



CONTINUING EDUCATION PROGRAM: FOCUS...

Coping with the problems of diagnosis of acute colitis

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KEYWORDS

Colitis;
Ischaemia;
IBD;
Pseudomembranous;
Neutropenia

Abstract Acute colitis is an acute condition of the colon. For the radiologist, it is mainly diagnosed during differential diagnosis of acute abdominal conditions. There are many causes of colitis and the degree of its severity varies. A CT scan is the best imaging examination for diagnosing it and also for analysing and characterising colitis. The topography, type of lesion and associated factors can often suggest a precise diagnosis but it is nevertheless essential to integrate these findings into the clinical context and take laboratory values into account. The use of endoscopy is still the rule where a doubt remains, or to obtain necessary histological evidence.

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Acute colitis is an acute condition of the colon and most often presents in the form of an acute abdominal picture with very variable clinical symptoms and laboratory test results. The most frequent symptoms encountered in colitis are abdominal pain, fever and diarrhoea [1]. Hyperleucocytosis and elevated CRP in laboratory tests are common. The different types of colitis vary in degree of severity, ranging from simple acute colitis to a picture of fulminant colitis indicating the presence of complications, such as obstruction, toxic megacolon, bowel infarction, colon perforation, or thrombophlebitis, or occurring in a weakened patient. At present, the diagnosis of colitis is based on the results of endoscopy, the procedure indispensable for diagnosing it with certainty, and obtained both from the visual appearance of the mucosa of the colon and from biopsy samples.

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First of all, provide the right imaging examination

AXR

The plain abdominal X-ray, for a long time decried in the literature for its lack of specificity [2], was finally removed in 2009 by the French *Haute Autorité de Santé* (HAS) from the list of examinations still indicated for exploration of acute gastrointestinal diseases [3].

X-ray opaque meal

An X-ray opaque meal (barium or water-soluble) is now no longer indicated in diagnosis of colitis. It may, however, in certain circumstances, be used together with a CT scan.

Ultrasonography

Digestive ultrasonography using high frequency transducers (5–10 MHz) theoretically allows useful study of the appearance and compressibility of the wall of the colon [4,5]. However, to be reliable, this ultrasound analysis requires expertise which is far from being the lot of all operators [6].

MRI

MRI, although extremely useful for studying and characterising chronic colitis, which includes IBD at the forefront [7,8], has no place among the diagnostic procedures for acute colitis.

CT scans

The CT scan is nowadays the indispensable examination for studying an acute abdomen in adults, both for positive and for differential diagnosis [9]. For this reason, it has today become the examination most often performed in acute colitis. It is indeed of real use for positive diagnosis of colitis due to often quite evocative CT signs, in which thickening of the wall of the colon by more than 4 mm, infiltration of the pericolonic fat with abnormalities in appearance, and density or enhancement of the wall of the colon may be associated [1]. The CT scan is also really helpful in the differential diagnoses of colitis [10], all the more important since diagnosis of acute colitis is clinically suspected in less than half of cases, and the main, rather vague, indication for CT examinations, where colitis is finally diagnosed, is in atypical and/or febrile acute abdomen. A CT scan is also very useful for diagnosing the topography of acute colitis; it can indeed distinguish precisely between a segmental lesion and pancolonic involvement. It is also useful in distinguishing between a continuous lesion (particularly in ulcerative colitis (UC) and a discontinuous lesion (highly suggestive of Crohn's disease)). Finally, CT can evoke an aetiological diagnosis in the light of the signs previously described and also when there are signs such as the accordion sign, initially reported as pathognomonic of pseudomembranous colitis, although its high specificity was widely debated [11]. In contrast, a CT scan is definitely of no use and is therefore absolutely not indicated for simple non-severe acute colitis (or ileocolitis) or in cases of colitis of known or obvious

aetiology such as spastic colitis, laxative colitis, antibiotic colitis, "tourist" colitis or when faced with an outbreak of colitis on a background of already documented inflammatory bowel disease (IBD).

