Acute Gastritis and Enteritis

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Abstract

In acute gastric or small bowel conditions, whether they are infectious, inflammatory, or ischemic, the CT scan objectifies submucosal oedema with parietal stratification, producing 'target' or 'double halo' images that can be easily analysed in venous phasis. Precise analysis of proximal peritoneal reactions and of endoluminal content are the first steps of diagnostic approach In an acute clinical context, a number of hypotheses must be discussed (perforation, infection, arterial ischaemia, capillary hyperpermeability, congestion by portal venous stasis, etc.) certain of which may be supported by abdominal/pelvic exploration, as a general rule complemented by thoracic exploration if there are no contra-indications for the radiation risk (young subjects and women of childbearing potential). In all cases, the clinical context and laboratory tests are fundamental for orientating the diagnosis: a history of abdominal pain and diarrhoea, a state of acquired immunosuppression, a recent stay in a country where there are endemic parasites, a purpuric rash on the lower limbs, a marked inflammatory syndrome seen in laboratory tests, etc., are all signs providing pointers for the right direction which one needs to know how to find out by precise, directed questioning and clinical examination

1 Acute Gastritis

There are many different causes of acute gastritis. The most frequent complaints are due to limited histological lesions of the mucosa. They are diagnosed

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exclusively by endoscopy and histology after biopsy. Only deep acute gastric lesions extending into the submucosa, and the muscular layers can be seen during a CT examination performed because of a painful set of hyperalgesic epigastric symptoms with vomiting which may be haemorrhagic.

1.1 General Characteristics

The term 'acute gastritis' covers a very wide spectrum of inflammatory damage to the gastric mucosa, differentiated by distinct characteristics which depend on the depth of the parietal damage and the physiopathological mechanisms involved.

Two major groups can be differentiated:

- erosive gastritis which includes the superficial forms, deep lesions and haemorrhagic forms;
- non-erosive gastritis, the main form of which is gastritis caused by *Helicobacter pylori*.

Most frequently, where involvement is limited to the mucosa, there is little or no correlation between the microscopic anatomopathological data, and the clinical symptoms which are often absent or non-specific (epigastric discomfort, nausea, vomiting). Diagnosis is essentially by endoscopy and biopsy, and there is no place here for any radiological method. There are many possible causes: alcohol, bile reflux, medicinal drugs, etc.

In severe erosive gastritis, in particular where there are deep ulcerations, and where the acute clinical picture reveals painful epigastric seizures associated with vomiting or haematemesis, a CT scan can be performed quite early and provides important semiotic data for a positive and differential diagnosis.

1.2 The Main Aetiological Forms of Acute Gastritis (Levine 2008; Kim and Pickhardt 2007; Gelfand et al. 1999)

1.2.1 Reactive Acute Gastritis or Reactive Gastropathy

There are many causes: non-steroidal anti-inflammatory drugs (NSAIDs), platelet aggregation inhibitors of the aspirin type whether they are administered orally or systemically, at therapeutic doses or in excessive doses, alcohol, stress, bile reflux and ischaemia. In NSAID gastritis, secondary to oral ingestion, the lesions are preferentially sited on the greater curvature due to the effect of gravity (Table 1).

Table 1 Aetiology of acute gastritis and gastropathies

Drug-induced	NSAIDs and aspirin
	Cocaine
	Colchicine
	Antimitotic agents
Massive acute alcoholism with highly alcoholic drinks: whisky, vodka, gin	
Bacterial infections	*H. Pylori*
	H. Heilmani
	Streptococci
	Staphylococci
	Enterobacteria of the genus Proteus
	Bacteria of the genus Clostridium
	E. coli
	Tuberculosis
	Syphilis
Viral infections	CMV
Fungal infections	*C. albicans*
	Histoplasmosis
Parasitic infections	Anisakiasis
Cardiogenic stress and shock	
Irradiation	
Allergies and food poisoning	
Biliary reflux	
Ischaemia	

1.2.2 Bacterial Gastritis

Phlegmonous gastritis is severe acute damage of bacterial origin, caused by various microorganisms such as *Escherichia coli*, *Pseudomonas aeruginosa*, *Clostridium perfringens*, *Enterobacter aerogenes*, *Proteus vulgaris*, *Staphylococcus aureus* or non-haemolytic streptococci; more rarely, its origin may be fungal (candidiasis, mucormycosis) (Jung et al. 2003; Asrani et al. 2007).

In general, it occurs in subjects in a poor general state of health. It is often secondary to massive ingestion of alcohol and coexists with respiratory infections. It is also found in AIDS patients (Fig. 1).

The infection affects the deep layers, the submucosa, muscularis propria and serous, it may have a gangrenous appearance and lead to peritonitis by perforation. The prognosis is very unfavourable, with a mortality rate in the order of 65%, even with treatment.

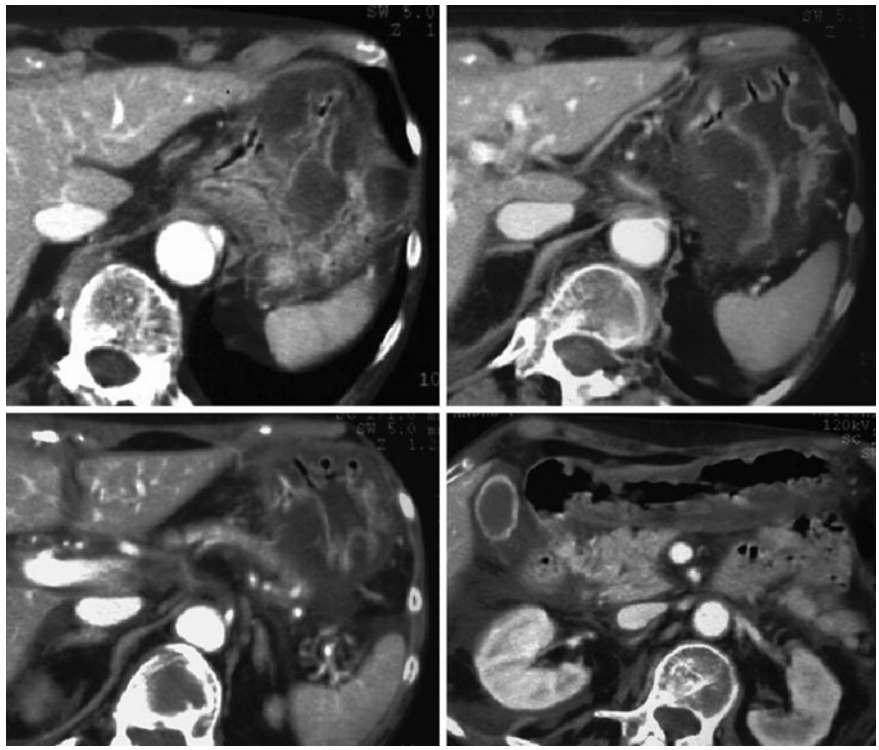

Fig. 1 Phlegmonous gastritis due to *Pseudomonas aeruginosa* (obs. by C. Aubé, Angers). Very considerable oedematous thickening of the whole of the gastric body and fundus, in places exceeding 20 mm

An intramural gastric abscess is a localised form of suppurative bacterial gastritis. It is a condition known since the time of Galen; diagnosis can be made with a CT scan when a liquid collection is seen within a gastric wall thickened by inflammation.

