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RESEARCH ARTICLE

IDENTIFICATION OF THE AETIOLOGY OF GASTROINTESTINAL PERFORATION: CLINICAL AND MDCT CLUES

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Abstract

A common medical emergency that has a high fatality rate between 30 and 50 % is perforation of the gastrointestinal tract (GIT). Oesophageal perforations can cause rapid chest pain, odynophagia, and vomiting; gastroduodenal perforations can cause acute, severe abdominal pain; and colonic perforations typically advance more slowly, resulting in localized abscesses or secondary bacterial peritonitis. Sepsis, an abscess that resembles an abdominal mass, or delayed symptoms are among the subsets of people who may present.

Ancillary findings may indicate underlying problems that require more examination after initial closure of the ruptured bowel, while direct multidetector computed tomography (MDCT) findings confirm the diagnosis and localize the perforation site. Findings from MDCT scans include extraluminal gas, gut wall thickening, apparent discontinuity in the intestinal wall, extraluminal contrast, aberrant mural enhancement, localized fat stranding and/or free fluid, and localized abscess or phlegmon in confined perforations.

In order to highlight the MDCT and clinical signs suggestive of the underlying aetiology and localization of the perforated site, this paper will examine the range of MDCT results found in GIT perforation.

I. KEY POINTS

- Extraluminal gas, wall discontinuity or thickness, and fat stranding are signs of a GIT perforation.
- There is a spectrum of abundance to absence in pneumoperitoneum and extraluminal oral contrast.
- Additional observations include masses, faecal impaction, ischemia, foreign substances, and excessive wall thickening.
- The presence of a ruptured peptic ulcer suggests supramesocolic pneumoperitoneum and hyperenhancing gastric wall.
- Iatrogenic perforation is indicated by ascites and/or persistent or growing free air after surgery.

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INTRODUCTION

Gastrointestinal tract perforation is still a disorder with a high death rate, between 30 and 50 percent, even with advancements in emergency treatment [1, 2]. Acute abdominal pain is the typical clinical presentation for duodenal perforations [1, 3], while colonic perforations typically follow a slower progression course and present with localized abscess formation or secondary bacterial peritonitis [1, 4]. Oesophageal perforations can present with non-specific symptoms such as acute chest pain, odynophagia, and vomiting [2]. A small percentage of patients have sepsis, delayed symptoms, or abscess formation that resembles an abdominal tumor [3]. Examining a patient's medical history thoroughly, finding out about previous episodes of the same pain, and determining whether any predisposing factors, such as previous surgery or instrumentation, abdominal trauma, ingestion of foreign bodies, peptic ulcer disease, or other medical conditions, are necessary before treating patients with abdominal, chest, or neck pain (NSAIDs). Treatment options for management could include long-term care for a medical condition like Crohn's disease (CD) made worse by perforation, or short-term care for the cause, such as extracting an ingested foreign substance. Since multidetector computed tomography (MDCT) can accurately localize the perforation site with an accuracy rate of 82–90% and has a high sensitivity for identifying extraluminal gas, it is the modality of choice for evaluating suspected perforations [2, 5–8].

In this study, we address the range of MDCT findings related to GIT perforation and highlight the imaging and clinical hints that may be crucial for locating the perforated site and providing a timely diagnosis of the aetiology of the perforation.

CT- TECHNIQUE

The thorax should be scanned from the oropharynx and thoracic inlet, respectively, to the upper abdomen in cases of suspected pharyngeal and oesophageal perforation [2]. Scanners should go from the lung bases to the pubic symphysis if gastroduodenal, small, or large bowel perforation is suspected [1, 7–10].

At a non-contrast and portal phase (100 mL, 70–80 s after intravenous delivery of low-osmolarity iodinated contrast media), axial pictures with a 2-mm slice thickness should be obtained [1, 6–12]. A biphasic approach in the arterial and portal phases, after 120–150 mL of contrast, is necessary in suspected ischemia infarction to identify vascular alterations and anomalies in perfusion [3]. To rule out low-flow active bleeding in blunt trauma, an additional 3–5 minute period is required [3, 13]. Since extraluminal air and foreign bodies are better visible in lung and bone window settings, respectively, reviewing pictures in several window settings is essential [2, 6–12, 14–18]. The precision of the location of the perforation site can be significantly improved by multiple reformations [2, 6–12, 14–18].

It is still up for debate whether water-soluble iodinated oral contrast should be used. Oral contrast may not be well tolerated, conceal radiopaque foreign items, and cause a major delay in care [5, 7–9, 11, 16, 19, 20]. Oral contrast leakage is a highly specific signal for localizing the hole site when it is present, although its sensitivity is only 19–42 percent [2, 10, 12–14, 20].

It takes three hours to prepare for the evaluation of colorectal perforation after CT scanning, especially in patients who have reduced intestinal motility [11]. As a result, some institutions advise injecting contrast material intrarectally [10, 11, 13]. This needs to be used sparingly and carefully to prevent more rupturing of a fragile colonic wall (i.e. in in- flammatory, ischemic or neoplastic conditions). It is recommended to utilize a rectal tube rather than a balloon catheter when there is a known low colorectal or coloanal anastomosis in order to prevent disruption of the anastomosis [10].

In our hospital, when there is a high suspicion of perforation, oral contrast is usually avoided. In patients in good clinical condition who can wait to undergo the bowel preparation scan, oral contrast is taken into consideration. This is especially true when there are unusual clinical findings with a broad differential diagnosis, when the patient has a complicated history such as a suspected abscess, when they are operated on, or when the results of an initial non-contrast scan are unclear and could be better described with oral contrast. These are especially relevant in cases of gastroduodenal and oesophageal perforations. Reserving intrarectal contrast

for difficult situations, including individuals recovering from surgery or radiation therapy, is appropriate.

CT -FINDINGS

The extraluminal gas and extraluminal oral contrast, along with the discontinuity in the gut wall when contrast, air, or luminal contents leak out, are direct CT signals that are not reliant on the perforation site [2, 5–8, 10, 12, 15]. Pneumoperitoneum is not always the result of bowel discontinuity; it can also lead to a localized phlegmon or abscess [2, 7, 8, 10, 12]. Some examples of indirect symptoms are localized fat stranding, aberrant wall enhancement, segmental colon wall thickening, and/or free fluid [2, 5–8, 10, 12, 15]. When it is feasible, it is crucial to differentiate between "contained" and "free" holes, as immediate surgery is necessary in the latter instance [3]. In the sections that follow, aetiology-specific indicators are examined.

OESOPHAGEAL PERFORATIONS

Oesophageal perforation is an uncommon but potentially fatal illness [21–23]. The overall death rate is almost 13.3%, although it increases dramatically within 24 hours of the beginning of symptoms [21, 23, 24]. The majority of patients arrive in substantial discomfort with non-specific symptomatology that includes subcutaneous emphysema in various combinations, fever, dysphagia, dyspnea, hoarseness, and dysphonia in addition to sudden-onset pain [2, 21, 23].

A CT scan may reveal foreign bodies, subcutaneous emphysema, pleural effusion, mediastinal or cervical fat stranding, mural gas, mural defect(s), and thickening of the oesophagus wall [2, 12]. Lung abscesses, recurrent aspiration pneumonia, and oesophago-respiratory fistulas can occur in cases of complex tumors or strictures [2]. Extraluminal gas that dissects inferiorly into the belly from lower oesophageal perforations can resemble gastric perforations [7, 10].

Iatrogenic causes account for the majority of oesophageal perforations, including spontaneous rupture, foreign body ingestion, trauma, tumors, and less frequently, caustic ingestion, pill oesophagitis, and infectious ulcers in AIDS patients [2, 21, 23–26].

The most common cause of oesophageal perforation is iatrogenic manipulation [1, 21, 24]. (Fig. 1). Rigid endoscopies carry the highest risk of oesophageal damage (0.11 percent), which can increase to 10–15 percent when therapy maneuvers such stricture dilatation and stent insertion are included [2, 21]. The most often associated perforation location, the oesophagus, is impacted in around half of cases with endoscopic retrograde cholangiopancreatography (ERCP) perforations, which have modest rates of 0.03–0.3 percent [2]. An rapid increase in intraluminal pressure during acute vomiting leads to inadequate cricopharyngeal relaxation, which in turn causes spontaneous oesophageal perforation (Boerhaave syndrome) [2, 27, 28]. Left pleural effusion and pneumomediastinum are frequently present along with a rupture at the distal left posterior oesophageal wall (Fig. 2). The symptoms that describe this syndrome are vomiting, chest discomfort, and subcutaneous emphysema [2, 23]. A uncommon intermediate kind of oesophageal damage linked to instrumentation, foreign body impaction, and forceful vomiting is known as submucosal dissection or intramural rupture, which can result in intramural air or contrast leakage (Fig. 3).

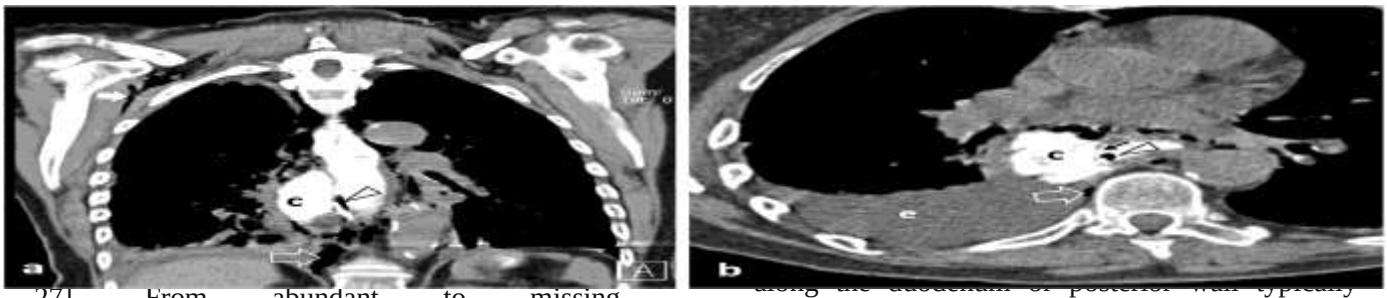
The most frequent foreign objects that result in perforation are food boluses, as they impaction might lead to wall ischemia and necrosis [2]. Usually, the oesophageal wall is directly punctured by fish and fowl bones. When children, elderly, or mentally challenged patients come with protracted dysphagia, foreign bodies should be taken into consideration (Fig. 4).

