Mistakes in CT performed for the acute abdomen and how to avoid them

Hameed Rafiee and Stuart Taylor

Abdominal CT (computed tomography) is among the most common imaging tests performed for the investigation of acute abdominal pathology. There are many pitfalls that clinicians and radiologists should be aware of when requesting these studies and interpreting the findings. This article covers ten mistakes frequently made with abdominal CT, focusing on gastrointestinal tract and hepatobiliary pathology. These mistakes and their discussions are based on the available literature where possible and thereafter on our clinical experience.

Mistake 1 CT scanning too early in patients with acute pancreatitis

Acute pancreatitis can usually be diagnosed accurately based on clinical features and biochemical markers alone. There is a considerable risk that a CT scan performed within 72 hours of admission will be normal or underestimate the degree of pancreatic necrosis (Figure 1), so early scanning should be avoided unless there is a high suspicion of severe early complications. After 72 hours, CT scanning is useful in cases of severe acute pancreatitis to assess the degree of necrosis and presence of complications (e.g. pancreatic duct disruption, pseudoaneurysm formation, venous thrombosis, fat necrosis, peripancreatic collections and bowel fistulation/ischemia). Occasionally, obstructing common bile duct (CBD) stones can be seen, although, overall, CT has suboptimal sensitivity for detecting gallstones. CT scans should be performed after intravenous contrast administration, ideally in dual phases—late arterial (35s) and portal venous (70s)—to help optimise detection of pancreatic necrosis and associated vascular complications. The lower abdomen and pelvis should be included to fully assess the extent of free fluid and collections.

MRI (magnetic resonance imaging) is a viable alternative to CT if available locally, and is more sensitive for identifying mild changes of pancreatitis. An MRCP (magnetic resonance cholangiopancreatography) sequence can be obtained at the same time to assess for pancreatic duct disruption and exclude ductal gallstones. MRI is also useful for assessing peripancreatic collections to determine their consistency (fluid versus necrotic tissue), as this can influence management.

Mistake 2 Performing a CT scan for acute gastrointestinal bleeding when the patient is clinically stable

CT scans can be useful for evaluating the cause of acute gastrointestinal bleeding, particularly small and large bowel sources that cannot be reached via upper gastrointestinal endoscopy. However, CT scans can only detect active bleeding >0.3–0.5 mL/min, and so are best utilised in patients who are haemodynamically unstable (but not so unstable that transferring them to the CT scanner would be dangerous). As such, these patients will usually require a medical escort to accompany them to the radiology department. Scanning haemodynamically stable patients increases the risk of a false-negative result and should be avoided.

In addition, the scanning protocol for suspected gastrointestinal bleeding must be optimised, using a triple-phase technique (unenhanced, arterial and portal venous phases). The unenhanced scan is used to identify dense luminal contents that may mimic contrast extravasation on post-contrast images. The unenhanced scan is also best placed to identify intraluminal blood clots and intramural haemorrhage. The arterial phase is used to identify the blush of active contrast extravasation into the bowel lumen, and the portal venous phase helps increase sensitivity by allowing more time for the extravasation (Figure 2). The portal venous phase also helps differentiate active bleeding from a pseudoaneurysm—active bleeding changes morphology between the

Figure 1 | CT scans in a patient with acute pancreatitis. a CT scan performed on the day of admission, demonstrating a rather fatty pancreatic head with some surrounding fat stranding and free fluid, but no evidence of necrosis. b CT scan performed 13 days later, demonstrating extensive necrosis of the pancreatic head (long arrow) with a significant increase in the volume of peripancreatic fluid causing compression of the superior mesenteric vein (SMV) (short arrow).

© UEG 2017 Rafiee and Taylor.

Cite this article as: Rafiee H and Taylor S. Mistakes in acute abdominal CT and how to avoid them. UEG Education 2017; 17: 18–23.

Hameed Rafiee is at the Norfolk & Norwich University Hospital, UK. Stuart Taylor is at University College Hospital, London, UK.

All images courtesy of: H Rafiee and S Taylor.

Correspondence to: hameed.rafiiee@nnuh.nhs.uk

Conflicts of interest: The authors declare there are no conflicts of interest.

