Hainaux B, Agneessens E, Bertinotti R et al (2006) Accuracy of MDCT in predicting site of gastrointestinal tract perforation. AJR Am J Roentgenol 187:1179–1183

Heffernan C, Pachter HL, Megibow AJ, Macari M (2005) Stercoral colitis leading to fatal peritonitis: CT findings. AJR Am J Roentgenol 184:1189–1193

Heinzerling JH, Huerta S (2006) Bowel perforation from bevacizumab for the treatment of metastatic colon cancer: incidence, etiology, and management. Curr Surg 63:334–337

Hibben JF, Gorodetsky AA, Wilbur AC (1995) Perforated jejunal diverticulum: CT diagnosis. Abdom Imaging 20:29–30

Hinchey EJ, Schaal PG, Richards GK (1978) Treatment of perforated diverticular disease of the colon. Adv Surg 12:85–109

Horrow MM, White DS, Horrow JC (2003) Differentiation of perforated from non perforated appendicitis at CT. Radiology 227:46–51

Imuta M, Awai K, Nakayama Y et al (2007) Multidetector CT findings suggesting a perforation site in the gastrointestinal tract: analysis in surgically confirmed 155 patients. Radiat Med 25:113–118

Kim SH, Shin SS, Jeong YY et al (2009) Gastrointestinal tract perforation: MDCT findings according to the perforations sites. Korean J Radiol 10:63–70

Kimchi NA, Broide E, Shapiro M, Scapa E (2002) Non-traumatic perforation of the small intestine. Report of 13 cases and review of the literature. Hepatogastroenterology 49:1017–1022

Korn O, Onate JC, Lopez R (2007) Anatomy of the Boerhaave syndrome. Surgery 141:222–228

Lau WY, Leung KL, Kwong KH et al (1996) A randomized study comparing laparoscopic versus open repair of perforated peptic ulcer using suture or sutureless technique. Ann Surg 224:131–138

Liu SI, Siewert B, Raptopoulos V, Hodin RA (2002) Factors associated with conversion to laparotomy in patients undergoing laparoscopic appendectomy. J Am Coll Surg 194:298–305

Livingston EH, Woodward WA, Sarosi GA, Haley RH (2007) Disconnect between incidence of non perforated and perforated appendicitis Implications for pathophysiology and management. Ann Surg 245:886–892

Loiseau D, Borie F, Agostini H, Millat B (2005) Diverticulite sigmoïdienne. Gastroenterol Clin Biol 29:809–816

Lorhmann C, Ghanem N, Pache G et al (2005) CT in acute perforated sigmoid diverticulitis. Eur J Radiol 56:78–83

Mabrut JY, Buc E, Zins M et al (2007) Prise en charge thérapeutique des formes compliquées de la diverticulite sigmoïdienne (abcès, fistule et péritonite). Gastroenterol Clin Biol 31:3527–3533

Ongolo-Zogo P, Borson O, Garcia P et al (1999) Acute gastroduodenal peptic ulcer perforation: contrast-enhanced and thin-section spiral CT findings in 10 patients. Abdom Imaging 24:329–332

Pagnoux C, Mahr A, Guillevin L (2003) Manifestations abdominales et digestives au cours des vascularites systématiques. Ann Med Interne 154:457–467

Park JJ, Wolff BG, Tollefson MK et al (2005) Meckel diverticulum: the Mayo Clinic experience with 1476 patients (1950–2002). Ann Surg 241:529–533

Pearl JP, McNally MP, Elster EA, Denobile JW (2006) Benign pneumoperitoneum after colonoscopy: a prospective pilot study. Mil Med 171:648–649

Ramakrishinan K, Salinas RC (2007) Peptic ulcer disease. Am Fam Physician 76:1005–1012

Rubesin SE, Levine MS (2003) Radiologic diagnosis of gastrointestinal perforation. Radiol Clin North Am 41:1095–1115

Saif MW, Elfiky A, Salem RR (2007) Gastrointestinal perforation due to bevacizumab in colorectal cancer. Ann Surg Oncol 14:1860–1869

Siu WT, Leong HT, Law BKB et al (2002) Laparoscopic repair for perforated peptic ulcer: a randomized controlled trial. Ann Surg 235:313–319

Tagliacozzo S, Tocchi A (1997) Antimesenteric perforations of the colon during diverticular disease: possible pathogenetic role of ischemia. Dis Colon Rectum 40:1358–1361

Taourel P, Garibaldi F, Arrigoni J et al (2004) Cecal pneumatosis in patients with obstructive colon cancer: correlation of CT findings with bowel viability. AJR Am J Roentgenol 183:1667–1671

Tsuboi M, Takase K, Kaneda I et al (2008) Perforated and nonperforated appendicitis: defect in enhancing appendiceal wall—depiction with multi-detector row CT. Radiology 246:142–147

Tulchinsky H, Madhala-Givon O, Wasserberg N et al (2006) Incidence and management of colonoscopic perforations: 8 years' experience. World J Gastroenterol 12:4211–4213

Yagan N, Auh YH, Fischer A (2009) Extension of air into the right perirenal space after duodenal perforation: CT findings. Radiology 250:740–748

Zins M, Bruel JM, Pochet P et al (2007) Quelle est la valeur diagnostique des différents examens dans la diverticulite simple et compliquée? Quelle doit être la stratégie diagnostique? Gastroenterol Clin Biol 31:3515–3519

Zissin R, Hertz M, Osadchy A, Even Sapir E, Gayer G (2008) Abdominal CT findings in nontraumatic colorectal perforation. Eur J Radiol 65:125–132