The oesophagus is especially vulnerable to severe rupture due to its thin wall and inadequate artery supply, with fatality rates exceeding 20 percent [21]. When there are penetrating injuries in the cervical and thoracic region, there is a high index of suspicion because oesophageal damage might be readily overlooked during a clinical examination [2, 23].

In the rare event that oesophageal cancer is present, perforation typically occurs as a result of iatrogenic medication, radiation therapy, or pressure necrosis brought on by a stent that has already been implanted [2].

I. GASTRODUODENALPERFORATIONS

Clinical presentation and causative mechanisms of gastric and duodenal perforations are similar. Due to the fast-evolving chemical peritoneitis brought on by the action of acidic, biliary, or pancreatic contents on the peritoneal cavity, patients with gastric or intraperitoneal duodenal perforation typically report with immediate abdominal pain, guarding, and reduced tenderness [1, 3, 15]. When a confined or retroperitoneal perforation occurs, immunosuppressed patients—including those receiving steroid treatment—may have non-specific symptomatology [3]. A higher than 90% accuracy rate can be achieved in demonstrating gastric and duodenal perforation with MDCT. Evidence of a gastroduodenal perforation is gas in the supramesocolic region. The presence of free gas bubbles in the smaller sac indicates a hole at the duodenal bulb and the



27]. From abundant to missing, pneumoperitoneum can occur [2, 3, 7, 8]. Pneumoretroperitoneum normally develops in the right anterior pararenal area as a result of retroperitoneal duodenal puncture [2, 10]. Moreover, CT scans must to be carefully examined for indications of oral cancer extravasation, segmental wall thickening, localized gastroduodenal wall thinning or discontinuity, perigastric or periduodenal fluid, and nearby fat stranding [3, 10, 27, 29]. There could be gas that reaches the mediastinum.

Gastroduodenal perforation is still most commonly caused by Peptic Ulcer Disease (PUD), with trauma, malignancy, and iatrogenic injuries coming in second and third, respectively [2, 7, 8, 15, 30, 31]. Less frequently occurring causes include ischemic and inflammatory conditions such as vasculitis, volvulus, intussusception, and mesenteric infarction [15, 32].

In 5–20% of PUD patients, perforation occurs [2–3], which accounts for 70% of ulcer-related deaths [32–34]. The infection caused by Helicobacter pylorii is one of the related risk factors. NSAIDs, acetylsalicylic acid, corticosteroids (Fig. 5), bevacizumab, stress, tobacco use, alcohol misuse, and, less frequently,

along the duodenum or posterior wall typically result in contained perforations, ulcers along the anterior wall and curvatures perforate directly into the peritoneal cavity (Fig. 6). The most frequent locations of perforations in ulcerative illness are the duodenal bulb and the gastric antrum [2, 3, 31]. Because post-bulbar ulcers are uncommon (3-5%), they may be a sign of Crohn's disease or Zollinger-Ellison syndrome [35].

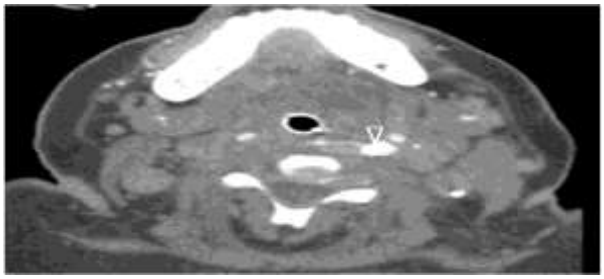
Because of its close proximity to and strong attachment to the spinal column, the retroperitoneal duodenum is the most often injured location in blunt trauma [2, 3, 8]. In addition to the aforementioned findings, CT is useful in differentiating between a duodenal hematoma, which is associated with wall thickening and surrounding fluid, and a duodenal perforation, which may manifest with gas and/or extravasated

oral contrast in the right anter- ior pararenal space [10,36]. Gastric trauma ought to be suspected in cases of diaphragmatic, splenic, or left hemiliver injuries [2, 29]. Finding the injury tract on a CT scan may be the only indication that there was a penetrating injury because the stomach typically collapses after a rupture [2, 29]. Empyema may develop when there is concurrent damage to the left diaphragm and gastric contents flow into the surrounding chest cavity [2].

Fig. 1 A 75-year-old patient experienced pharyngeal perforation as a result of multiple nasogastric tube insertions. The left portion of the pharyng

Fig.2A patient suffering from Boerhaave syndrome, aged 67. After oral contrast medication, (a) coronal and (b) axial unenhanced pictures are obtained. A paraoesophageal collection has contrast leakage because to a significant mural defect (black arrow) (c). Observe several gas bubbles anteroposterior to the oesophagus, a right-sided pleural effusion, and retrocrurally (open arrow) at the right axilla (e). An endotracheal tube (arrowhead)eal wall exhibits a contrast leak, as seen in the axial contrast-enhanced image of the neck after oral contrast delivery (arrowhead)

After oesophagogastroduodenoscopy, inferior vena cava filter implantation, ERCP (0.03–0.3 percent) (Fig. 7), and biliary stent installation, iatrogenic perforations are infrequently observed [2, 3]. ERCP-related duodenal perforations have been classified in four categories, depending on the mechanism and per- foration site [37]. Type I perforations of the lateral or medial wall usually necessitate immediate surgical intervention, but type II and III distal common bile duct lesions can be treated conservatively. Endoclipping is beneficial for patients with type II symptoms [37, 38]. After 48–72 hours, conservatively treated cases could need a follow-up CT scan [38]. When a patient is asymptomatic (type IV), isolated retroperitoneal air from insufflation does not require therapy [37, 38].



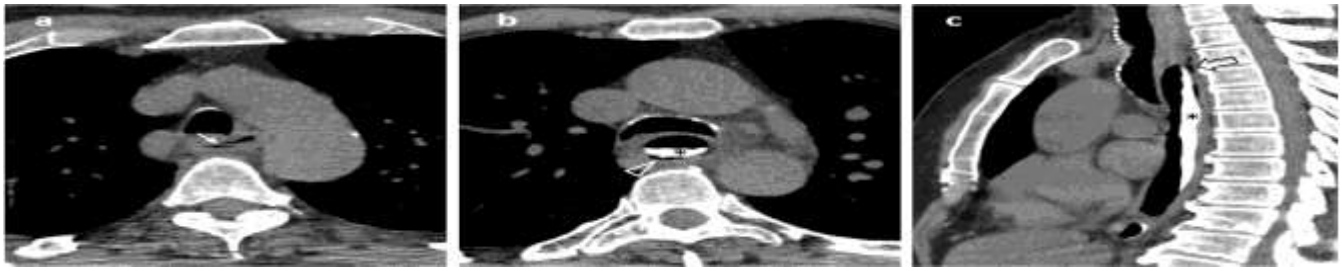
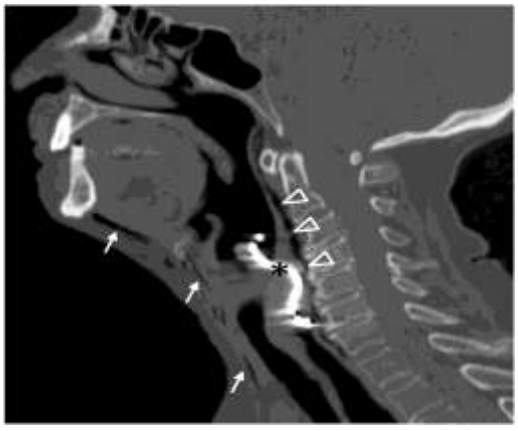


Fig. 3 A patient, 75 years old, has an intramural rupture of the oesophagus. The paraoesophageal fat is blurred in an axial unenhanced image, which also shows a double lumen filled with air and an intervening diaphragm (arrow) that is consistent with a flap. The oesophagus appears to be double-barreled in b axial and (c) sagittal unenhanced images taken after oral contrast administration. These images show contrast at the dependent part of the lumen (*) and a submucosal curvilinear collection of gas (arrowhead), which is better seen on the sagittal plane (open arrow).



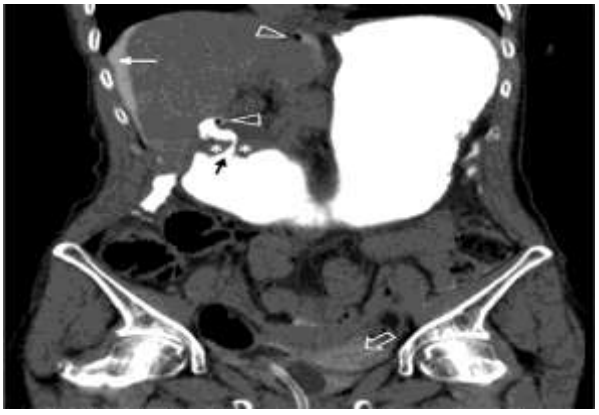


Fig. 4 A patient aged 77 years old has had persistent dysphagia. A ingested perforating denture (*) impinging on the upper oesophagus is seen in the sagittal reformatted picture in the bone window. Both superficial (arrows) and deep (arrowheads) cervical emphysema exist.

Fig. 6 A patient 65 years old with a duodenal ulcer that has perforated. A thicker duodenal wall (*), contrast leakage (black arrow) to the peritoneal spaces, and free gas bubbles may be seen on the coronal unenhanced picture (modified soft tissue window) after oral contrast implantation (arrowheads). In the rectouterine pouch, see the open arrow representing the diluted contrast and the white arrow representing the hyperdensity of the perihepatic free fluid.