Published online: May 25, 2017
Paralytic ileus can mimic small bowel obstruction in the first 48 hours, particularly in the immediate postoperative period. CT is usually the investigation of choice; however, interpreting scans from the immediate postoperative period is difficult. Paralytic ileus can mimic small bowel obstruction in the first 48 hours, particularly in the presence of an ileostomy as it may appear indistinguishable on imaging (figure 3)—the surgical team should be consulted if there is any doubt.

**Mistake 4 Not recognising ischaemic bowel**

Bowel ischaemia is often fatal if unrecognised, and can be a difficult clinical diagnosis to make. When assessing this on CT it is vital to give IV contrast to assess vascular patency and bowel wall enhancement—both arterial and portal venous phases are recommended. A pre-contrast scan may help to identify intramural haemorrhage, which can mimic mural enhancement on post-contrast images alone, but is not always necessary as other post-contrast features will usually indicate the diagnosis. It is also important not to give positive oral contrast, as this will mask mucosal enhancement (in fact, positive oral contrast is generally not recommended in the setting of the acute abdomen because of the risk of missing bowel ischaemia). In some cases, the CT features are clear cut (i.e. mural oedema, poor mural enhancement, intramural gas, free fluid and associated vascular filling defects +/- the presence of gas in the portal system).

The features present can differ depending on the cause—venous occlusion tends to cause more mural oedema and mesenteric congestion than arterial occlusion, whereas arterial occlusion tends to reduce mural enhancement earlier and also causes earlier transmural infarction. The mesenteric arteries and veins should always be carefully assessed for the presence of filling defects representing an embolus (in arteries) or a thrombus (in veins or arteries). In the mesenteric arteries, thrombosis usually occurs near the origin of the superior mesenteric artery (SMA)/inferior mesenteric artery (IMA), whereas emboli tend to wedge at branching points. Occasionally in cases of arterial embolism, small infarcts may be seen in the spleen or kidneys, and in rare instances a thrombus may be visible in the left atrial appendage acting as a source for the emboli.

Venous thrombosis has many different causes, such as thrombophilia, myeloproliferative disorders, malignancy, inflammation, recent surgery/trauma, portal hypertension and oral contraceptives. It is not uncommon to see typical features of ischaemia without a visible arterial/venous occlusion—in these cases the differential diagnosis also includes vasculitis (e.g. polyarteritis nodosa, Henoch–Schönlein purpura, systemic lupus erythematosus and Behçet syndrome), overdistension of the bowel (e.g. due to bowel obstruction, faecal impaction or paralytic ileus) and low-flow states (e.g. hypovolaemic shock).
Mistake 5 Not recognising a closed loop small bowel obstruction
CT is the imaging test of choice when investigating small bowel obstruction. One of the most important considerations is whether a closed loop obstruction is present (i.e. two transition points at a single location creating a bowel loop that is obstructed at both ends [figure 5a]). In most cases an adhesive band (usually related to previous surgery) has crossed over a loop of bowel, thereby obstructing the afferent and efferent limbs (figure 5b). However, volvulus and hernias (both external and internal) may also be responsible. Closed loop obstruction requires urgent surgical intervention because of the risk of strangulation at the point of obstruction, causing mesenteric venous occlusion and subsequent venous ischaemia and infarction (Figure 5c). When features of venous ischaemia are present, it is usually straightforward to diagnose closed loop obstruction on CT, as the oedematous dilated bowel and congested mesentery stand out from the rest of the dilated thin-walled bowel.

In cases secondary to band adhesions, the point of obstruction can be difficult to identify, as the adhesions are not usually visible (except in rare cases where a little fat becomes entrapped within the band [figure 5b]). The small bowel faeces sign (semisolid content in the small bowel lumen), if present, can help to identify the point of obstruction. The cardinal signs of closed loop obstruction include two tightly angulated bowel loops in close proximity with beaked tapering and convergence at the point of obstruction, focal narrowing/obliteration of mesenteric veins as they pass through the point of obstruction followed by venous engorgement within the closed loop mesentery, a cluster of stacked oedematous bowel loops, and a ‘whirl’ sign within the mesentery as it approaches the point of obstruction. The ‘whirl’ sign can be seen in any cause of closed loop obstruction, but is particularly prominent in cases of volvulus. Patients with small bowel volvulus also usually have a predisposing congenital intestinal malrotation.