Apply a suitable examination protocol and be familiar with the signs of the different forms of colitis

CT protocol

The most suitable CT protocol for examination of an acute abdomen and, by extension, of acute colitis, is based on five principles:

- acquisition without injection (to show up a hyperdense wall particularly if there is a doubt about perforation in looking for pneumoperitoneum or if there is a doubt with an ischaemic picture);
- possible opacification (but this particularly depends on your school of thought) via the rectum with water-soluble material or water;
- intravenous injection of 90 ml of iodinated contrast agent at 2–3 ml/s;
- acquisition of arterial phase images as required (mainly if there is doubt with acute mesenteric ischaemia);
- and in all cases, acquisition of a porto-parenchymal equilibrium phase image (70–90 seconds), the essential phase for any acute abdominal picture.

The signs of colitis

The classic CT signs of acute colitis are comprised of three main aspects: thickening of the wall by more than 4 mm (Fig. 1), infiltration of the pericolonic fat (Fig. 2) and abnormal appearance or density of the wall of the colon that may be seen as a halo sign (resulting from submucosal oedema) (Fig. 3), hyper-enhancement of the mucosa (reflecting hyperaemia of inflammatory or infectious origin) (Fig. 4) or even spontaneous hyperdensity (indicating a haemorrhagic transmural infarction) (Fig. 5). It is important to note that all these signs may be present in other



Figure 1. Elementary signs of acute colitis: parietal thickening of more than 4 mm (arrowheads).

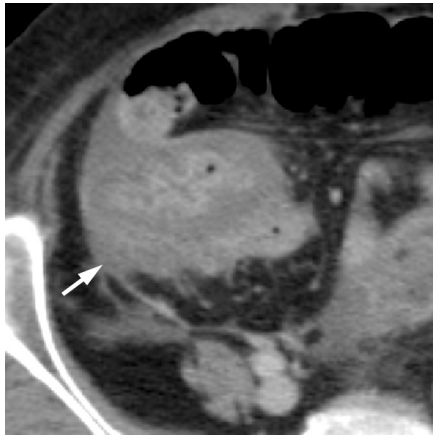


Figure 2. Elementary signs of acute colitis: infiltration of pericolic adipose tissue, with the presence of liquid in the right parietocolic gutter (arrow).



Figure 3. Elementary signs of acute colitis: halo sign (arrow) indicating submucosal oedema.

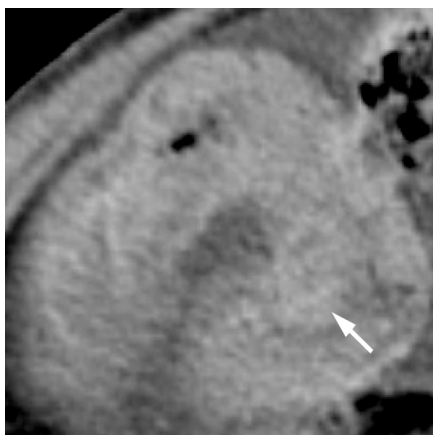


Figure 4. Elementary signs of acute colitis: hyper-enhancement of the mucosa of the wall (arrowheads), indicating hyperaemia of inflammatory or infectious origin.

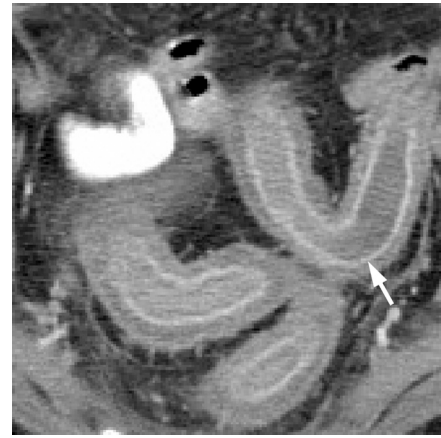


Figure 5. Elementary signs of acute colitis: spontaneous hyper-density of the wall of the colon showing haemorrhagic transmural infarction (arrow).

conditions and are therefore not at all specific to colitis [1,9].

Identify the main traps

The main traps arise from conditions that share some of the CT signs of colitis.

Colon adenocarcinoma

Thickening of the wall of the colon can also occur in colon adenocarcinoma. The short and stenosing nature of the lesion and especially the absence of the halo sign nevertheless usually point the diagnosis towards the tumour process.

Colonic lymphoma

In colonic lymphoma, thickening of the colon wall is considerable, circumferential and never shows the halo sign. The presence of associated adenomegalies and/or splenomegaly suggests the diagnosis.

Acute sigmoid diverticulitis

Acute sigmoid diverticulitis has transmural segmental thickening of the sigmoid colon related to the inflammation of one or more diverticula. There is no halo sign.