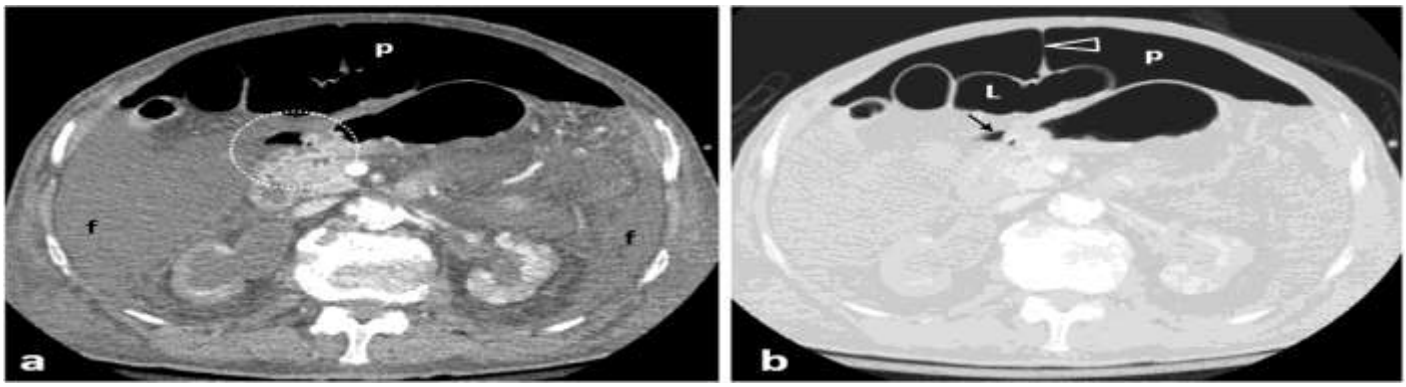
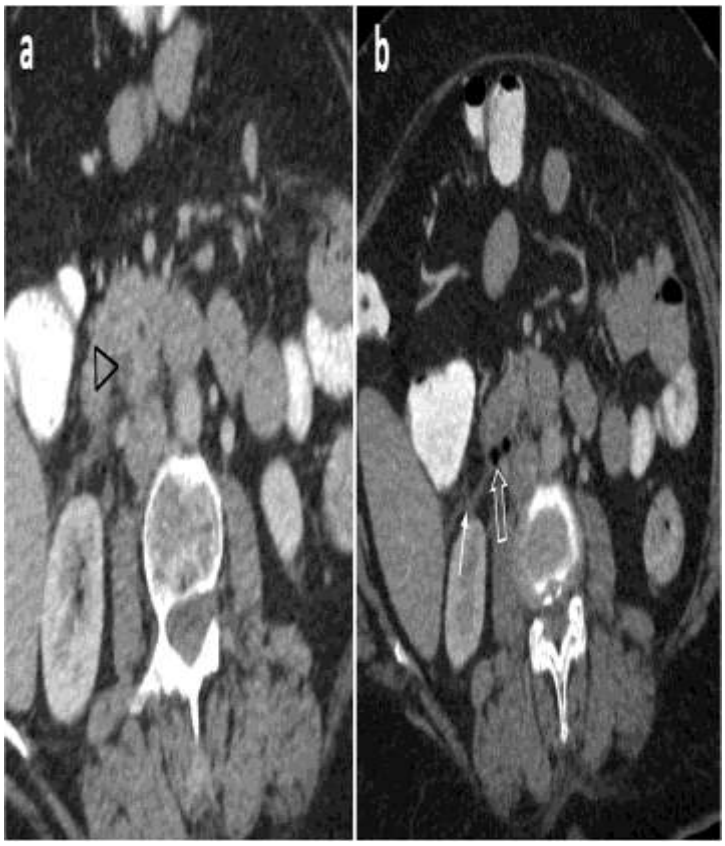


Fig.5A patient, 92 years old, is taking corticosteroids continuously. aThe axial pictures with contrast-enhanced soft tissue window and lung window (b) show a discontinuity of the hyperenhancing stomach mucosa, postero-medially to an extraluminal gas bubble (area within circle). The falciform ligament and air-filled bowel loops (L) are delineated by free fluid (f) and pneumoperitoneum (p) (arrowhead). The distinct, triangle-shaped gas bubble (arrow) indicates the location of the puncture.

Fig. 7 A patient, 75 years old, had a duodenal perforation following ERCP. a, b An axial contrast-enhanced picture reveals thickening of Gerota's fascia and a focal discontinuity of wall enhancement (arrowhead), which are connected to retroperitoneal gas bubbles (open arrow) posterior to the third duodenal segment (white arrow)



A subphrenic abscess, fat stranding, or free or loculated extraluminal gas encircling the band are common signs of perforation in the context of gastric banding, although it can also occur as an acute or, more frequently, a chronic problem due to transmural band erosion [17, 29]. In the stomach lumen, a portion of the band is visible [17, 29]. Malignant gastric perforation is uncommon (0.4–6%); it usually happens in conjunction with ulcerated masses such as large gastrointestinal stromal tumors (GISTs), lymphomas, and adenocarcinomas [2, 29]. Figure 8 indicates that several symptoms such as irregular wall thickening, submucosal mass, ulcer craters that are heaped up, perivisceral soft-tissue extension, peritoneal dissemination, lymphadenopathy, and distal metastatic illness strongly point to an underlying malignancy [2, 29, 35].

Perforation occurs in less than 1% of instances involving swallowed foreign

substances [16, 18]. Some individuals may exhibit nonspecific symptoms, not remember eating a foreign material, and receive a diagnosis months or years later [16]. Individuals who use dentures, have reduced palate sensitivity, abuse alcohol, are young children, elderly, mentally challenged, or all of these characteristics put them at higher risk [16, 18]. A foreign body's radiopacity might vary based on its composition [18]. With the exception of the oesophagus, fish bones are the most frequently found foreign objects in the GIT. They typically cause confined holes that are closed off by the surrounding omentum and inflammation [16]. Pneumoperitoneum that is related is therefore uncommon [16, 18]. Rarely, a foreign body may pierce through and move into a nearby organ, usually the left hemiliver, where it may manifest as an abscess and fistula (Fig. 9) [16, 18]. When a foreign body slowly erodes through the intestinal wall in a neglected or chronic instance, the ensuing inflammatory changes may resemble malignancy. Specifically, duodenal perforations may resemble

Fig. 8 A patient with perforating gastric cancer, age 62. The enhancing antral mucosa (arrowhead) and a focal discontinuity of mucosal enhancement (arrow) associated with fat stranding, pneumoperitoneum (p), free fluid (f), gas bubbles by the posterior wall of the antrum coursing cranially within the lesser sac (arrowheads), co-existing metastases (*), and periaortic lymphadenopathy are visible on axial and sagittal contrast-enhanced images (L)

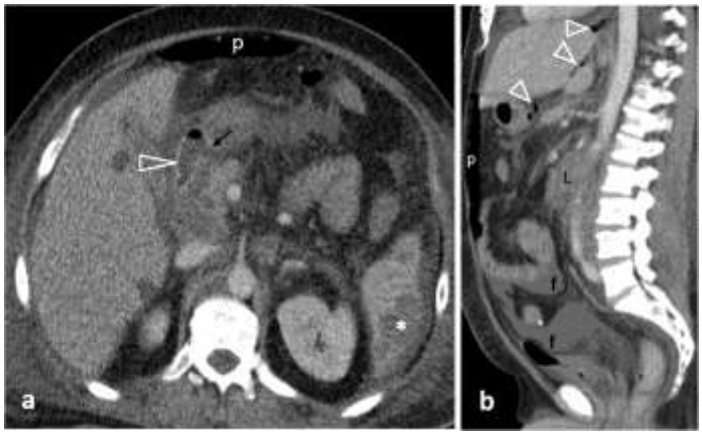
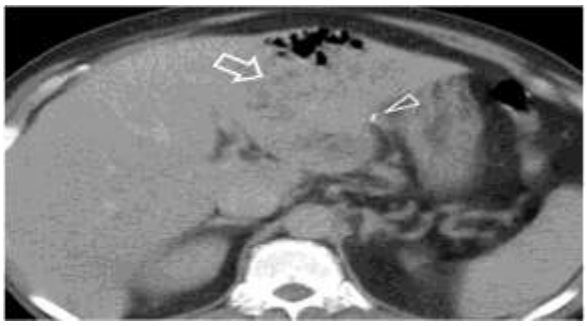


Fig. 9 A 70-year-old patient presents with three days of fever and pain in the right upper quadrant. An axial contrast-enhanced image shows a hyperdense foreign body in the hepatogastric ligament that appears to be a penetrating fishbone and a gas-containing liver abscess



a pancreatic tumor or pancreatitis, as they may have a longer, comparatively asymptomatic course. Identification of a hyperdense structure as the offending foreign material is crucial in these circumstances [16].

A summary of the current symptoms, imaging, and clinical considerations related to the location and etiology of upper gastrointestinal tract perforations is provided (Table 1).

II.

III. SMALLBOWELPERFORATIONS

Perforations in the ileum or jejunum account for 0.4 percent of cases of acute abdomen and have an incidence of 1 in 300–350,000 [20]. The nonspecific presentation is characterized by sudden onset, persistent, medication-unresponsive abdominal discomfort that, if ignored, progresses to sepsis and peritonitis [2, 5, 39].

The most common cause of perforation is trauma, which is followed in affluent nations by closed-loop obstruction and tumors, and in underdeveloped countries by infection (including typhoid fever, TB, HIV, and hookworms) [5, 20]. Less frequently occurring reasons include Crohn's disease (CD), ischemia, iatrogenic interventions,

foreign materials, small bowel diverticulitis, and drugs (e.g., potassium chloride, NSAIDs) [2, 5, 20, 27].

About half of the cases [5, 7, 10, 20, 27] have pneumoperitoneum, which may be missing or too subtle to be seen. Image analysis should thus be done carefully to look for indirect findings such wall thickening, mesenteric fluid, and stranding, as well as ancillary findings like a tumor, an abscess, an in-carcerated hernia, or a foreign substance that could be signs of the underlying etiology [5, 12, 20, 27]. It is important to pay attention to localized interloop collections of extraluminal gas or fluid because they can be misconstrued for intraluminal materials [20]. Though rare, trauma is the most common cause of jejunal/ileal perforation [5]. After the liver and spleen, the small bowel is the site that is affected by abdominal blunt trauma the third most often [5]. The abdomen should be carefully examined for any concurrent injuries because abdominal traumatic lesions are rarely isolated (Fig. 10). Due to the frequent absence of particular indications associated with intestinal injury, CT identification of small bowel blunt injury is difficult [20]. The most reliable marker of intestinal damage is a combination of mural discontinuity and gut wall thickening [14]. Mesenteric fat stranding and a moderate to large volume of unexplained intraperitoneal fluid in the absence of solid organ injury are indirect indicators that should arouse suspicion for occult intestinal injury but not necessarily perforation [2, 14]. Because it can cause a significant delay in surgical care, CT investigation of penetrating injuries is controversial [2]. Since the entry incision may allow air to enter the peritoneal cavity, free intraperitoneal gas is not diagnostic in penetrating injuries [14]. (Fig. 11). The most sensitive observation in this context is the presence of a wound track that extends to a damaged intestinal segment [2, 14].