Internal hernias are a rare cause of closed loop obstruction and occur through peritoneal defects, foramina and recesses (e.g. foramen of Winslow, paraduodenal/pericaecal fossae, perirectal/supravesical recesses, and transomental/transmesenteric/broad ligament defects), which may be congenital or acquired (e.g. the Petersen’s defect in the transverse mesocolon in patients who have had a retrocolic roux-en-Y anastomosis).

Mistake 6 Not recognising mimics of Crohn’s disease
Patients with Crohn’s disease often present with an acute abdomen, and distinguishing active Crohn’s disease from its mimics is important as the treatment for active Crohn’s disease (i.e. steroids and other immunosuppressants) can exacerbate the other conditions. The terminal ileum is the most frequent site of inflammation in active Crohn’s disease and is represented on CT by mural thickening and enhancement, +/- stricturing, +/- an adjacent inflammatory phlegmon or abscess and +/- fistulation with adjacent bowel loops or the bladder. However, terminal ileal thickening can also be seen in other acute conditions, most commonly acute appendicitis, for which there may be secondary oedema of the terminal ileum and an appendix abscess mimicking a Crohn’s abscess (figure 6).
A careful review is required to locate the appendix and assess it for any signs of

**Figure 5** | Closed loop small bowel obstruction. a Formation of a closed loop small bowel obstruction most often occurs when an adhesive band has crossed over a loop of bowel, obstructing the afferent and efferent limbs, but can also occur as a result of a volvulus, which is the twisting of a loop of intestine around itself. b Closed loop small bowel obstruction with venous ischaemia of the closed loop (long arrow) demonstrating mural and mesenteric oedema, reduced mural enhancement and free fluid. Note the visible adhesive band traversing the small bowel (short arrow) due to entrapment of fat within the band. c High-grade closed loop small bowel obstruction with two adjacent transition points (long arrow) and no appreciable mural enhancement within the closed loop. There is a little intramural gas within the closed loop (short arrow) in keeping with infarction.
inflammation. In some cases, the appendix is engulfed or obliterated by the abscess and is not identifiable, making it more difficult to differentiate appendicitis from Crohn’s disease. Assessment of the rest of the small and large bowel can help to identify skip lesions distant from the inflammation in the right iliac fossa that would point towards a diagnosis of Crohn’s disease.

Another important mimic of Crohn’s disease is tuberculosis, which in the gastrointestinal tract most often involves the ileocaecal region. It can be difficult to differentiate the two on CT, but there are certain helpful differentiating features. Tuberculosis affects the caecum much more commonly than Crohn’s disease, often causing contraction and fibrosis, giving the caecum a conical appearance. The presence of large centrally hypoattenuating (necrotic) mesenteric lymph nodes, peritoneal thickening/nodularity and significant ascites also point towards tuberculosis.

Other infections that affect the ileocaecal region include those caused by *Yersinia, Salmonella* and *Campylobacter* species, but they are usually easy to differentiate from Crohn’s disease based on clinical features and a stool sample. On CT imaging, they cause thickening/eoedema of the bowel wall without skip lesions, fistulation or phlegmon/abscess formation. In immunocompromised patients, neutropenic colitis and CMV enterocolitis should also be considered, although both of these more commonly involve the colon rather than the small bowel. Anisakiasis and histoplasmosis can mimic Crohn’s disease on imaging, albeit rarely, but careful history taking will usually differentiate them. Actinomycosis is a rare infection that can involve the bowel, and causes infiltrative enhancing soft tissue masses that extend readily through soft tissue planes. The appearance may mimic an inflammatory phlegmon, but there is usually no significant bowel wall oedema and no ascites.

In patients with multifocal small bowel strictures, considerations other than Crohn’s disease should include radiation enteritis (usually involving pelvic small bowel loops) and NSAID (nonsteroidal anti-inflammatory drug) enteropathy (usually causing very short shelf-like strictures). Less frequent mimics of Crohn’s disease also include lymphoma, eosinophilic gastroenteritis, sarcoidosis, amyloidosis, systemic mastocytosis and endometriosis.