Other infectious causes of severe acute gastritis include:

- Viral infections, particularly cytomegalovirus infections, generally seen in immunosuppressed subjects, particularly in transplanted patients, those with cancer, and in AIDS. CMV gastritis may be haemorrhagic (Fig. 2). Acute gastritis caused by herpes simplex virus is exceptional.
- Acute fungal infections, in particular caused by *Candida albicans*, may be serious and even lethal. They occur above all in immunosuppressed subjects. This is also the case for the uncommon type of gastritis due to mucormycosis (pre-disposing factors: above all diabetes, but also leukaemia, lymphoma, chronic renal impairment, solid organ transplantation, septicaemia, severe burns, malnutrition, corticosteroid treatment and long-term antibiotic therapy). *Histoplasma capsulatum* can also be the cause of ulcerated gastritis which can be erosive or with large rugae, but does not generally occur in an acute form.
- Acute gastric parasitic infections, including anisakiasis caused by a nematode contaminating sushi or other dishes based on raw fish. The worm digs into the gastric mucosa along the greater curvature, leading to symptoms of acute pain that can persist for several days, linked to erosive and/or ulcerative lesions associated with large oedematous folds (Fig. 3).
- Gastric tuberculosis is generally a subacute condition most often found in the context of immunosuppression (AIDS) or disseminated tuberculosis.

Secondary syphilis has become a rare cause of gastritis that is generally subacute or chronic.

1.2.3 Haemorrhagic and Ulceronecrotic Acute Gastritis

As a general rule, these are seen in patients who are critically ill, particularly with shock, severe infections, respiratory insufficiency or acute renal impairment, head injuries, extensive burns, etc.). The mechanism is essentially ischaemia secondary to hypotension and hypovolaemic shock, to which are added the effects of the vasoconstrictor treatment administered, but the aetiology often remains unknown.

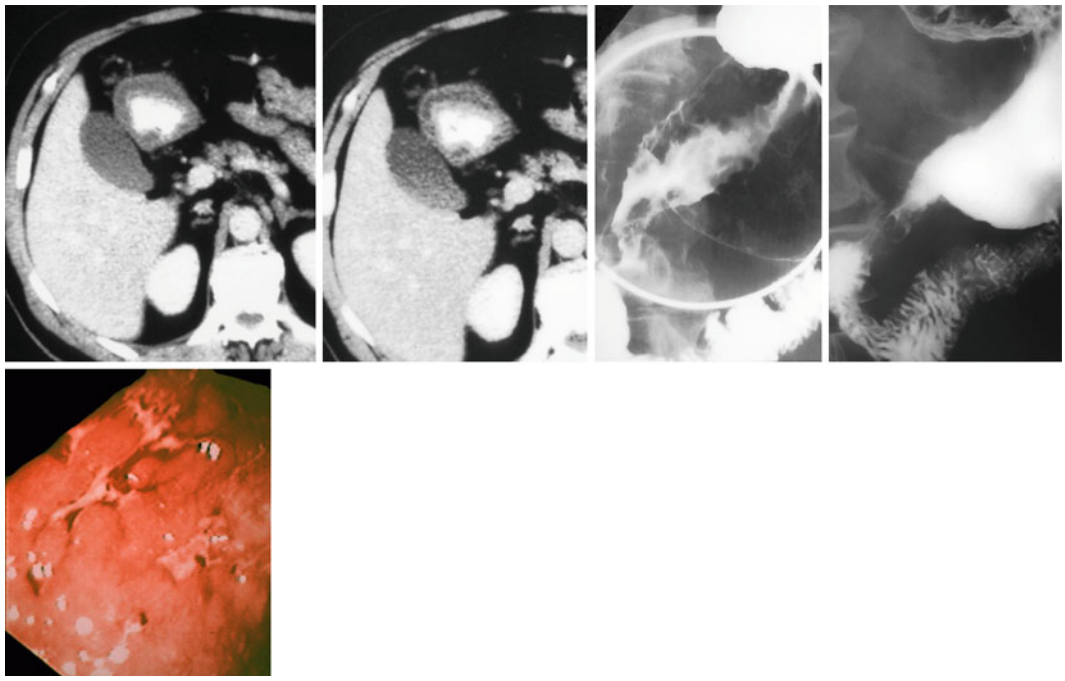

Fig. 2 Gastritis due to CMV. Irregular thickening of the antral wall with submucosal hypodensity. Presence of eroded parietal nodules on the double contrast image. Endoscopy confirmed the presence of whitish ulcerations surrounded by hypervascularised folds of a nodular character

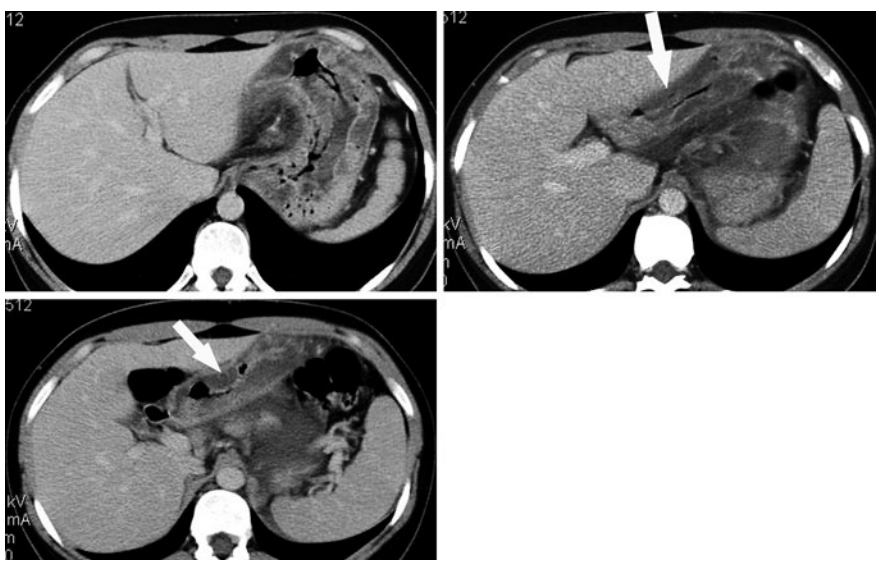

Fig. 3 Anisakiasis (obs. by Dr J. M. Hervochon, La Rochelle). Acute painful epigastric symptoms in a young woman. Very considerable diffuse oedematous thickening of the whole gastric body and antral region (*white arrow*) with oedematous infiltration of the lesser omentum. Aetiological questioning revealed recent ingestion of sushi and endoscopy confirmed anisakiasis

The lesions, essentially petechial or diffuse haemorrhages are similar to those seen in acute drug-induced acute gastritis (aspirin, NSAID), except that their distribution is different, occurring in the fundus and body of the stomach.

1.2.4 Acute Gastritis Caused by Physical or Chemical Agents

Acute radiation gastritis is seen after exposure greater than 16 Gy and more frequently affects the antrum than the fundus; it can be complicated by pyloric stenosis.