Either a primary vascular event (vasculitis, major vessel blockage, or venous outflow obstruction) or an either strangulated bowel obstruction (Figs. 12 and 13) can cause a perforation due to ischemia [20]. Further causes of ischemia include severe and protracted hypotension brought on by infection, congestive heart failure, acute myocardial infarction, and hypovolemic shock. The underlying cause, the length, and the intensity of the ischemic attack all influence the CT changes suggestive of underlying ischemia. These abnormalities include segmental bowel wall thickening, localized fluid/fat stranding, bowel wall hyperenhancement, reduced or absent wall enhancement, and emboli or thrombi in mesenteric vessels. In the context of mesenteric ischemia, pneumatosis intestinalis and portomesenteric gas suggest transmural infarction [2, 5, 20].

An uncommon reason of intestinal perforation is small vascular vasculitis. The distribution of several, occasionally discontinuous ischemic bowel segments in a non-vascular territory is one of the indirect indicators used to make the diagnosis: [2, 40]. An almost universal indicator of vasculitis is ischemic involvement of the duodenum (Fig. 14) [40].

Because stricturing or penetrating illness develops within the first 5–20 years of diagnosis, about 75–80% of Crohn's patients need surgery [41, 42]. Bowel loops or adhesions between neighboring structures can cause confined perforation in transmural Crohn's disease [20, 27]. Phlegmon and abscess development may ensue, accompanied by localized peritonitis [14]. Fistulas between the gut and other visceral organs, as well as sinus tracts, are additional extraluminal consequences of penetrating CD. In 1-3 percent of cases, free perforation is an uncommon but potentially fatal complication [2, 14, 20]. (Fig. 15). Further observations such as mural stratification, engorgement of the vasa recta adjacent to an inflamed bowel loop, discontinuous and/or long segment intestinal wall thickening, and these

features characterize active illness and may indicate the aetiology in otherwise undetected CD [2, 14]. In patients with CD, inflammatory stranding in the small bowel mesentery next to a section of the colon with thick walls is not unique to perforation [27].

Typically exhibit regions of hemorrhage or necrotic degeneration in a lamellated pattern with diverse attenuation [2]. Ascites is a rare complication of GISTs, hence a thorough examination for tumor rupture and/or metastatic spread should be prompted by this observation.

Just 0.06-2.3% of people have small bowel diverticula, and they hardly ever rupture [20]. (Fig. 16). Meckel's Diverticulum can become complicated by bleeding and, in rare cases, perforation. It is situated at the antimesenteric side wall of the distal ileum and typically contains gastric mu-cosa (62 percent)[27, 44]. Images that have been resized can more clearly depict the connection between diverticula and the gut lumen and perhaps hint at their location as the puncture site [20].

Small bowel tumor-related perforations are more common in primary malignant lymphomas, particularly in those treated with chemotherapy and steroids, in post-transplant lymphoproliferative disorders, and after radiation therapy [2, 14, 20]. However, metastases, adenocarcinomas, and GISTs can also cause perforations [20]. The presence of luminal aneurysmal dilatation along with circumferential thickening of the gut wall strongly suggests lymphoma [2]. Additional evidence of GIT lymphoma includes hepatospleno-megaly, lymphadenopathy, and multifocal bowel involvement [2, 43]. Rarely do GISTs puncture, and in this context

Fig.10A patient, 7, had a handlebar injury that resulted in a jejunal rupture. A, B The wall thickening of distended jejunal loops (L), mesenteric stranding (*) combining to form a mesenteric hematoma (h), an unanticipated abdominal wall dehiscence (open arrow), and pneumoperiton

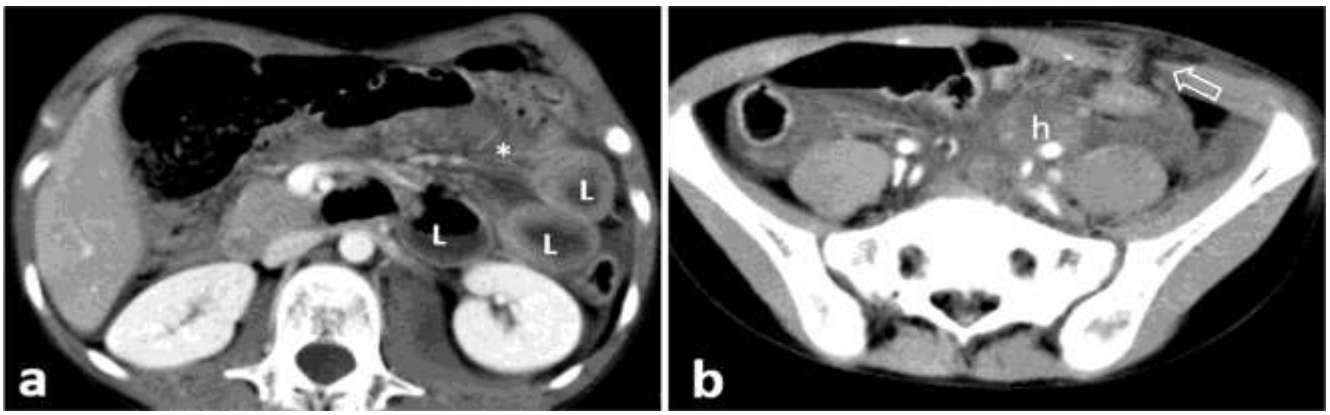


Table1 Presentingsymptoms,imagingandclinicalconsiderationsinrelationtositeandcausesofupper GITperforation

	Presentation	CTfindings	Causes	Cause-specificfindings	Considerations
Oesophagus	Severedistress, sudden-onsetpain, fever, dysphagia,dyspnea, hoarseness,dysphonia,t achycardia, crepitus	Muraldefect, pneumomediastinum,free mediastinalcontrast, free mediastinal fluid, muralgas, subcutaneousemphysema,wa llthickening,mediastinal or cervical fat stranding,pleuraleffusion	Iatrogenic		Historyofinstrumentation
			Spontaneous	Hyperemesis	
			Foreign body	Visiblefoodbolus, impacted foreignbody	Investigateforunderlyings tricture
			Trauma		Historyofpenetrating injury
			Tumour	Massivewallthickening, oesophago-respiratoryfistula	Historyofradiotherapy,inst rumentation, stentplacement
	Fever, SIRS, shock*Gastroduodenal Acuteabdominalpain, guarding, rebound tenderness,non-specific pain (in RP)	Supramesocolicpneumoperiton eum, gas inligamentum teres, gas infalciform ligament, gas inlesser sac, oral contrastleakage, mural defect, gas inanteriorpararenalspace(inRP)	PUD	Mucosalhype renhancement	Helicobacter pyloriiinfection
				Luminal outpouching	Drugs,stress,tobacco, alcohol
			abuseTrauma	Gas in wound track Solidorganinjuries	Historyoftrauma
			Iatrogenic	Gasoutlininggastric band,subphrenicabscess	Peritoneal/nodalspreadMetast atic diseaseHistoryofinstrumentat ionor history of gastricbanding Foreignbody
				Intraluminalband/suture s	
			Tumour		
				Irregularwa llthickening	
				Mucosal/submucosal enhancement	
				Perivisceral soft-tissueextension	

Rarely, minor intestinal perforations might result from ingested foreign substances [27]. Common locations include the ileocecal region and other constricted or angulated areas of the GIT [14, 16, 18, 27]. Similar to gastroduodenal perforations, there is little to no pneumoperitoneum because the foreign body is walled off by the omentum and inflammatory changes as it is gradually impacted [2, 16, 20]. (Fig. 16). The diagnosis is confirmed by identifying a partly extraluminal foreign body, which is easier to see in bone window settings (Fig. 17) [2, 14].

Since it is possible to create a perforation and subsequently travel distally inside the gut lumen, the foreign body may be recognized distal to the perforation site [20]. Iatrogenic perforation during laparoscopic or open surgery typically affects the small bowel and is associated with a significant risk of morbidity and death, particularly if it is not identified during the procedure [2, 20].

During the first week following surgery, aseptic leaking typically happens [10]. In addition to endoscopic operations, other causes of small bowel injury and subsequent perforation include radiation-induced injury, misplaced percutaneous drain-age catheters, and paracentesis [20]. Given that it is typically expected post-laparotomy for up to two weeks and roughly for up to three days after laparoscopic surgeries, intraperitoneal free gas is challenging to understand [7]. In this situation, oral contrast could be helpful since contrast leakage with an unbroken anastomotic site points to the possibility of incidental iatrogenic intestinal injury [2, 10, 14]. Anastomotic leakage or perforation should be taken seriously in cases of ascites and/or persistent or steadily growing free air [8, 10].