Colonic endometriosis

Colonic endometriosis causes parietal thickening of the colon. Its often stenosing character, the absence of the halo sign and the extremely focal topography of the lesion (which is often limited to the rectosigmoid junction) are all distinctive signs that should suggest this diagnosis.

Peritoneal carcinomatosis

Thickening of the colon wall secondary to peritoneal carcinomatosis results in sheathing of the colon. The absence

of the halo sign and the presence of omental or peritoneal contact lesions should provide the diagnosis.

Fatty involution of the submucosa

Fatty involution of the submucosa can produce a halo sign. A distinctive feature is that this halo has negative fat density (-100 HU) (Fig. 6) [12,13]. Moreover, in the absence of any acute process, there is almost never any peri-intestinal infiltration.

Portal hypertension

A halo sign may also indicate portal hypertension [12]. A context of cirrhosis associated with ascites, portosystemic anastomoses and/or splenomegaly can correct the diagnosis.

Right heart insufficiency

In the event of right heart insufficiency, the wall of the colon may appear thickened with submucosal engorgement resulting in a halo sign [12]. However, right heart insufficiency is often already known at the time of diagnosis; the presence of ascites synchronous with a mosaic appearance of the liver in the equilibrium phase after injection of the contrast agent also indicates the cardiac origin of the picture.

Segmental infarction of the greater omentum

Segmental infarction of the greater omentum causes significant infiltration of the pericolonc fat and sometimes induces thickening of the wall of the colon in contact with it [14]. The lesion is nevertheless centred on the omentum and colic parietal signs are often discreet compared with the engorgement of the adipose tissue (disproportionate fat stranding), thereby confirming this differential diagnosis [15].

Acute appendicitis

Infiltration of the pericolonc fat combined with thickening of the wall of the lower caecum can be observed during



Figure 6. Fat halo sign. Note the negative density (-100 HU) of the submucosa (arrow).

acute appendicitis. Right-sided colitis must be eliminated from the diagnosis by careful analysis of the caecum and appendix.

Haematoma of the wall of the colon

Spontaneous hyperdensity of the wall of the colon, representing a haematoma in the wall, can be found when there has been overdose of anticoagulants or other major problems with coagulation [12]. Finding out whether anticoagulants have been taken or observing extremely disturbed coagulation test results helps correct the diagnosis.

Identify complicated forms

Acute pylephlebitis

Infectious acute colitis, acute sigmoid diverticulitis or acute appendicitis may be complicated by acute pylephlebitis, which is thrombophlebitis of all or part of the mesenteric-portal venous drainage network of the infected colon (Fig. 7).

Infarction of the colon

In rare cases, acute colitis may be complicated by transmural parietal infarction characterised by lack of enhancement of the wall of the colon and/or by the presence of dissecting parietal pneumatosis (Fig. 8) [16].

Toxic megacolon

Acute colitis can also be complicated by toxic megacolon, which is dissecting fulminant colectasia following ulcerative colitis (UC) or pseudomembranous colitis (Fig. 9). Septic shock occurring suddenly during acute colitis is the most common clinical expression of this rare diagnosis [17,18].



Figure 7. Pylephlebitis on a background of infectious colitis. Partial portal thrombosis with the presence of a gas bubble (arrow) indicating infection.



Figure 8. Infarction of the colon. Parietal pneumatosis in the caecum (arrowheads).

Perforation of the colon

Acute colitis can also be complicated by perforation of the colon, especially in ischaemia of the colon or toxic megacolon (Fig. 10).

Be familiar with the various aetiologies of colitis

Acute colitis is related to various aetiological pathogenic mechanisms, the main examples being inflammatory (UC and Crohn's disease), ischaemic, infectious (bacterial, parasitic or viral), pseudomembranous, neutropenic, toxic (drugs) or even caused by radiation [1]. The context and specific CT signs must always be considered together.



Figure 9. Toxic megacolon complicating ulcerative colitis (UC). Pulmonary windowing. Tubularised transverse colectasia (arrowheads), with a thin-walled appearance, loss of colonic haustrations and the presence of characteristic pseudopolyps.