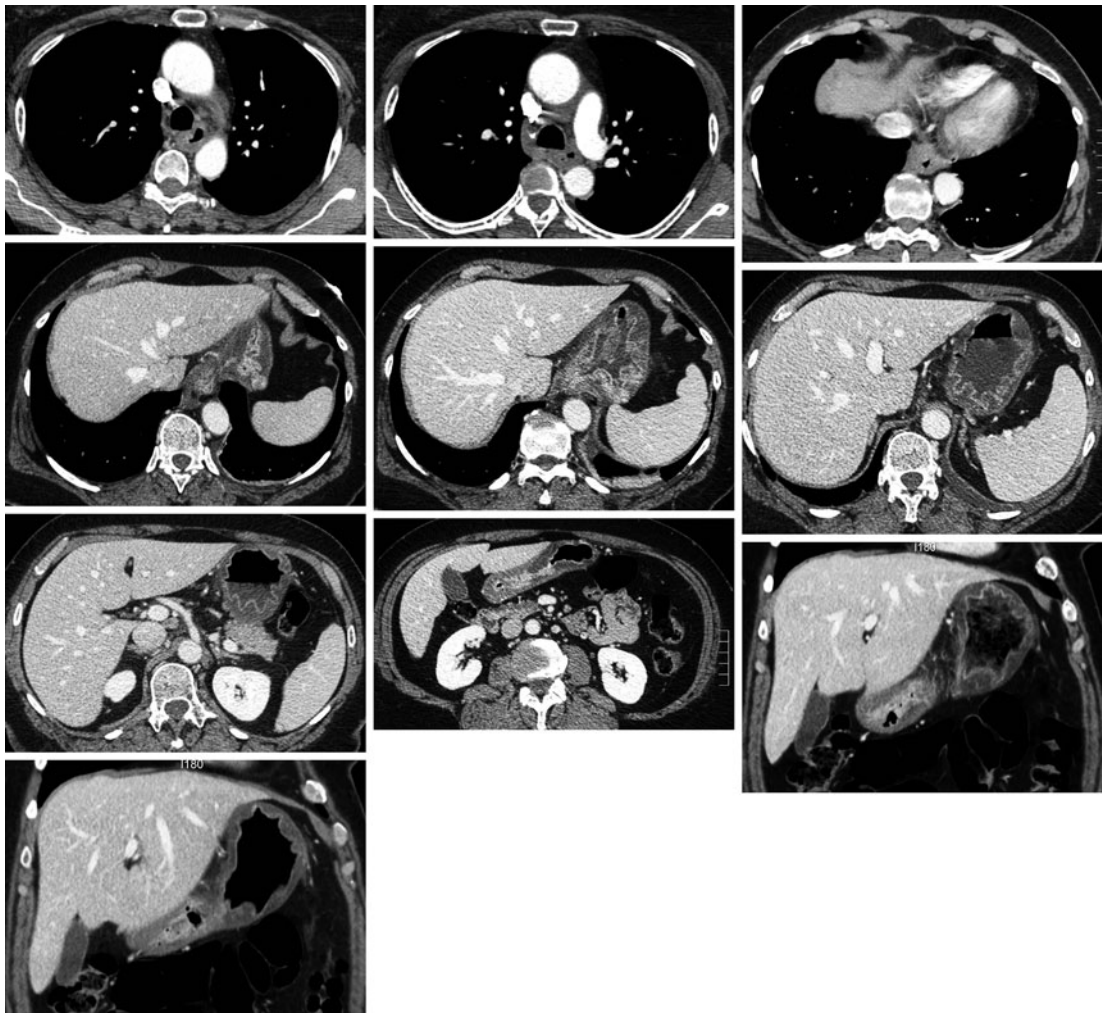

Fig. 4 Acute oesophagitis and gastritis after ingesting bleach. The CT scan confirmed diffuse circumferential thickening of the lower oesophagus and all gastric walls with marked submucosal oedema

Caustic gastritis is the consequence of accidental or voluntary ingestion of corrosive substances, usually acids (Fig. 4).

In both cases, a CT scan shows inflammatory parietal thickening and contributes to revealing early complications and associated lesions.

1.2.5 Idiopathic Inflammatory Gastritis May Quite Rarely Present in an Acute Clinical Picture

- Crohn's disease only affects the stomach in 2–7% of patients in whom the classic ileal and/or colic locations coexist.
- Eosinophilic gastritis is often seen in the context of eosinophilic gastroenteritis but it may be encountered in association with many other conditions, such as food allergies (eggs, milk, soya proteins), collagenosis, digestive parasitosis, gastric cancer, lymphoma, Crohn's disease, vasculitis, drug allergies or *H. pylori* infection. The eosinophilic infiltrate can be limited to the mucosa or extend to the whole intestinal wall.

1.3 Diagnosis of Acute Gastritis by CT Scan

The semiotic data on which the positive diagnosis of acute gastritis and possibly the orientation of the aetiological diagnosis may be based need to be

considered. Moreover, the direction taken by the differential diagnosis must take into consideration the other possible causes of 'acute' thickening of the gastric wall.

1.3.1 Positive Diagnostic Data for Acute Gastritis on a CT Scan

- The main CT scan signs are seen in major transmural acute inflammatory conditions, in particular in bacterial phlegmonous gastritis, and combine:
  - localised or diffuse thickening of the gastric wall with visible stratification indicating the oedema of the muco–submucosa complex; enhancement of the mucosal and muscle capillary networks, seen better in venous phasis with the 'target sign' appearance and best perceived if the stomach is distended with liquid (Jung et al. 2007). In other cases, a 'double halo' appears, defined as the juxtaposition of two concentric rings, the more internal, hypodense ring corresponding to the oedema of the submucosa, while the external hyperdense ring corresponds to the thickening of the muscularis propria. This semiotic data are valuable for differentiating inflammatory parietal thickening from tumoral thickening (specially from an adenocarcinoma in which the collagen fibrous reaction stroma can be objectified by late enhancement in the post-equilibrium phase).
  - Oedematous hypertrophy of regular or nodular rugae is easier to analyse in a stomach distended with liquid.
- Phlegmonous gastritis is a suppurative bacterial infection of the gastric wall. It can present in a diffuse or localised form, generally antral. Clinically, it appears in the form of an acute abdomen combined with an infectious syndrome, epigastric pain, nausea, vomiting—sometimes haemorrhagic in heavy drinkers—often following a serious respiratory infection. The different imaging techniques show the presence of localised or more diffuse parietal thickening, specially concerning the submucosa, that can be difficult to differentiate from a gastric adenocarcinoma in a scan (and by endoscopy!). The acute clinical context of the revelation, a septic and inflammatory picture in laboratory tests, an inflammatory reaction of the peritoneal serosa (thickened appearance with persistent enhancement, surrounding liquid reaction, etc.) even, in certain cases, the appearance of a partially liquid parietal collection (gastric wall abscess) may orientate the diagnosis.
- The presence of gas in a thickened gastric wall is a key sign that can be observed in various circumstances which must be clearly differentiated:
  - Emphysematous gastritis is either a form of bacterial infectious gastritis caused by microorganisms producing large quantities of gas (by a mechanism similar to that incriminated in emphysematous cholecystitis) or phlegmonous gastritis of bacterial or fungal origin which can form an abscess (Jung et al. 2007; Loi et al. 2007). The appearance in the scan combines more or less extensive thickening of the gastric wall, generally in the fundus and the greater curvature with irregular bubbles of gas in spots or bunches; these gas bubbles remain in place whatever the subject's position or the degree of aspiration by the naso-gastric ~~tube naso-gastric~~ aspiration.
  - Gastric emphysema is usually a regular linear infiltration of the gastric wall, which is not thickened or little thickened, by gas from the gastric lumen, the surface of the peritoneal serosa or from the connections of the stomach with the oesophagus or duodenum. Gastric emphysema usually results from barotrauma without any infectious bacterial involvement; it is particularly seen in chronic liquid and or gas distension of the stomach ~~whatever the mechanical~~ (neoplastic antro-pyloric stenosis or stenosis of inflammatory origin by an ulcerative disease) or functional nature (gastroparesis) (Figs. 5 and 6) (Buyt et al. 2003). This gastric emphysema is usually asymptomatic and is generally reabsorbed without treatment. A case has been reported in a 16-year-old adolescent boy, after ingestion of a large quantity of Coca Cola. It was combined with hepatic portal venous gas and duodenal wall emphysema in a serious clinical condition which resolved with treatment (Hadas-Halpren et al. 1993).