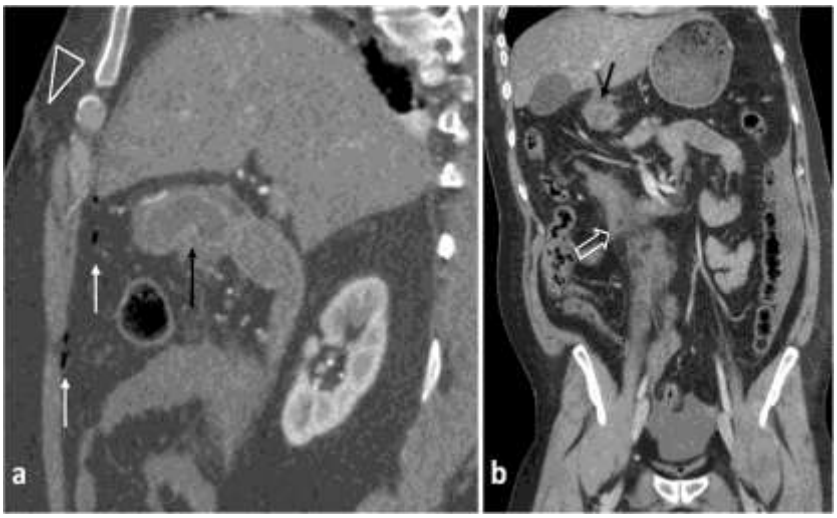


Fig.12A patient, aged 72, who had closed loop strangulation and a minor intestinal perforation. A and B. The internal hernia's sac contains enlarged, poorly enhancing intestinal loops (L) filled with

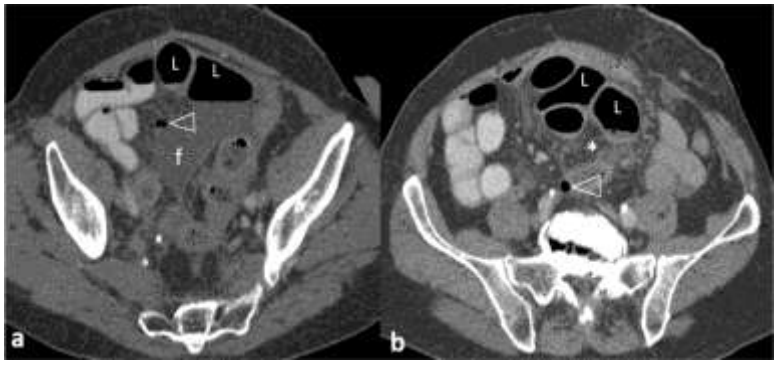


Fig.11A patient, age 35, suffered a gunshot wound and subsequent duodenal and mesenteric damage. A mesenteric root hematoma, scattered air bubbles (white arrows),

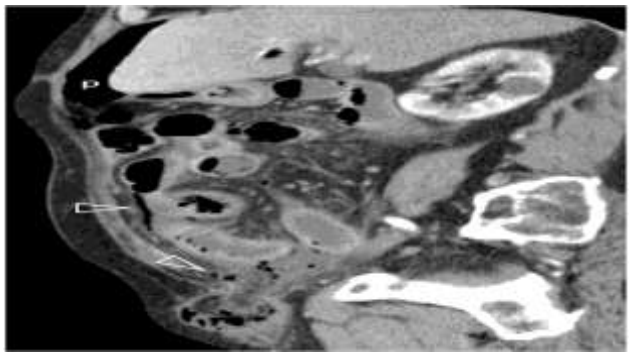


Fig.13A patient, 60 years old, has an imprisoned inguinal hernia in his right side. A poorly enhancing ileal loop inside the hernia, free intraperitoneal gas bubbles (open arrowheads), and periportal gas coalescing topneumoperitoneum are seen in the sagittal oblique contrast-enhanced image (p)

APPENDICEAL PERFORATION

In cases of appendicitis, appendicolith blockage causes appendiceal perforation. Rarely, it is linked to an underlying tumor or mucocele [8, 27]. Fruit seeds, vegetables, lymphoid hyperplasia, intestinal worms (Ascaris), cancer, and foreign bodies are uncommon causes of appendicitis [45]. In the case of appendicitis, there is a substantial correlation between presentation delay and perforation [45].

Particularly in pediatric patients, the role of CT in early detection of appendicitis or micro-perforations in the appendix is controversial [2]. CT has a well-established role in older patients, complex or neglected cases, and potential combined pathologies.

Perforation is strongly suggested by the presence of extraluminal gas, which is often minimal (< 2 mL) or absent, an appendiceal wall defect, peri-appendiceal abscess, and extraluminal appendicolith [2, 7, 8, 10, 27]. Figure 18. Mural calcifications in conjunction with appendix cystic dilatation measuring more than 1.3 cm in luminal diameter point to a mucocele or mucinous neoplasm [2, 46]. Pseudomyxoma peritonei arises from secondary perforation brought on by an appendiceal mucinous tumor [2]. (Fig. 19).

This section provides an overview of the signs and symptoms, imaging, and clinical considerations related to the location and causes of appendiceal and small bowel perforations (Table 2).

COLORECTAL PERFORATION

Comparing colorectal perforation to other GIT perforation sites, colorectal perforation had the highest risk of complications ($55%) [47]. This makes sense in light of the large bowel's bacterial colon, which can cause bacterial peritonitis [1, 4]. The majority of the time, retroperitoneal area perforations exhibit mild symptoms [6, 27]. Comparing colorectal perforation to other GIT perforation sites, colorectal perforation had the highest risk of complications ($55%) [47]. This makes sense in light of the large bowel's bacterial colon, which can cause bacterial peritonitis [1, 4]. The majority of the time, retroperitoneal area perforations exhibit mild symptoms [6, 27].

The most frequent cause of perforations, accounting for 36% of cases, is malignancy [48]. Additionally, 20% of perforations are iatrogenic, 19% are diverticula-related, and less frequently, they are caused by trauma, ingestion of foreign bodies, faecal impaction, ischemia, inflammatory bowel disease, endometriosis, connective tissue disease, radiation, medications, and spontaneous [4, 6, 7, 10, 11, 49]. As a result, neoplastic, spontaneous, diverticular (in western countries), blunt trauma, and ischemia perforations frequently occur on the left side of the colon, whereas inflammatory perforations are more likely to occur elsewhere [4, 8, 10].

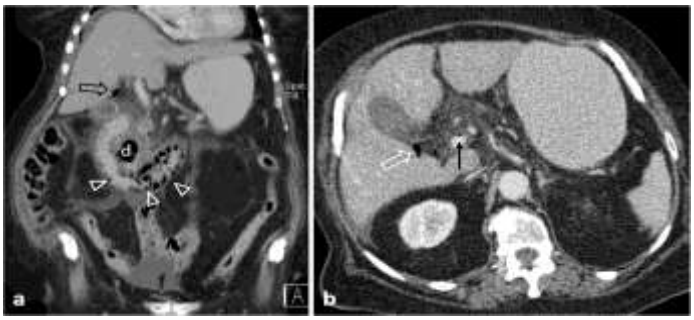


Fig. 14 A patient with vasculitis who is 75 years old. A coronal contrast-enhanced image shows contiguous fat stranding, inadequate enhancement of the horizontal and ascending duodenum (arrowheads), and pneumatosis intestinalis. Take note of the gas bubbles (open arrows) at the porta hepatis, the incidental diverticulum (d) at the second portion of the duodenum, and the moderate amount of free fluid (f). b The

Bowel illness, diverticulitis (in the East), and perforations from penetrating trauma are often symmetrical. When iatrogenic interventions and foreign bodies occur, the rectosigmoid is most frequently impacted [4, 6]. In cases of bowel blockage with a functional ileocaecal valve, toxic megacolon, stercoral colitis, and acute colonic pseudo-obstruction, the cecum may perforate when its diameter reaches 12–14 cm [4, 8, 11]. With the exception of right-sided retro pneumoperitoneum, which can also be the result of duodenal perforation, extraluminal gas limited in the pelvis or inside retroperitoneal compartments suggests colorectal perforation.

In contrast to upper GIT perforations, colorectal perforations have a lower perforation location prediction accuracy with MDCT [9]. Direct findings include extraluminal gas and oral/rectal contrast, together with wall discontinuities, foreign items floating free within the abdominal cavity or bulging through the colonic wall, and faecal material appearing "dirty" mass

[4, 8, 11,27, 50]. A localized inflammatory mass next to the colon, intestinal wall thickening, pericolonic fat stranding, free fluid, aberrant wall enhancement, and abscess are among the associated findings. [4, 8, 11, 27].

Massive amounts of free gas can occur, particularly after a colonoscopy or if there is concurrent blockage [4, 27]. Pneumoperitoneum is caused by perforation of the intraperitoneal colon, which includes the cecum, transverse, sigmoid, and upper two thirds of the rectum [7]. Pneumoretroperitoneum occurs at the right and left anterior pararenal spaces, respectively, as a result of rupture of the retroperitoneal ascending and descending colon [2, 7, 10, 11]. Bilateral pneumoretroperitoneum can result from posterior rectal perforations that dissect superiorly [10, 11]. When diverticulitis or cancer are present without mechanical obstruction, the perforation site frequently experiences small volume pneumoperitoneum. 8, 10]. Only free gas in the pelvis increases the risk of colonic rather than small bowel perforation [2, 6, 11, 27].

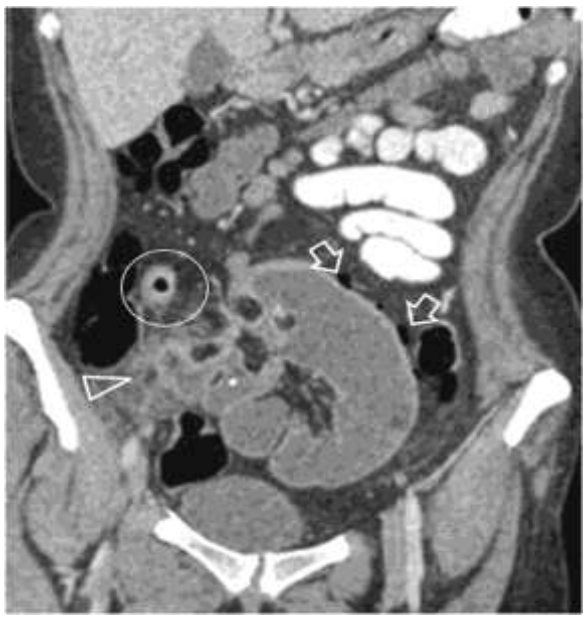


Fig. 15 A patient, age 40, suffering from an unidentified Crohn's illness and an ileal perforation. An enlarged picture with contrast reveals pockets of intraperitoneal free gas, surrounding mesenteric phlegmon (arrowhead), and fluid-filled fistulous ileoileal tracts (*). (arrows). The terminal ileum's mucosal hyperenhancement (inside circle) and mural thickening with stratification are indicative of

Perforations, which most commonly happen in the sigmoid colon (47.3 percent) and cecum (24.8 percent), complicate between 2.6 and 10 percent of colon cancers [2, 6, 11, 49, 51]. There are two established processes [2, 4, 6, 11, 49]. In the first, the tumor has necrosis and is followed by a hole where it formerly was. The second typically occurs around the malig-nancy as a result of intestinal distension associated with blockage and causes severe pneumoperitoneum (Fig. 20) or pneu-

moretroperitoneum. It usually happens close to the cecum. A hole brought on by tumor necrosis usually produces a small amount of free gas [11]. Rarely, people who have a colon perforation into their abdominal wall may show signs of extensive cellulitis (Fig. 21). Distinguishing between inflammatory and malignant origins might be challenging; nevertheless, if the mural wall is thicker than 1.39 cm, the existence of uneven wall

Configuration, localized lymphadenopathy, and metastatic disease all favor cancer because a perforation is usually associated with an advanced tumor stage [51]. Figure 22. The sigmoid colon is the primary site of 4/100,000 population-year perforations resulting from diverticulitis [11, 52]. Extensive peritonitis develops in the event of a free perforation into the peritoneal cavity, whereas extraluminal air/contrast and pericolic abscesses are present in localized perforations in addition to the usual symptoms of diverticulitis (Fig.23)[6].