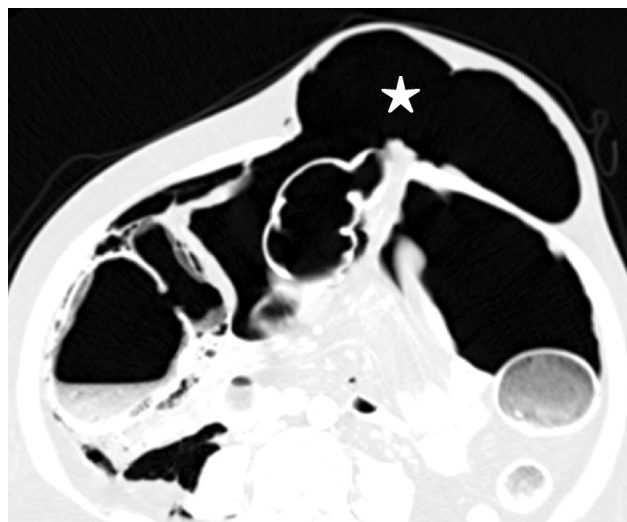


Figure 10. Colonic perforation in ischaemic colitis. Pulmonary windowing. Colonic parietal solution of continuity with voluminous pneumoperitoneum in a hernia (asterisk).

Crohn's disease

Crohn's disease most often occurs in young subjects with a first occurrence around 20 years of age. The disease can affect the entire digestive tract from the mouth to the anus. Colic involvement may in some cases be isolated, but involvement of the final loop of the small intestine is very frequently associated with it. Bloody mucoid diarrhoea suggests the condition. Digestive and extra-digestive symptoms may be associated. A family history of IBD is very common and the diagnosis has often been made prior to this diagnosis, given the episodic nature and the development in successive flare-ups that characterise the disease [19]. On a CT scan, Crohn's disease is seen as thickening of the wall of the colon by more than 10 mm, and asymmetric discontinuous and transmural involvement [20] (Fig. 11). The presence of ascites, but particularly of fistulas and abscesses, and associated signs of the chronic nature of the disease, such as a 'comb' sign, sclerolipomatosis and adenomegaly, provide pointers to the diagnosis [5,19].

Ulcerative colitis

The epidemiological characteristics of UC are similar to those of Crohn's disease. Onset is often early, with an average age of 20 on diagnosis. Bloody mucoid diarrhoea is often at the forefront of the symptoms. It is known to be associated with primary sclerosing cholangitis and this should be sought routinely. In CT scans, UC is seen as an exclusive, retrograde, continuous attack on the colon (very different from Crohn's disease) [21]. The left colon is more frequently affected than the right. The wall of the lower colon thickens as a rule by less than 10 mm. Pericolonic infiltration is variably associated. The lesion is not transmural and is limited just to the mucosa and submucosa (Fig. 12). Because of this, there are never any fistulae or abscesses.

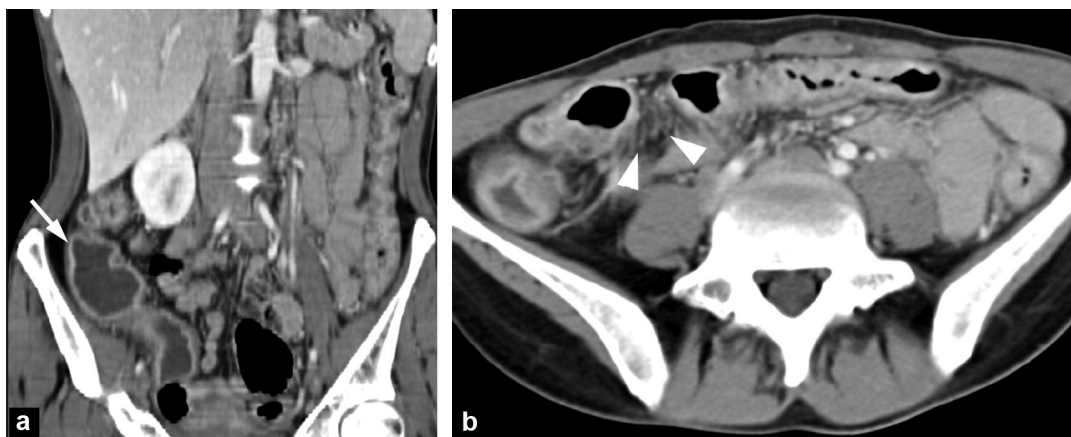


Figure 11. Crohn's disease: a: coronal reconstruction. Thickening of the colonic wall with considerable transmural enhancement (arrow); b: axial slice. Sclerolipomatosis and vascular engorgement resulting in a "comb" sign (arrowheads).