The prognosis for these various conditions is therefore very different, and the presence of gas in the gastric wall must be carefully correlated with the clinical situation and probabilistic physiopathological hypotheses to correctly orientate therapeutic management.

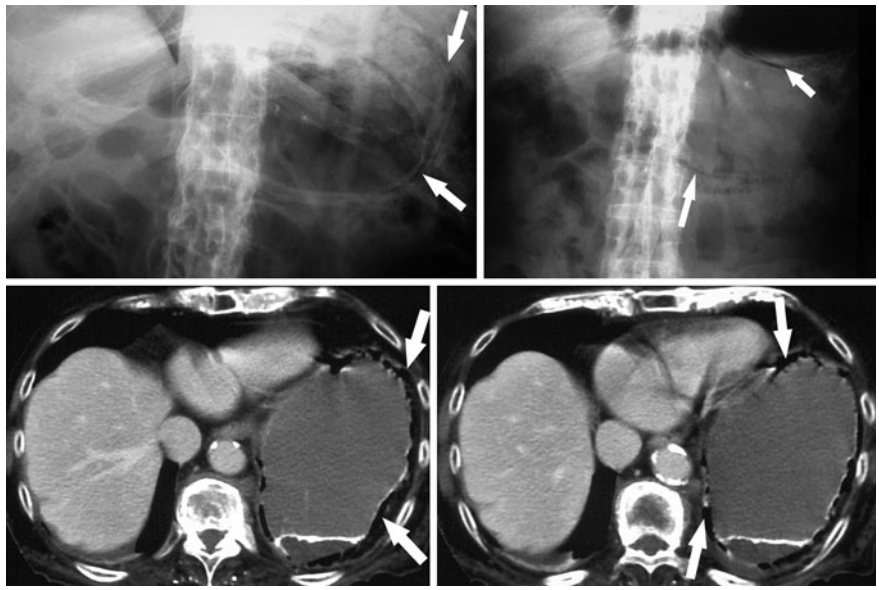

Fig. 5 Gastric emphysema in a patient with ankylosing spondylitis. The intraparietal gaseous images in a stomach distended with liquid are already clearly visible on the plain film pictures. The CT scan confirms the cystic gaseous dissection. The gastric emphysema occurred with gastroparesis. Clinical and radiological resolution after aspiration of the gastric contents

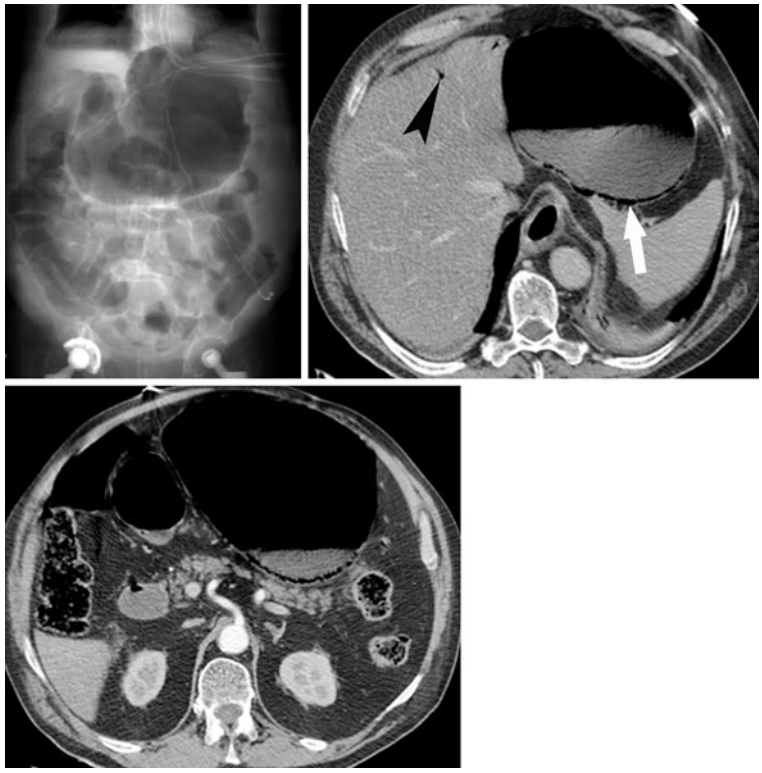

Fig. 6 Gastric emphysema in a 74-year-old patient with hepatic portal venous gas, on D3 after fitting a total hip prosthesis. The abdominal plain film showed considerable gaseous distension of the stomach and, to a lesser degree, of the small intestine and colon, the whole picture corresponding to postoperative ileus. The CT scan confirmed the parietal gaseous dissection of the stomach (*white arrow*) and hepatic portal venous gas (*point of black arrow*). The condition evolved favourably after resumption of transit

1.3.2 Differential Diagnosis of Acute Gastritis on a CT Scan

A not inconsiderable number of acute abdominal clinical pictures can be accompanied by marked thickening of the gastric wall with muco-submucosal oedema, the origin of the 'target' signs on the scan section. Causes may be local or more remote; they must be looked into systematically to avoid upsetting diagnostic or therapeutic orientations.