Fig.18A patient, age 42, has a hole in her appendix. On the contrast-enhanced parasagittal reformatted picture, there is an abscess (ab) with air-fluid levels, surrounding fat stranding, an obstructive appendicolith (black arrowhead), and a dilated appendix (open arrowhead).

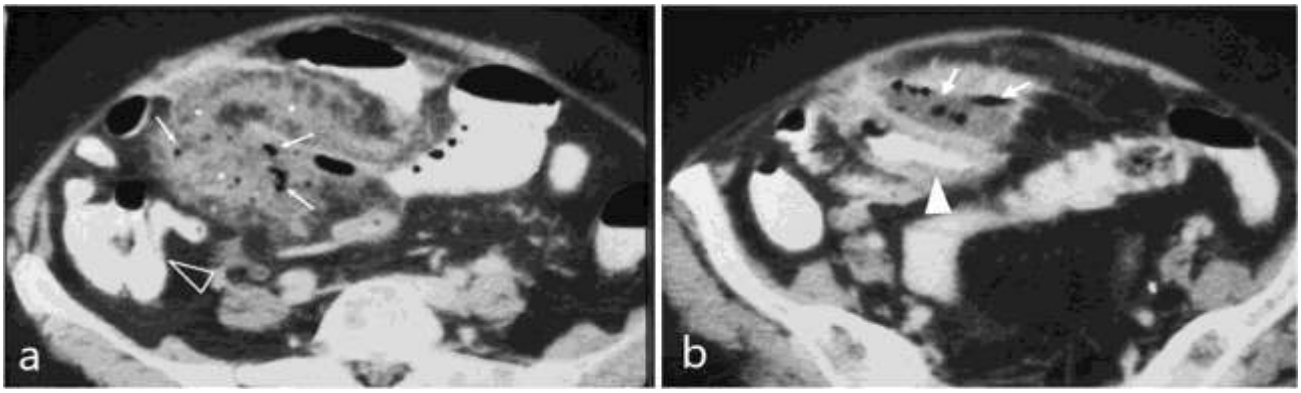


Fig. 16 A patient with a perforated jejunal diverticulum, age 82. A and B Extensive mesenteric fat stranding (*) and free gas bubbles (arrows) that are comparatively contained in a fluid collection around a contrast-filled jejunal loop with wall thickening are seen in axial contrast-enhanced images (white arrowhead). Observe how the ileocaecal valve (arrowhead) and terminal ileum appear normally, which reduces the likelihood of an inflammatory bowel disease diagnosis.

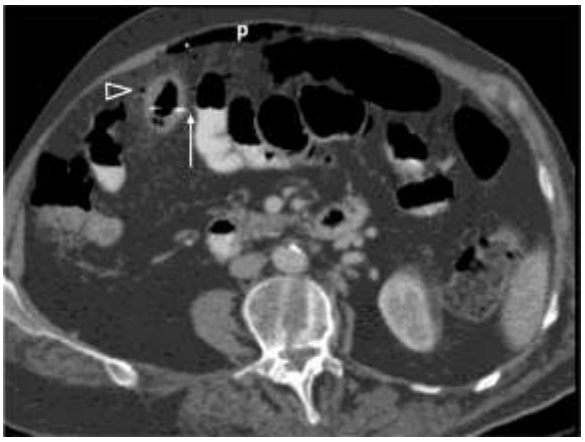
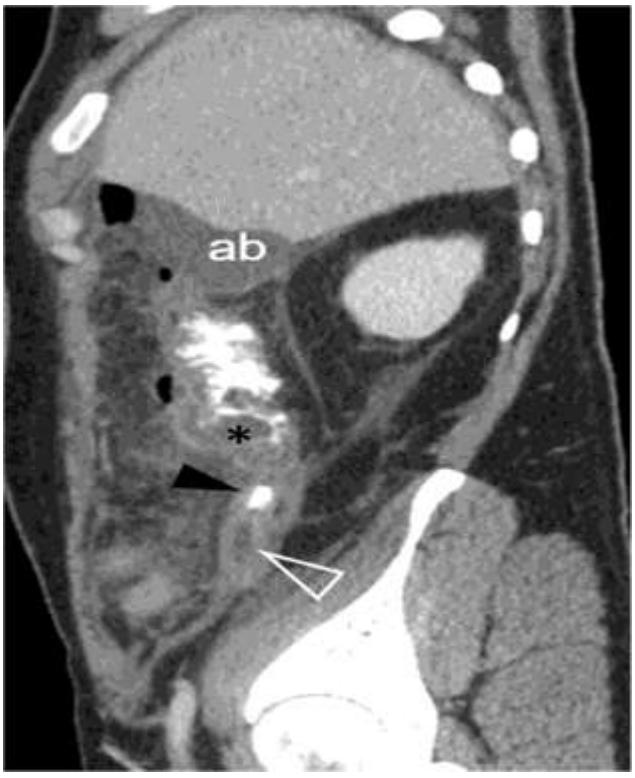


Fig. 17 A patient, age 78. A linear hyperdense foreign body, like a fishbone, is partially visible through the ileal wall in the contrast-enhanced axial picture (arrow). Observe the damaged loop's concentric wall thickening, a few nearby gas bubbles (arrowhead), and the pneumoperitoneum (p)



Perforated sigmoid diverticulitis occurs less commonly and travels through the extra peritoneum [11, 27]. Completely retroperitoneal forms are rather uncommon [53]. The likelihood of iatrogenic perforations is higher in impaired gut than in healthy bowel [4]. They account for 20 percent of colorectal perforations; they typically happen after a colonoscopy and most frequently involve the sigmoid (40.7%), rectum, and cecum [4, 6, 11]. Colic tears on the right side are caused by barotrauma resulting from pneumatic distention, while ruptures on the left side are the consequence of mechanical trauma caused by the endoscope on the antimesenteric gut wall [2].

The perforation rate at the location of the removed polyp is marginally elevated by polypectomy [4, 6]. Post-polypectomy syndrome needs to be considered in these situations since it might manifest as focal mural thickening and resembles perforation clinically. fat stranding in the presence of pericolonic fluid and no free gas [2]. Additional iatrogenic reasons for perforation include anastomotic leakage, injuries sustained during robotic or laparoscopic manipulations, electrocautery, and localized percutaneous/endoscopic procedures like colonoscopic stent placement, paracentesis, abscess drainage, and infrequently, after a cleansing enema [4, 6]. Usually appearing 5-7 days after surgery, anastomotic leaking [11] occurs. When a colonic stent perforates, the diagnosis is verified by observing the stent's extension through the site of disruption of the colonic wall [2].

A number of chemotherapy regimens, such as those involving taxanes, cytarabine, CHOP (cyclophosphamide, hydroxydaunorubicin, vincristine, prednisolone), axitinid, fluorouracil, cisplatin, mitomycin C, IL-2, ipilimumab, rituximab, erlotinib, and bevacizumab, are not without their uncommon side effects [11, 54–56]. In the context of metastatic colorectal cancer and epithelial ovarian cancer, bevacizumab in particular has been identified as the most prevalent cause of drug-induced perforation [33, 55]. Perforation often happens six months after the start of treatment (Fig. 24). Perforation in the context of sigmoid has been linked to corticosteroids, NSAIDs, and opiates.

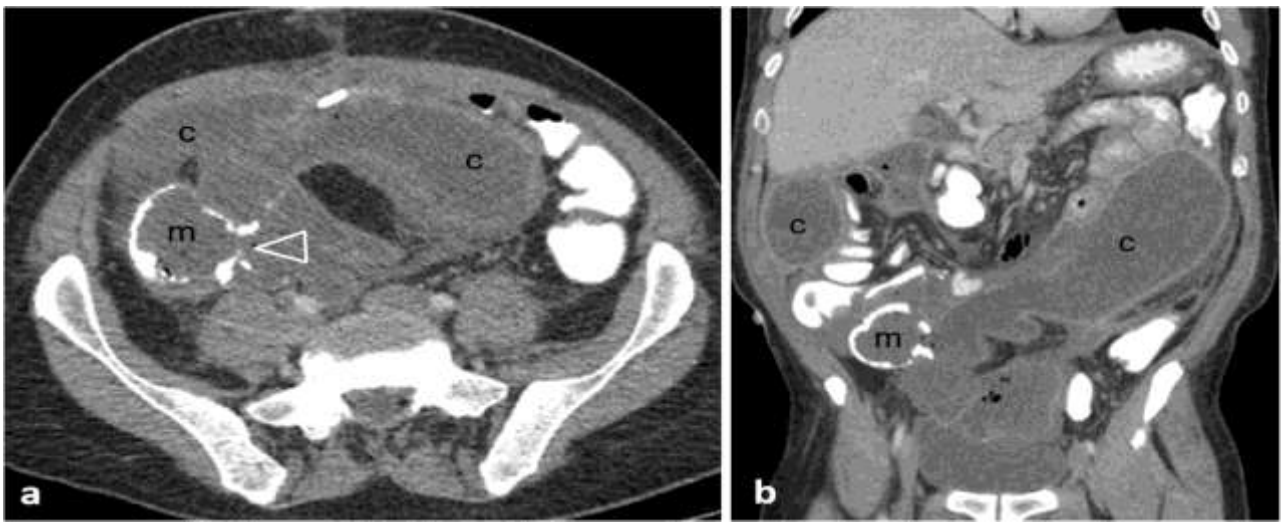


Fig.19A 65-year-old patient suffered a burst mucinous tumor in the appendix. An image with improved coronal contrast (b) and axial contrast (a) reveals a hypodense mass (m) encircled by curvilinear calcifications. Observe the broad, pseudomyxoma peritonei-typical enhancing enclosed fluid collections (c), which originate from a focal discontinuity (arrowhead) in the tumor's calcified wall.