**Mistake 7 Missing small bowel diverticulosis**

Small bowel diverticula are often missed on CT scans because they can be difficult to pick out from the rest of the small bowel, particularly in thin patients in whom the small bowel is tightly packed. Diverticula can cause various symptoms via diverticulitis, perforation (figure 7), enterolith formation (with resultant small bowel obstruction), intussusception, gastrointestinal bleeding, or malabsorption due to bacterial overgrowth. Identifying the presence of small bowel diverticula aids accurate diagnosis and appropriate management, which is particularly important in those patients presenting acutely. Small bowel diverticula occur more frequently and are larger in the jejunum than the ileum. They are usually found on the mesenteric border where the mesenteric vessels penetrate the bowel wall, causing a focal weakness in the muscularis propria, allowing mucosa and submucosa to herniate through. Careful assessment of CT scans in the axial, coronal and sagittal planes usually allows identification of diverticula. Another helpful feature of diverticula is the absence of valvulae coniventes, aiding differentiation from normal small bowel loops.

Another type of small bowel diverticulum is a Meckel’s diverticulum, a congenital malformation caused by embryological failure to obliterate the omphalomesenteric duct. A Meckel’s diverticulum arises from the antimesenteric border of the distal ileum and is said to follow the ‘rule of twos’—2% of the population, 2 inches long, 2 feet from the ileocaecal valve, 2/3 contain ectopic mucosa (usually gastric), and 2% become symptomatic (most often in males). The most frequent symptom is gastrointestinal bleeding, although inflammation, perforation and small bowel obstruction (due to adhesions, enterolith formation, volvulus, intussusception or internal hernia related to a persistent omphalomesenteric duct) can also occur. In patients who have acute complications, a Meckel’s diverticulum is usually easy to identify, but in outpatients who have more chronic symptoms (e.g. intermittent gastrointestinal bleeding), a Meckel’s diverticulum can be difficult to see on CT. CT enterography can help improve sensitivity by distending the small bowel loops with fluid and making them easier to follow, and should be considered if there is a high clinical suspicion for a Meckel’s diverticulum. A Technetium-99m pertechnetate scan can detect diverticula containing ectopic gastric mucosa, but has a limited sensitivity of 60%.

**Mistake 8 Mistaking a perforated colonic carcinoma for perforated diverticulitis**

Colonic diverticulitis and carcinoma can both cause perforation of the bowel, and can be difficult to differentiate on CT—they both present as thick-walled strictures and the presence of perforation inevitably creates surrounding fat stranding in either case. Obtaining an endoscopic diagnosis can also be difficult, particularly if the stricture is impassable with a scope. There are, however, a few CT features that can help differentiate the two (figure 8).

Malignant strictures tend to be shorter than diverticular strictures and usually have shouldered margins with straightening of the thick-walled segment. The mesenteric...
Mistakes in...

**Figure 8** Colon carcinoma versus diverticular stricture. A sigmoid tumor (long arrow) demonstrating irregular mural thickening, loss of mural stratification, straighthening of the bowel loop and focal areas of low attenuation and calcification due to mucin content. A markedly enlarged mesenteric node is also seen (star). Just upstream of the tumour is a segment of diverticular disease (short arrow) demonstrating milder mural thickening with preservation of mural stratification and small gas-filled diverticula within the thickened segment.

Lymph nodes are also often larger and may contain hypoattenuating foci (representing mucin or necrosis), which are highly suggestive of malignancy. Malignant strictures are also more likely to cause large bowel obstruction. Diverticular strictures tend to be longer, with tapered margins and preservation of the normal colonic curvature. The presence of gas-filled diverticula within the thick-walled segment is suggestive of a diverticular stricture rather than malignancy. Preservation of stratified mural enhancement within the thickened colon is also suggestive of benign inflammation, whereas tumors usually demonstrate more homogenous enhancement (except for mucinous tumors, which can appear heterogeneously hypovascular). In many cases, however, it is difficult to be definitive and repeat endoscopy or follow-up imaging may be required to exclude an underlying tumor (if the patient does not undergo surgery for the perforation).

**Mistake 9 Not recognising fat necrosis**

Fat necrosis can occur in several settings and be mistaken for other pathologies on CT. In patients with acute pancreatitis there may be extensive fat necrosis throughout the mesenteric and retroperitoneal fat that can appear quite nodular (figure 9a), mimicking disseminated malignancy. Fat necrosis will involve on subsequent CT scans in the following days to weeks, unlike malignancy which will progress.