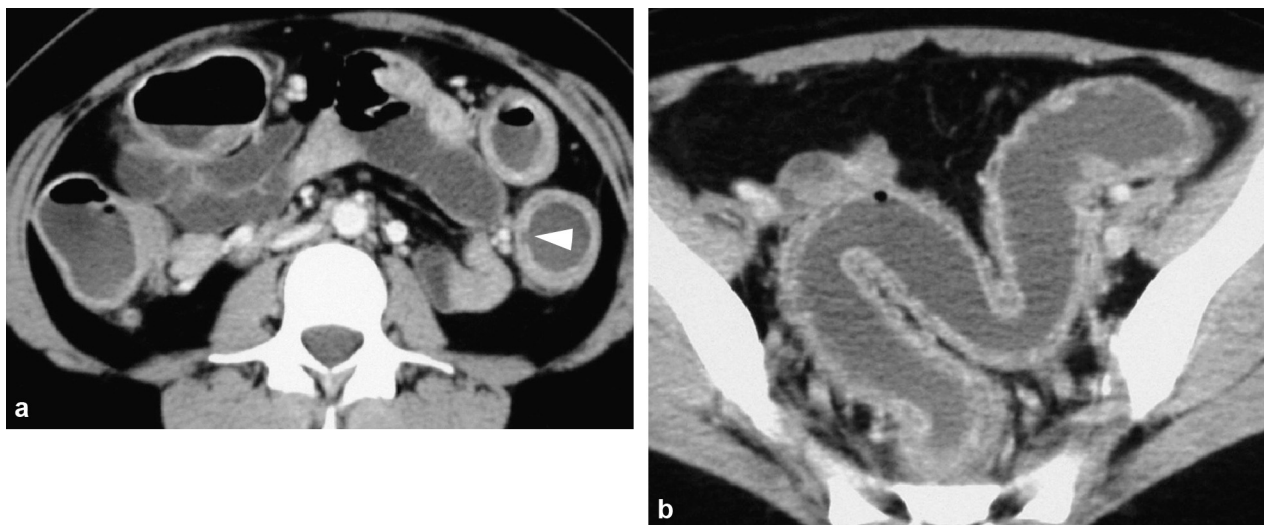


Figure 12. Ulcerative colitis: a: continuous parietal thickening with the presence of mucosal ulceration (arrowhead); b: staged ulcerations and strictly continuous appearance of the colon lesion.

Ischaemic colitis

Acute ischaemic colitis usually occurs after 60 years of age. The two main risk factors are generalised vascular disease and non-insulin dependent diabetes mellitus (NIDDM). Abdominal pain and diarrhoea combined with rectal bleeding are the general symptoms, but elevation of lactates may also be found. Two types of ischaemic colitis must be differentiated: an early reversible (wet) form and a late gangrenous (dry) form [22]. The main cause of ischaemic colitis is non-occlusive [23,24], although it can also have an embolic or atheromatous origin. It mainly involves the left colon and sigmoid colon [16]. In CT scans, the early reversible form is associated with submucosal oedema with considerable mucosal enhancement and often marked peri-intestinal signs (Fig. 13a), whereas the late irreversible form characteristically presents a sometimes spontaneously dense wall, a decrease in or

even no enhancement (reflecting the transmural infarction) (Fig. 13b) and/or parietal pneumatosis.

Infectious colitis

There are many infectious causes for colic lesions. Acute infectious colitis may be of bacterial origin (*E. coli*, *Salmonella*, *Shigella*, *Yersinia*, *Campylobacter*, *Mycobacterium tuberculosis*), viral origin (CMV, *Herpes*), parasitic origin (*Entamoeba histolytica*, *Schistosoma*) or fungal origin (*Candida*, *Histoplasma*). The clinical picture varies depending on the pathogen. Laboratory and serological tests are important since they can sometimes be specific. The diagnosis is confirmed from samples (the yield of which is unfortunately low) and/or from endoscopic biopsies. The topography of acute infectious colitis is variable and sometimes suggests the type of pathogen [1]: involvement can be pancolonic (*E. coli*, CMV), of the right colon, possibly



Figure 13. Ischaemic colitis: a: early reversible form (wet). The three basic parietal layers can still be seen (arrowheads); b: late irreversible form (dry). Marked lack of parietal enhancement and dedifferentiation (arrowheads).

with involvement of the terminal ileum (*E. histolytica*, *M. tuberculosis*, *Salmonella* and *Yersinia*) or of the left colon (*Shigella*, *Herpes* and *Schistosomia*).