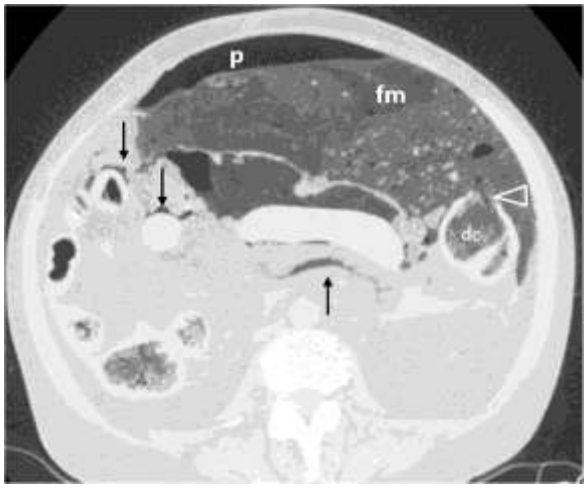


Fig. 20 A patient, who is 75 years old, has rectal cancer and has a perforation caused by obstructive ileus (not shown). feces that have leaked is seen in the lung window's axial image

diverticulitis [54]. High dosages of corticosteroids can conceal acute abdominal symptoms and cause a considerable delay in diagnosis. After radiation therapy, strictures, fistulae, abscesses, perforations, and bleeding can occur as soon as two months or as late as thirty years later in chronic radiation enteritis [57].

The faecal retention conditions faecal impaction and stercoral colitis are common in the elderly, in individuals with scleroderma, in persistent constipation, and in bedridden patients [6]. When there is no thickening of the intestinal wall,

faecalimpaction is characterized by colonic distention brought on by a faecaloma, a localized hard faecal mass [11, 50]. The development of bowel wall perfusion abnormalities and the progression of faecal impaction to stercoral colitis [11], a rare and potentially fatal condition that results in pressure necrosis and perforation [6, 8, 11, 13, 50], are possible if the intraluminal pressure rises to a sufficient degree. When there is fat stranding at a faecal impaction site (Fig. 25), colon dilatation (> 6 cm), and bowel wall thickening (> 3 mm), these conditions raise the possibility of colonic colitis [58]. Diagnosed as 3.2 percent of all colonic perforations, stercoral perforation is a difficult diagnostic that typically affects the anterior rectum, the antimesenteric border of the rectosigmoid junction (24 percent), and the apex of the sigmoid colon (50 percent).

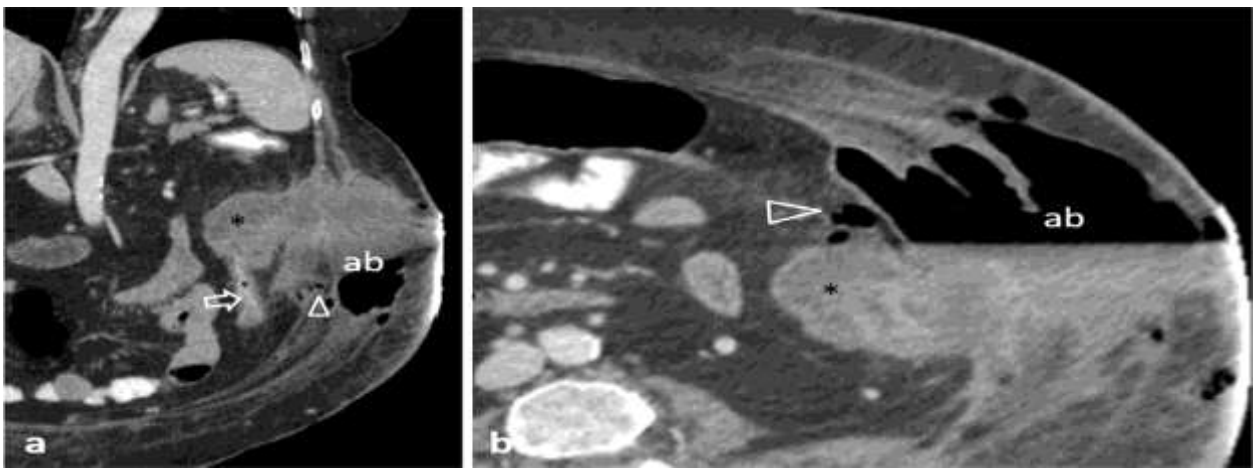


Fig. 21 A 62-year-old patient suffered an abdominal wall colonic rupture. a A soft tissue mass (*) originating from the descending colon (arrow) is seen in coronal and (b) axial contrast-enhanced images. It extends into an abscess (ab) that contains air-fluid level. Take note of the arrowhead-shaped extraperitoneal gas bubbles next to the muscle fascias.

Table2 Presenting symptoms, imaging and clinical considerations in relation to site and causes of small bowel and appendicealperforation

Site	Presentation	CTfindings	Causes	Causespecificfindings	Considerations
Small bowel	Non-specific,abrupt sudden-onsetpain,vomit-ing,anorexia,nau-sea,sepsis, peritonitis	IP gas (minimal/absent), oralcontrastleakage,muraldefect ,wall thickening, poor/inhomogeneous mural enhancement, mesenteric fluid,fat stranding, extraluminalfaecal material	Trauma	Pneumoperitoneum notdiagnosticinpenetratingtrauma, wound track extending tointestinal segment.	Historyoftrauma
			IIschemia	Decreased/absentbowel enhancement, pneumatosisintestinalis, gas/thrombi withinmesenteric/portal vessels	Mechanical obstruction, largevesselocclusion,venousoutf lowobstruction, vasculitis, sepsis,congestive heart failure, acuteMI, hypovolemic shock
			InflammatoryPhlegmon/abscessformation		Typhoidfever,HIV,tuberculosis, hookworms
			IBD	Phlegmon/abscess formation,lengthybowelwallthi ckenning,sinus tracts, fistulas	Crohn'sdisease
			Tumour	Circumferentialwallthickening aneurysmalluminaldilatation multifocalbowelinvolvementl ymphadenopathyhepatospleno megalyheterogeneous mass	Commonly lymphomaadenocarcinoma malignantGISTs metastases
			Diverticulae	Inflameddiverticulum	Meckel's diverticulum
			Foreign body	Foreignbody, maybelocateddistaltope rforation site	CommoninileocaecalareaA void oral contrast
			Iatrogenic	persistent/progressively increasing free gas and/orascites,oralcontrastleakag e	Laparoscopic surgery,anastomoticleakage,endo scopicprocedures.Pneumoperiton eumnormal <2weekspostlaparoscopy
Appendix	Longstanding abdominalpain,f ever, muscleguarding	Periappendiceal/IP gas,appendicealwalldef ect,phlegmon/abscess, fatstranding, free fluid	InflammationExtraluminal appendicolithTumour		
			Luminaldiameter>1.3cm, mural calcifications,pseudomyxoma peritonei, mass,enhancing wall nodularity		

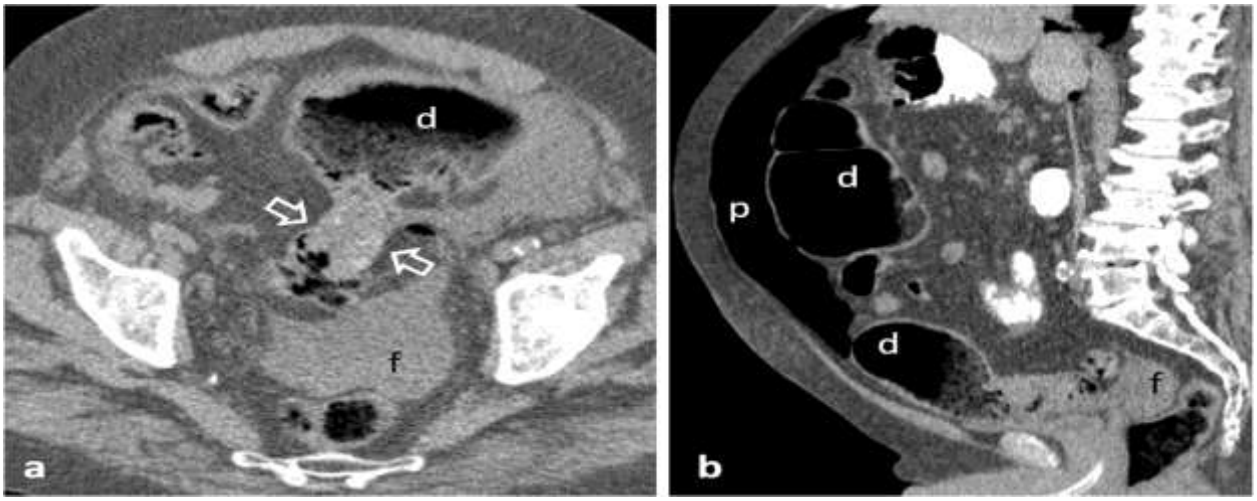


Fig.22 An obstructive sigmoid cancer patient, 75 years of age. Prestenotic colonic dilatation (d), significant pneumoperitoneum (p), concentric narrowing of the sigmoid lumen (between arrows), and free fluid are all seen on non-enhanced axial and sagittal images (f)