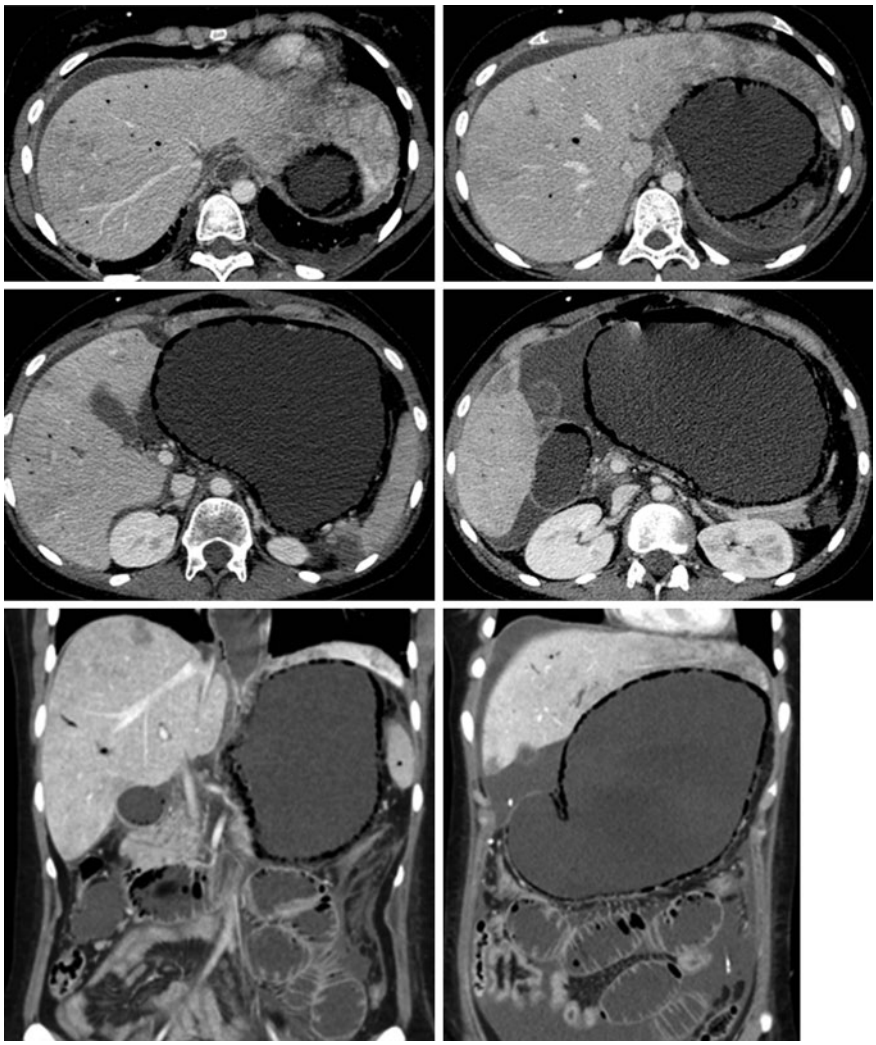

Fig. 7 Gastric necrosis. Transmural gastric necrosis in a 24-year-old patient with an antiphospholipid antibody syndrome and high-grade stenosis of the coeliac trunk by the arcuate ligament of the diaphragm. A CT scan was performed because of hyperalgesic painful epigastric symptoms. The CT scan confirmed the presence of massive gaseous dissection of the walls of the stomach which were not enhanced after contrast medium injection. Presence of a large quantity of intraperitoneal fluid but the patient has a ventriculoperitoneal shunt for a cerebral expansive lesion. Hepatic portal venous gas is present. The surgical procedure confirmed transmural necrosis of all the gastric walls

- Parietal pneumatosis of the stomach, seen in the context of a painful acute abdomen and usually accompanied by intrahepatic portal venous gas, must be clearly differentiated from parietal emphysema. It is associated with the absence of parietal enhancement after injection of contrast medium and generally indicates transmural ischaemic necrosis of the stomach (Fig. 7). It can be observed after starting chemotherapy in carcinomas (Fig. 8).
- Portal hypertensive gastropathy is a rare cause of digestive haemorrhage, and is generally not very severe in cirrhotic patients. Its probably multifactorial physiopathological mechanism remains open to discussion, involving raising the pressure in the portal system, increasing splanchnic blood flow and local modifications to the regulation of microcirculation. The prevalence of portal hypertensive gastropathy varies from 7 to 98% in the series published, depending on the diagnostic criteria and methods chosen for selecting patients, etc. The circumferential thickening of the gastric wall with an image of parietal stratification by submucosal oedema, seen in severe portal hypertension with ascitic decompensation, may suggest the diagnosis (Figs. 9 and 10) but it is known that there is no correlation between the portal pressure values measured, the severity of the cirrhosis, the degree of cellular dysfunction and the severity of the gastropathy when viewed endoscopically (Curvelo et al. 2000). Multiphase exploration objectifies defective segmental or subsegmental perfusion of the mucosa of the gastric body or

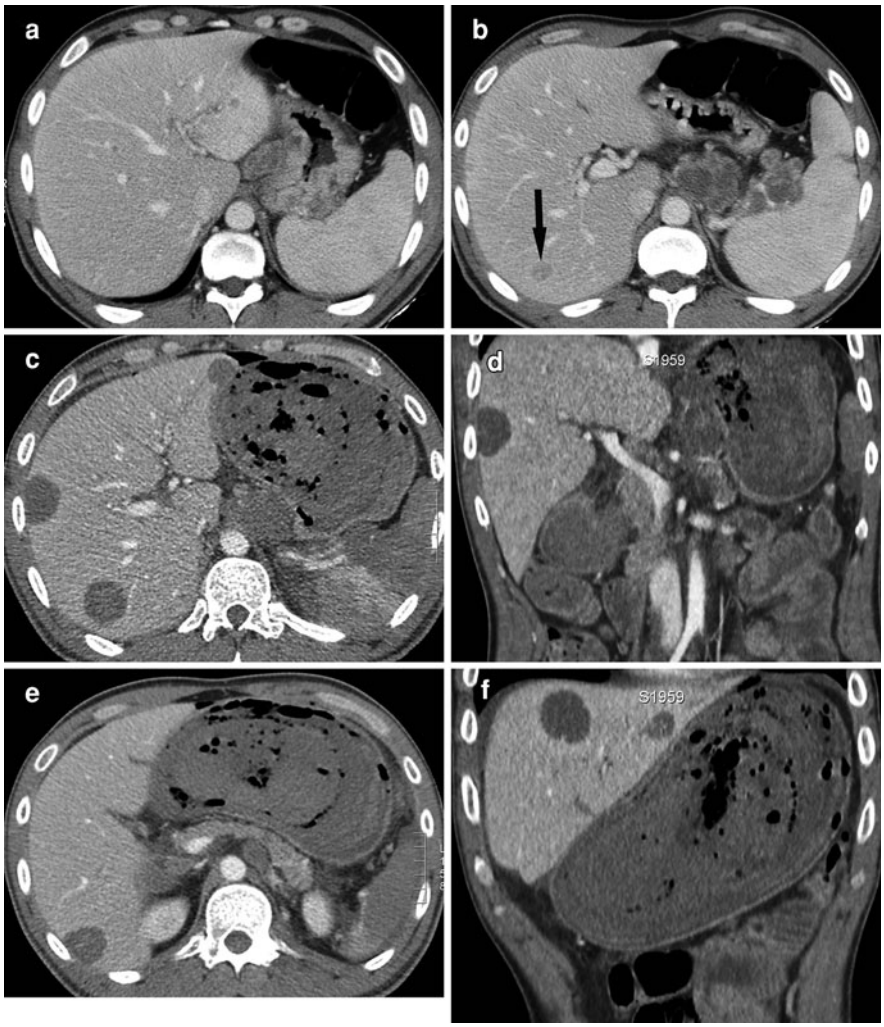

Fig. 8 Tumoral necrosis in a 30-year-old patient with a very large anaplastic gastric adenocarcinoma. **a**, **b** Initial findings: massive thickening of the gastric wall with very voluminous hypodense adenopathies of the lesser omentum, coeliacs and splenic hilum. Presence of hepatic metastasis (*black arrow*). **c**–**f** A week after the start of chemotherapy: massive necrosis of the gastric wall tumour with lack of parietal enhancement and gaseous dissection. Massive metastatic and lymph node dissemination

fundus. This has been observed to disappear in the portal phase or during post-equilibrium in 75% of cases of endoscopically controlled portal hypertensive gastropathy but in only 11% of cirrhotic patients without endoscopically visible gastropathy. This method would therefore seem to provide useful, more specific information for diagnosis (Kim et al. 2008).