E. coli colitis is the most common infectious colitis. It is pancolonic, often causing few peri-intestinal abnormalities.

CMV colitis always appears in the characteristic context of immunocompromised or immunosuppressed patients. Involvement is pancolonic and sometime complicated by perforation of deep parietal ulcers [25] (Fig. 14).

E. histolytica colitis (or amoebiasis) occurs following a stay in a tropical zone where the parasite is endemic. Usually this colitis is ulcerated and fulminant, with bloody serous diarrhoea and evocative liver abscesses if they are synchronous [26] (Fig. 15). Classically the condition involves the right colon and sometimes appears pseudo-nodular [27].

M. tuberculosis colitis almost always occurs in the context of already known tuberculosis. It usually involves the right colon, often with transmural, fibrosing lesions [1]. Voluminous adenomegalies, ascites and signs of peritonitis



Figure 15. Amoebic colitis in a patient returning from an endemic zone. Thickening of the lower caecum (arrowheads) associated with evocative synchronous liver abscesses (arrow).



Figure 14. CMV colitis in a patient with an allograft. Marked parietal thickening and the presence of considerable mucosal enhancement (arrowheads).

are frequently associated with it at the time of diagnosis [28] (Fig. 16).

Pseudomembranous colitis

Pseudomembranous colitis secondary to *Clostridium difficile* infection occurs almost exclusively in conjunction with long-term treatment with broad-spectrum antibiotics (principally aminopenicillins). The clinical picture is often severe with profuse diarrhoea, intense abdominal pain and a high fever. The presence of toxin in the faeces and finding pseudomembranes during endoscopy (and sometimes even with CT) provides certainty of diagnosis [29]. In CT scans, the appearance of pseudomembranous colitis is almost unequivocal, with pancolonic involvement, thickening of the wall by more than 10 mm and an accordion sign (Fig. 17) [11,30,31]. There is frequently ascites.

Neutropenic colitis

Neutropenic colitis (or typhlitis) only occurs in immunocompromised or immunosuppressed patients. It is thought to be due to proliferation of the colon's commensal bacteria,

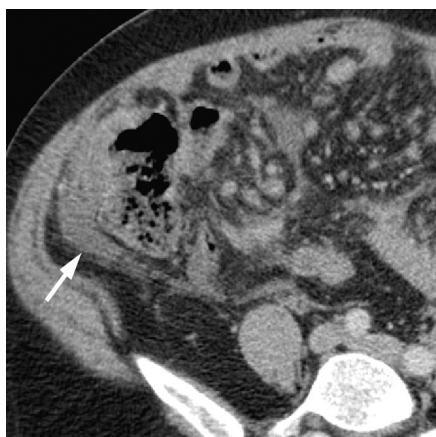


Figure 16. Tuberculous colitis. Thickening of the right colon wall associated with ascites (arrowheads) and many adenomegalies.

related to neutropenia, that can lead to necrosis of the intestinal wall [32]. In CT scans, the right colon is involved, or more precisely the caecum. There is often considerable, circumferential parietal thickening which often continues into the final loop of the ileum. As a rule there are voluminous adenopathies with abundant ascites [33,34] (Fig. 18).

Rare acute forms of colitis

Caustic colitis

Caustic colitis is caused by direct contact of a toxic substance with the mucosa. Some laxatives and also glutaraldehyde, formerly used as a disinfectant for colonoscopes [35–37], have been responsible for a significant number of cases. Involvement is mainly of the rectum and left colon.

Radiation colitis

Radiation colitis may occur where there has been pelvic radiation, particularly of the prostate or gynaecological organs. In most cases the sigmoid colon is involved. The condition is suggested by parietal thickening together with a rigidified appearance of the affected segment (Fig. 19).



Figure 18. Neutropenic colitis in an immunocompromised patient. Considerable thickening of the wall of the caecum (arrowheads). Presence of neighbouring adenomegalies (arrow).