Omental infarction presents as a swollen encapsulated fatty mass (usually >5 cm) containing fat stranding that overlies the bowel loops, often adjacent to the ascending colon since the right lateral margin of the greater omentum has the weakest blood supply.

This can be mistaken for colitis with adjacent fat stranding because the colon adjacent to the inflamed omentum may be secondarily inflamed/oedematous, but a careful assessment usually reveals that the bowel wall thickening and adjacent fat stranding is too eccentric to represent colitis (figure 9b). Sometimes the inflamed omentum may appear somewhat mass-like and mimick a liposarcoma or an omental cake, but it is usually possible to differentiate these on CT—if there is any doubt, follow up will demonstrate involution of the omental infarct. Omental flaps used in surgical procedures (e.g. abdominoperineal resection) may also undergo infarction and mimic local tumour recurrence, but awareness of this phenomenon helps avoid this pitfall.

Epiploic appendagitis (infarction of an epiploic appendage of the colon due to torsion or occlusion of its central vessel), presents as a small (<5 cm) halo of fat stranding, sometimes containing a central dot, adjacent to the colon anywhere from the caecum to the rectosigmoid junction (figure 9c). This usually has a characteristic appearance but may be quite subtle, and the adjacent colon is not usually inflamed.

Encapsulated fat necrosis is an unusual entity that can occur anywhere in the body and is thought to be related to trauma. It presents as a well-defined encapsulated fatty mass, sometimes containing a fat-fluid level, which may demonstrate a little capsular enhancement. Such necrosis can mimic a liposarcoma, but follow-up imaging will demonstrate involution rather than progression. Most forms of fat necrosis are self-limiting and resolve with conservative management, so it is important to recognise them to avoid unnecessary invasive procedures.

**Mistake 10 Missing gallstones**

Ultrasound is the primary imaging modality for assessing gallbladder and biliary pathology, and is much more reliable than CT for identifying gallstones. Ductal calculi can, however, be difficult to see on ultrasound due to overlying bowel gas, and will often require cross-sectional imaging to diagnose—usually MRCP because it is much more sensitive than CT. Occasionally, however, gallstones can be picked up on CT scans performed in cases for which the diagnosis is uncertain (e.g. in cases of acute pancreatitis) or incidentally on CT scans performed for other reasons.

Approximately 80% of gallstones are visible on CT. Some are calcified, others may contain gas, but many gallstones are only visible due to a subtle ring of increased density in their periphery (figure 10). In patients who have acute pancreatitis or unexplained biliary dilatation on CT, the CBD must be inspected carefully, because if these subtle calculi are identifiable on CT it avoids the need for MRCP. An unenhanced CT can be helpful to increase the conspicuity of gallstones. Patients who present with recurrent abdominal pain after cholecystectomy may undergo CT to exclude postoperative collections. As well as looking carefully for retained ductal stones, the abdominal cavity (particularly the peripancreatic space) should be assessed for any rounded...
lesions that could represent dropped gallstones, as these are a recognised cause of post-cholecystectomy pain and can act as a nidus for recurrent abscess formation, sometimes many years after the cholecystectomy. Occasionally, dropped gallstones can migrate into unusual places such as the retroperitoneum, abdominal wall, intestine, genitourinary tract, pleural cavity and even the bronchial tree. Recognising dropped gallstones is crucial because the definitive treatment is usually surgery rather than percutaneous drainage.

References

Your imaging the acute abdomen briefing

**UEG Week**
- “MRI” Presentation at UEG Week 2016 [https://www.ueg.eu/education/document/mri/129067/].

**Standards and Guidelines**
- Further relevant articles can be found by navigating to the “Radiology and imaging” category in the “Standards & Guidelines” repository. [https://www.ueg.eu/education/standards-guidelines/]

**“Role of imaging in the diagnosis of IBD” Presentation at UEG Week 2013** [https://www.ueg.eu/education/document/role-of-imaging-in-the-diagnosis-of-ibd/104103/].

Figure 10 | A subtle gallstone. a Subtle gallstone in the distal common bile duct (CBD) with a rim of slightly increased attenuation. b Coronal image of the same patient demonstrating the subtle distal CBD stone.

Mistakes in…