- Inflammatory reactions of the gastric wall during acute pancreatitis are frequent (1/3 of cases) and must be clearly identified. They have been known for a long time (Balthazar 1979) and predominate on the posterior wall, resulting in generally extensive oedematous muco-submucosal thickening in contact with anterior peri-pancreatic infiltrations and collections, whether recent or organised (true pseudocysts) (Figs. 11, 12, and 13). Analogous images can be seen on contact with infected collections (left subphrenic abscesses) complicating ulcerous or surgical perforations (Chen et al. 2007; Brown et al. 1982).
- Persistent pseudotumoral hypertrophy of the gastric wall, in a context of epigastralgia which may be major, which is resistant to anti-ulcer treatment, in a young subject (in his 20 or 30s), with heterogeneous density and above all progressive enhancement increasing in the areas of tissue in the post-equilibrium phase, indicating the significant presence of collagen, should bring to mind an inflammatory pseudotumour (Fig. 14). Only surgical ablation can confirm the diagnosis as endoscopic biopsies cannot reach the significant areas.

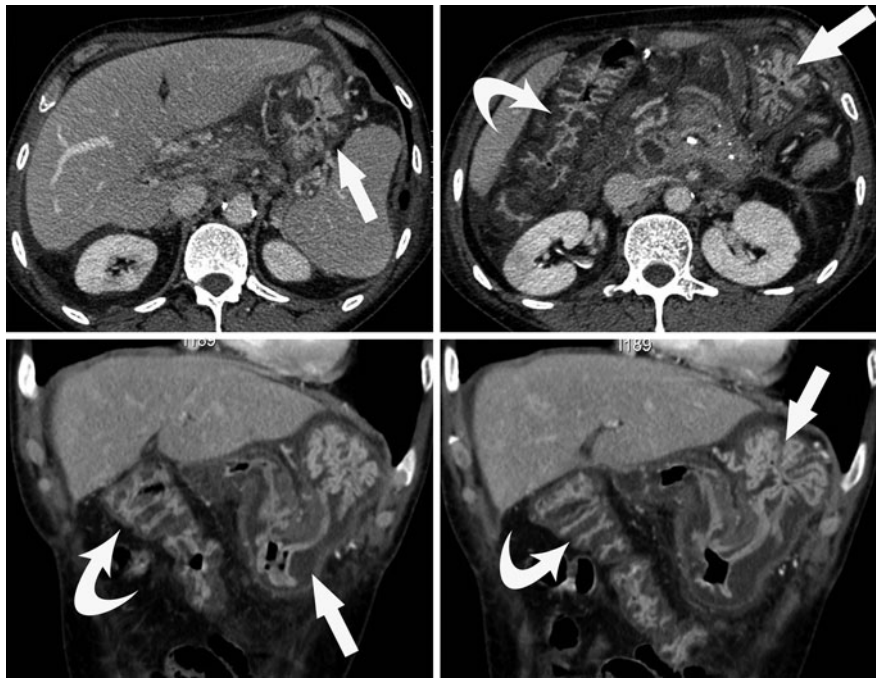

Fig. 9 Portal hypertensive gastritis. A 72-year-old cirrhotic patient with portal cavernoma and chronic calcifying pancreatitis. Very considerable submucosal oedema of the stomach wall (*straight white arrow*) and colon (*curved arrow*) in a context of extreme generalised oedema due to decompensated cirrhosis. The parietal submucosal thickening exceeds 15 mm in the body of the stomach

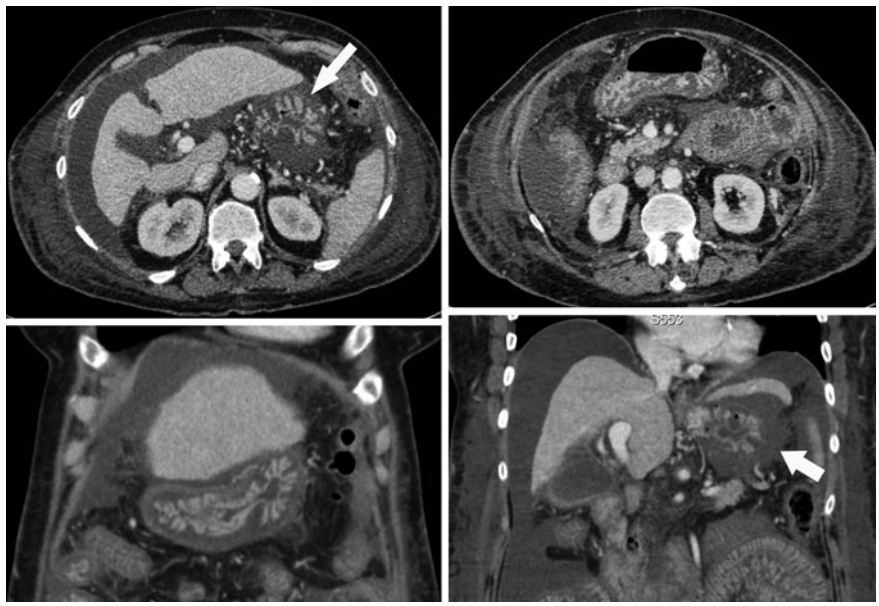

Fig. 10 Portal hypertensive gastritis in a patient with ascitic decompensated cirrhosis. Very considerable submucosal oedema of the whole stomach predominantly in the fundic region (*white arrow*)

- The presence of gastric trichophytobezoar, generally seen in trichotillomaniac girls, is associated with diffuse regular thickening, with intense, persistent enhancement of the gastric wall and no hypertrophy of the rugae (Fig. 15). The trichophytobezoar may extend a long way into the lumen of the duodenum and small intestine, producing Rapunzel's syndrome, named after the Grimm brothers' story with this name.
- Other possible causes of inflammatory thickening of the gastric wall which can be seen in the clinical context of an acute abdomen include:
  - Lymphocytic varioliform gastritis, for which an immunoallergic origin is plausible. It appears