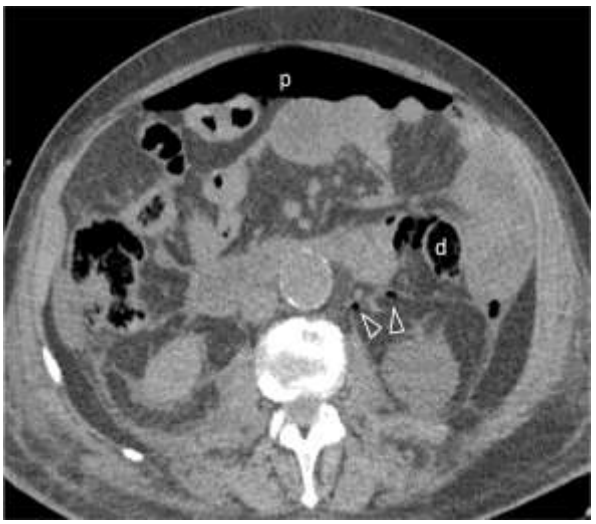


Fig. 23A patient suffering from perforated diverticulitis, aged 73. Gas bubbles connected to sparse retroperitoneum and pneumoperitoneum (p) agglomerate towards the descending colon (d) (arrowheads)

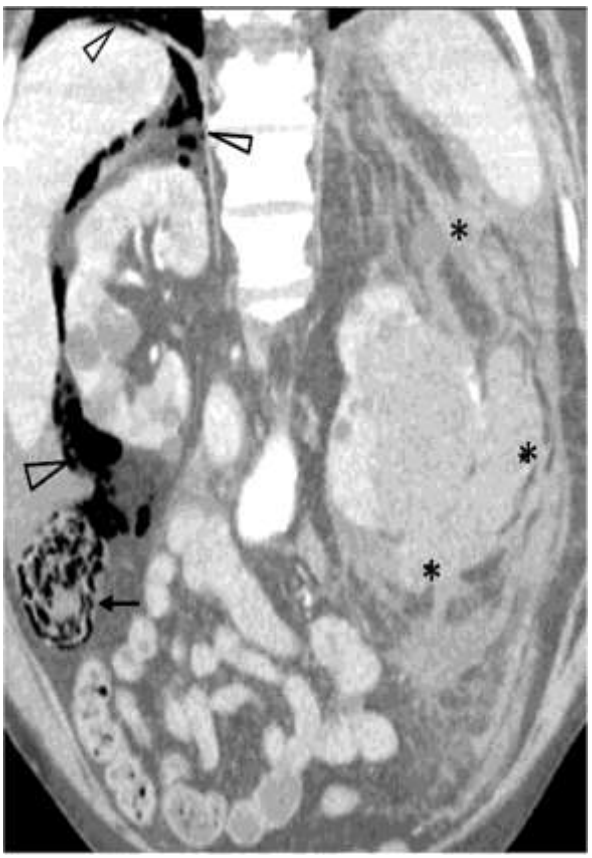


Fig. 24 An 82-year-old cancer patient presents with an ascending colon perforation that occurred spontaneously. Pneumatosis intestinalis (arrow) and free gas spreading to the right pararenal space and to the exposed portion of the liver (arrowheads), consistent with retro pneumoperitoneum, are seen in the coronal oblique contrast-enhanced picture (modified soft tissue window). An independent finding of a left retroperitoneal hemorrhage is observed (*).

Table3 Presentingsymptoms,imagingandclinicalconsiderationsinrelationtositeandcausesoflargebowelperforation

Site	Presentation	CTfindings	Causes	Cause-specificfindings	Considerations
Colorectal	Abdominal pain,nausea, anorexia,vomiting,fever, sepsis	IP gas (cecum, transverse, sigmoid,upper 2/3 of rectum), EP gas(ascending, descending colon,lower 1/3 rectum), extraluminalfaecal contents, oral/rectal contrastleakage, wall defect, faecal materialprotruding through wall/lyingwithinabdominalcavity,bowelwallthickening (> 5mm), fat stranding,abnormal wall enhancement,abscess, inflammatory massadjacent to colon, free fluid	Tumour	Wallthickness>1.39cm,irregularwall configuration,lymphadenopathy, metastaticdisease, free gas, minimal intumournecrosis,freegasmassive following obstruction	Tumour necrosis/followingobstruction
			Iatrogenic	Disproportionateamountof extraluminalgas,stentextendingthrough wall defect	Historyofinstrumentation, opioids, radiation therapy,NSAIDs, chemotherapeuticregimens, corticosteroids Severelyill,postoperative patients
			Spontaneous	Caecaldiameter>14cm,diffuse boweldilatationwithouttransitionpoint	
			Diverticulae	Inflameddiverticulum, pneumoretroperitoneum	
			Trauma Foreign body	Foreignbody,colovesicalfistula,inflammatory mass	
			Stercoral	Faecalimpactionwithwallthickening, Faecaloma protruding throughcolonicwall/inabdominalcavityElderly,chroniccostipation,scleroderma, bedriddenpatients	

Infectious		Salmonella,yersinia, tuberculosis, amoebiasis, Cl.difficile, E. coli, schistosomiasis,shigellosis,herpes,gonorrhoea,syphilis, LGV, CMV
Ischemia	Poor/absentmuralenhancement, pneumatosis intestinalis, vascularocclusion, portomesenteric gas	
IBD	Skiplesions,intramuralfat,fistulaformation, marked colonicdilatation in UC	

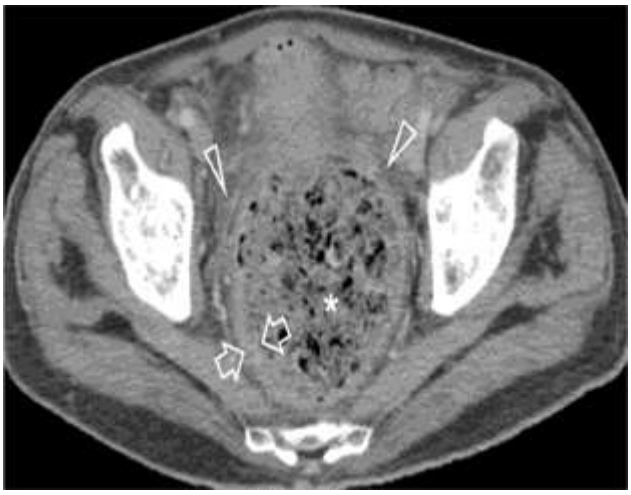


Fig.25A patient, 87 years old, has impending stercoral perforation and stercoral colitis. An enormous faecaloma (*) connected to perirectal fat stranding can be seen crushing a thicker rectal wall (between arrows) in an axial contrast-enhanced picture (arrowheads)

disease (mean age of presentation: 24–26 yapproximately seven percent) proximal to the peritoneal reflection [6, 11, 13, 50]. 57% of people die at this point. Because the colon is sensitive to mechanical restraint and has watershed vascularity, colon-related ulcers are usually numerous and situated on the antimesenteric boundary of the colon [11, 50]. An obstructing mass should be closely examined in the bowel and rectum distal to a faecaloma as the root cause of faecal stasis. Patients with scleroderma may experience vascular dysfunction or colonic perforation due to collagen replacing the smooth muscle of the colon or bowel [6].

Simple radiography is used to identify colorectal foreign bodies, whether they are swallowed or have been inserted retrogradely [2]. CT determines the precise position of the foreign body and evaluates potential problems when there are peritoneal symptoms present or when transanally removing it is not feasible [2, 4, 13]. Direct indications of a perforation include bowel wall rupture and the object's extraluminal position; however, free gas is rare since the hole site is typically "sealed-off" [2, 6, 11, 16, 18]. One may occasionally come across an inflammatory mass or colovesical fistula [16]. Rather than total vascular occlusion, non-occlusive disease (low-flow condition) is more common in cases of perforation owing to ischemic colitis [11].

Arterial occlusion affects entire vascular regions, while low-flow situations mostly affect watershed locations, such as Grif- fith's and Sudeck's crucial sites [11-16]. When the symptoms first appear, these patients may have minor stomach pain and tenderness. Within 24 hours, bloody diarrhea may occur. A

poor prognosis is linked to pneumatosis intestinalis when there is ischemia [17]. Poor or absent mural enhancement suggests ischemia, whereas transmural necrosis is pathognomonic for free air [59].

Acute colonic pseudo-obstruction, also known as Gilvie's syndrome, typically occurs postoperatively or in individuals with substantial comorbidities [11]. The CT results show significant dilatation of the colonic and rectal lumens without a transition point to the collapsed bowel [11]. Perforation is a rare complication (1-3%) with a 50–71 percent mortality rate; however, if the caecal diameter is more than 14 cm, the risk increases to 23 percent [11].

Spontaneous perforation is the most frequent GIT consequence in Ehler-Danlos syndrome, typically involving the sigmoid colon [60]. Perforation is typically the initial symptom in a young patient who would not otherwise be identified with Ehler-Danlos syndrome. It typically happens early in the ears), usually prior to arterial or solid organ rupture [60, 61].

A blunt trauma's perforation is rare; just about 0.5 percent of all large blunt traumas have been reported [4, 13], with the sigmoid, right colon, and cecum being the most common locations [4]. It is possible for transverse colon damage to coexist with duodenal or pancreatic injuries [4]. Oral contrast administration has the potential to enhance the identification of minor mesenteric hematomas and thickening of the intestinal wall when used appropriately in an emergency situation [13]. In mesenteric folds and between loops, free fluid usually collects in polygonal shapes [4]. retroperitoneal segment that has been traumatized tends to stay localized near the site of injury [4].

Colonic perforation in inflammatory bowel disease is uncommon; free perforations, which are less prevalent than sealed-off perforations, occur in roughly 3% of Crohn's disease patients [6]. Free perforation from ulcerative colitis is linked to toxic megacolon and affects roughly 2% of patients [6].

The following summarizes the clinical factors, imaging findings, and presenting symptoms in relation to the location and etiology of colon perforation (Table 3).

CONCLUSIONS

The contribution of CT is twofold: although the diagnosis of GIT perforation may be simple based on clinical and radiographic signs, the scan can show subtle features not seen on x-rays, supporting the diagnosis and guiding proper care. Furthermore, findings that are not directly connected to the perforation might point to underlying issues that require additional research after the first repair of the damaged colon. To achieve effective care, it is imperative that both the surgeon and the radiologist have knowledge of the varied causes of GIT perforation.

ABBREVIATIONS

CD: Crohn’s disease; CT:Computedtomography; ERCP:Endoscopicretrograde cholangiopancreatography; GISTs:Gastrointestinalstromaltumours;GIT:Gastrointestinaltract; iv:Intravenous;MDCT:Multidetectorcomputed tomography; NSAIDs: Non-steroid anti-inflammatory drugs;PUD: Peptic ulcer disease.

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