Figure 19. Radiation colitis. Parietal thickening with little enhancement and rigidified appearance of the sigmoid loop (arrowheads).

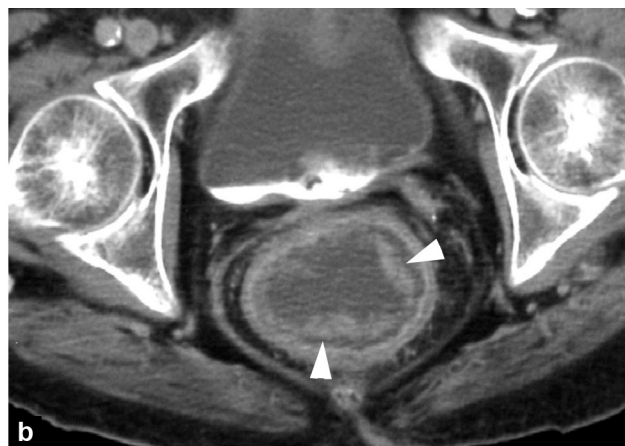


Figure 17. Pseudomembranous colitis: a: very marked thickening of the colon wall with the presence of an evocative accordion sign (arrow); b: false membranes (arrowheads) visible in the lumen of the colon.

Graft versus host (GVH) disease colitis

Graft versus host (GVH) disease colitis is autoimmune in origin and related to allogeneic bone marrow transplantation in nearly 50% of cases. Generally speaking this is more of a generalised ileocolitis [38]. Mucosal hyperaemia, a substantial halo sign and considerable infiltration of peri-intestinal spaces are usual [32] (Fig. 20).

Eosinophilic colitis

Eosinophilic colitis occurs in allergy with non-specific clinical signs, but highly evocative eosinophilia. Associated gastric involvement should bring the diagnosis to mind [39].



Figure 20. Ileocolitis in graft versus host disease (GVHD). Marked thickening of the intestinal wall with a considerable halo sign (arrowheads).

Conclusion

The diagnosis of colitis using imaging is not always simple and usually comes about during the differential diagnosis of acute abdominal conditions. There are many causes of colitis and the degree of its severity varies. CT should be used to differentiate between them, since it is obviously the best type of imaging for diagnosis and also for analysing and characterising the different forms of colitis. The topography, the type of involvement of the colon and the presence of associated features can frequently suggest the aetiology. It is nevertheless essential to integrate the findings into the clinical context and consider the laboratory values. The use of endoscopy is still the rule where a doubt remains or to obtain any histological evidence necessary.

TAKE-HOME MESSAGES

- Use CT for positive diagnosis of colitis and to determine its aetiology.
- Be familiar with the basic signs of colitis.
- Avoid the differential diagnosis traps.
- Recognise complications when they are present.
- Know the list of possible aetiologies.
- Match the context and CT signs for an aetiological diagnosis.
- Use endoscopy where there is doubt or need for a biopsy.

Clinical case

This 54-year-old man with a history of coronary disease and ulcerative colitis presented with bloody diarrhoea then rapidly and abruptly developed septic shock. On clinical examination, the surgeon noted generalised guarding. This CT scan was performed as an emergency (Fig. 21a–e).