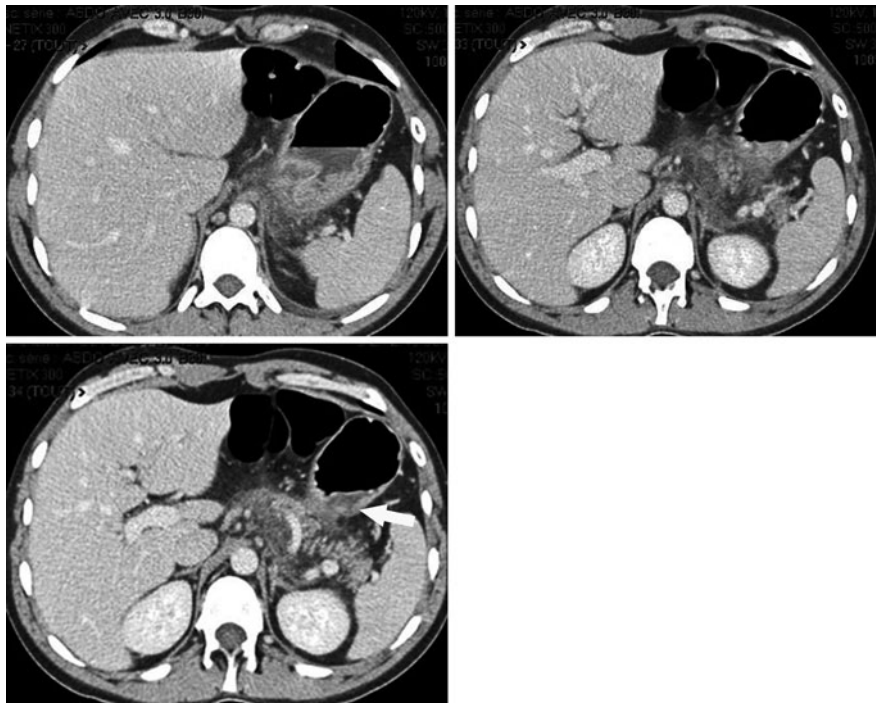

Fig. 11 Inflammatory reaction of the posterior wall of the fundus during acute caudal pancreatitis. The CT scan objectifies the parietal thickening limited to the floor and posterior wall of the fundus (*white arrow*) in direct relation with moderate inflammation of the caudal pancreas

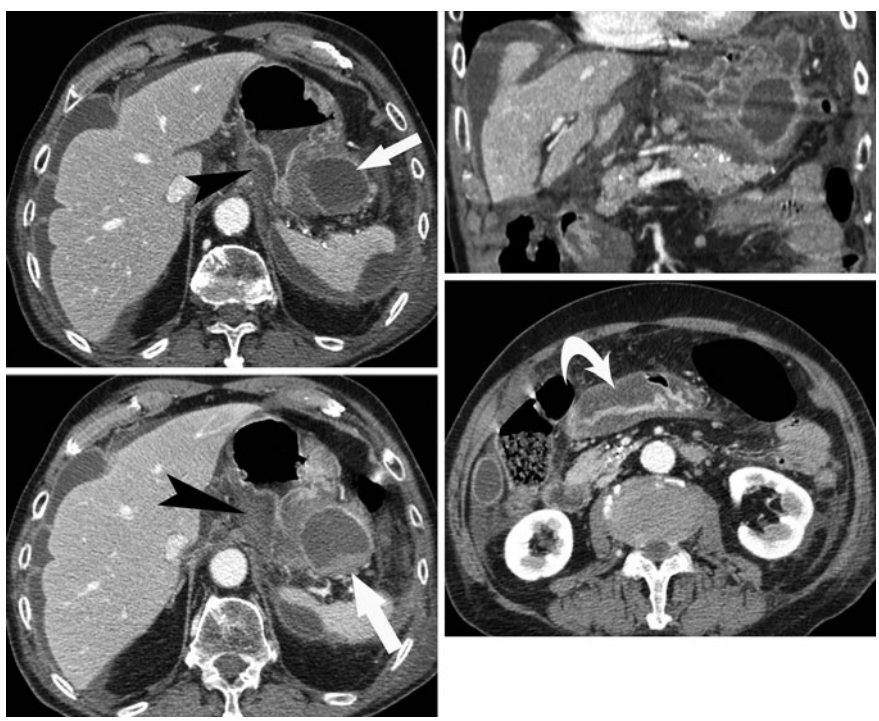

Fig. 12 Acute caudal pancreatitis with pseudocyst and peritoneal fluid effusion. The CT scan confirms massive gastric parietal thickening affecting the lesser curvature (*point of black arrow*), the fundus and the antral region (*curved white arrow*). The pseudocyst (*white arrow*) is the site of bleeding responsible for the hyperdensity of the lower part. Gastric parietal thickening is massive, exceeding 20 mm

as parietal thickening and large rugae with umbilicated nodules viewed by endoscopy, an abundant liquid content in the stomach sometimes accompanied by gastric loss of proteins with oedema and hypoalbuminaemia. Associations with coeliac disease, collagenous colitis,

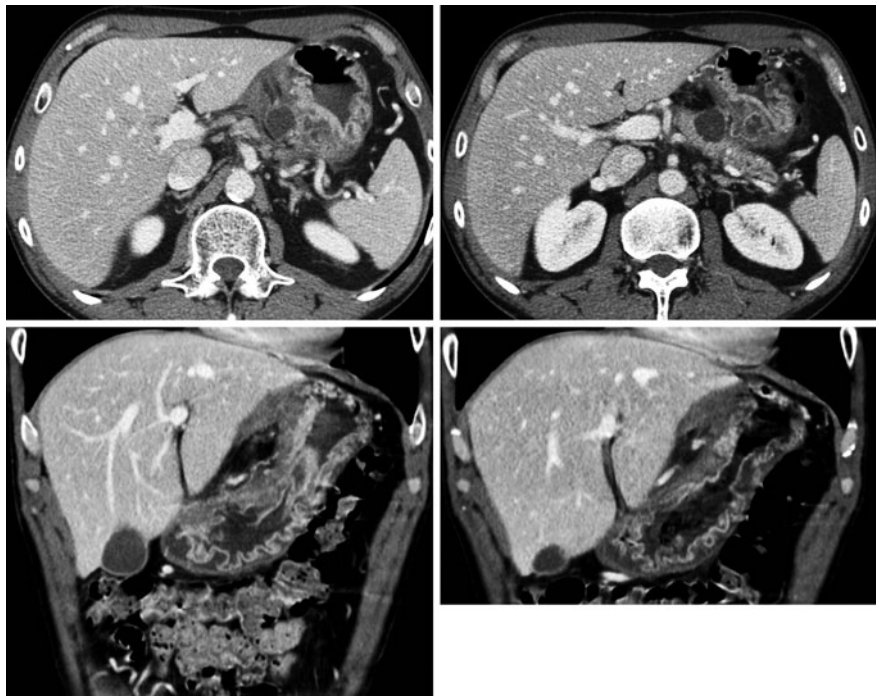

Fig. 13 Acute pancreatitis with small caudal pancreatic pseudocyst. Presence of a diffuse oedematous inflammatory reaction of the whole of the stomach wall