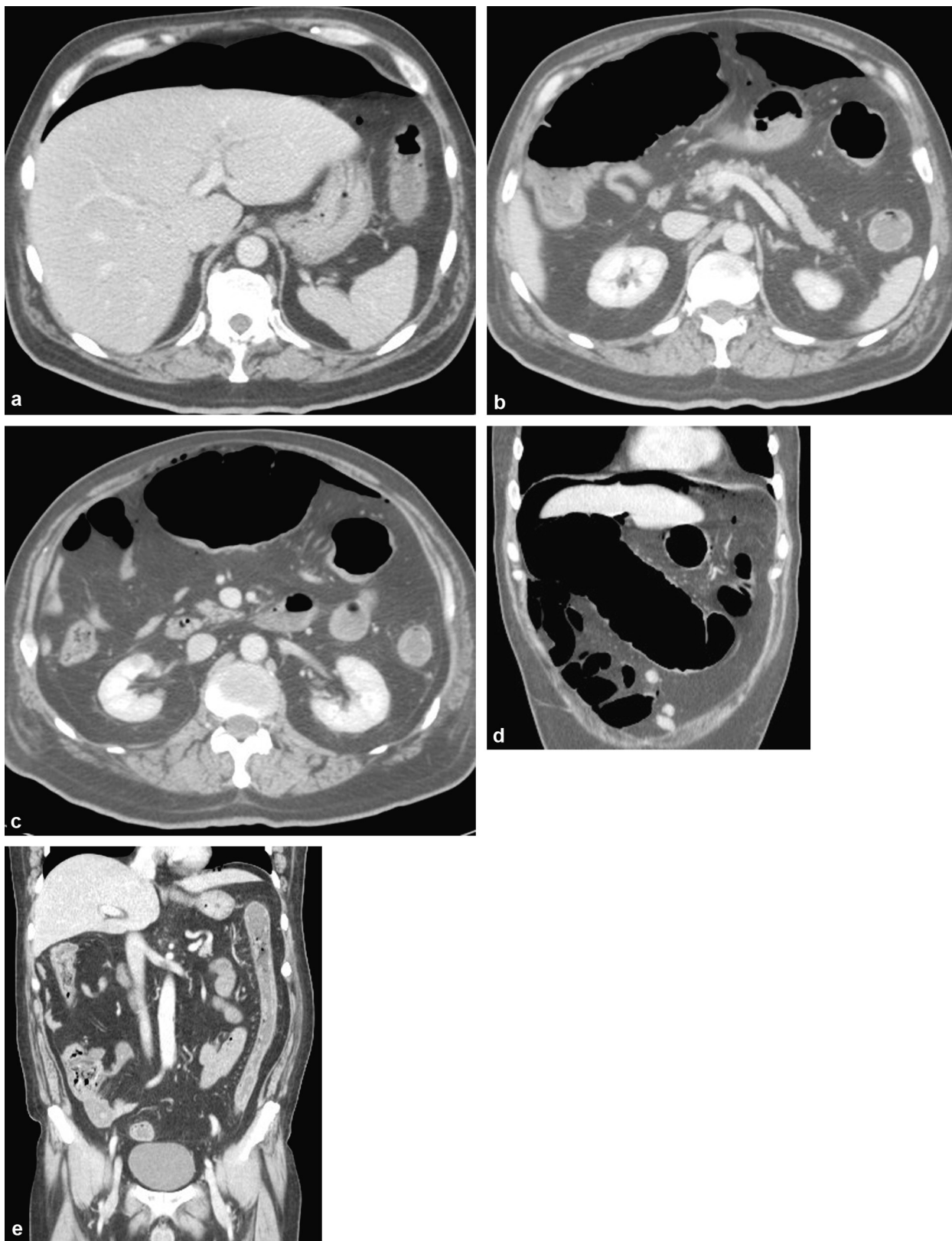


Figure 21. Perforated toxic megacolon complicating ulcerative colitis (UC): a: presence of predominant voluminous pneumoperitoneum in a prehepatic position; b: considerable distension and rigidified appearance of the transverse colon associated with a few pneumoperitoneal bubbles trapped in the fat of the mesocolon; c: loss of colonic haustrations and transmural thickening of the wall of the transverse colon; d: coronal reconstruction. Major transverse colectasia (> 6 cm) and tubularised appearance; e: coronal reconstruction. Parietal thickening with submucosal oedema of the left colon reflecting the subjacent acute colitis. Note the continuous character of the lesion and the presence of numerous staged mucosal ulcerations evoking a flare-up of UC.

Questions

1. What sign of complication can you quickly identify in these CT images?
2. Comment on the appearance of the left colon.
3. What pathological signs are present on the transverse colon?

Answers

1. Presence of voluminous supramesocolic pneumoperitoneum, suggesting a rather high, intestinal perforation (Fig. 21a,d).
2. The left colon looks rigidified, the wall is thickened with many mucosal ulcerations throughout its height, all of which is highly suggestive of a new flare-up of UC (Fig. 21e).
3. Presence of major transverse colectasia (of more than 6 cm) of tubularised appearance, with thinning of the colon wall, disappearance of colonic haustrations and the presence of evocative pseudopolyps (Fig. 21b–d). Given the state of septic shock, the diagnosis has to be toxic megacolon.

Final diagnosis

Toxic megacolon with perforation of the transverse colon in a flare-up of UC.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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