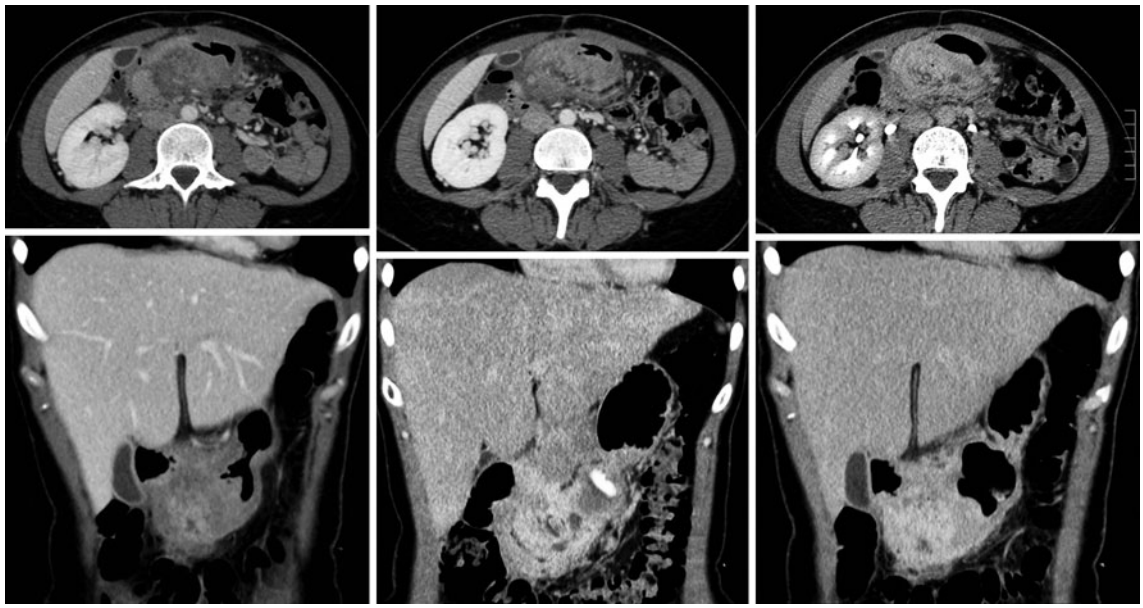

Fig. 14 Gastric inflammatory pseudotumour in a young (27-year-old) patient. Painful epigastric symptoms, ulcerous pain developing over the past 4 months resistant to PPI. Presence of parietal pseudotumoral thickening of the antral region with massive persistent enhancement accentuated on late images. Surgical procedures confirmed the diagnosis of an inflammatory pseudotumour complicating an intramural antral ectopic pancreas.

lymphocytic colitis, Ménétrier's disease and *H. pylori* gastritis have been described.

– Zollinger–Ellison syndrome with diffuse thickening of the gastric wall and rugae in the fundus with spontaneous liquid distension of the lumen by hypersecretion linked with hypergastrinaemia (Fig. 16). The endocrine lesion must be sought in the pancreatico-duodenal region; this may be

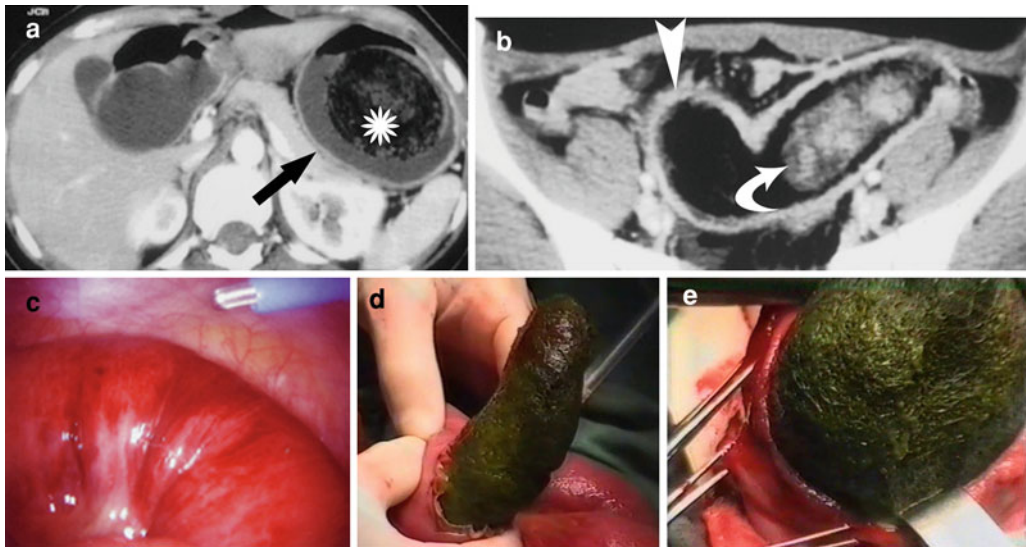

Fig. 15 Gastric and jejunal bezoar in a young (16-year-old) female trichotillomaniac patient. The heterogeneous spherical content of the gastric fundus (*white asterisk*) is accompanied by thickening of the gastric wall with significant persistent enhancement (**a**). Identical images are seen in the proximal jejunum (**b**) which confirmed a heterogeneous compact endoluminal foreign body (*curved white arrow*). Surgical exploration showed very considerable transparietal inflammatory reaction of the proximal jejunum affecting the serous side (**c**). The surgical procedure extracted the jejunal bezoar (**d**) and the gastric trichophytobezoar (**e**)

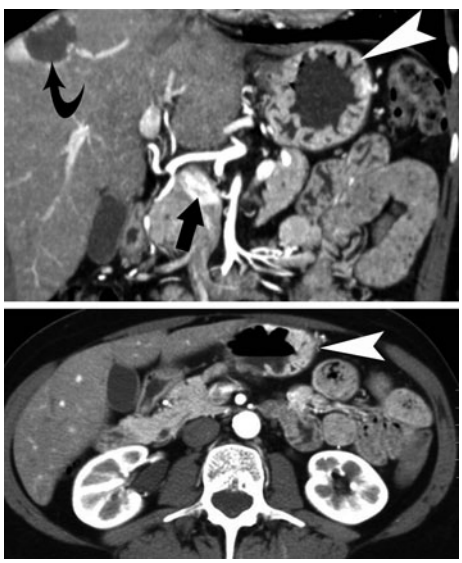

Fig. 16 Gastrinoma in a 50-year-old patient. The CT scan objectifies considerable hypertrophy of the gastric mucosal rugae with major enhancement (*point of white arrow*) and the presence of a hypervascularised lesion of the head of the pancreas corresponding to the gastrinoma (*black arrow*). There is a hepatic angioma (*curved black arrow*)

small in size and hypervascularised or more voluminous and metastatic (Fig. 17).

– Ménétrier's disease, which can be revealed by pseudo-ulcerous pain.

The parietal thickening is related to considerable hypertrophy of the muscularis propria and to cystic dilatation of mucosal glands, at the origin of a cerebriform hypertrophy of the fundic rugae (Fig. 18). The disease essentially affects middle-aged men, usually alcohol and tobacco users; it is accompanied by leakage of gastric protein, causing oedema and microcytic anaemia.

– Submucosal fatty metaplasia or submucosal pseudolipomatosis, thickening of the submucosa with a regular homogeneous fatty density. Initially described in chronic inflammatory conditions of the small intestine and colon after corticosteroid therapy, then in patients treated by chemotherapy in which it can appear within a few weeks (Muldoney et al. 1995), it is, in fact, not unexceptional to see it in routine examinations, generally in overweight adults, more often males. It is easily identified because of its typical

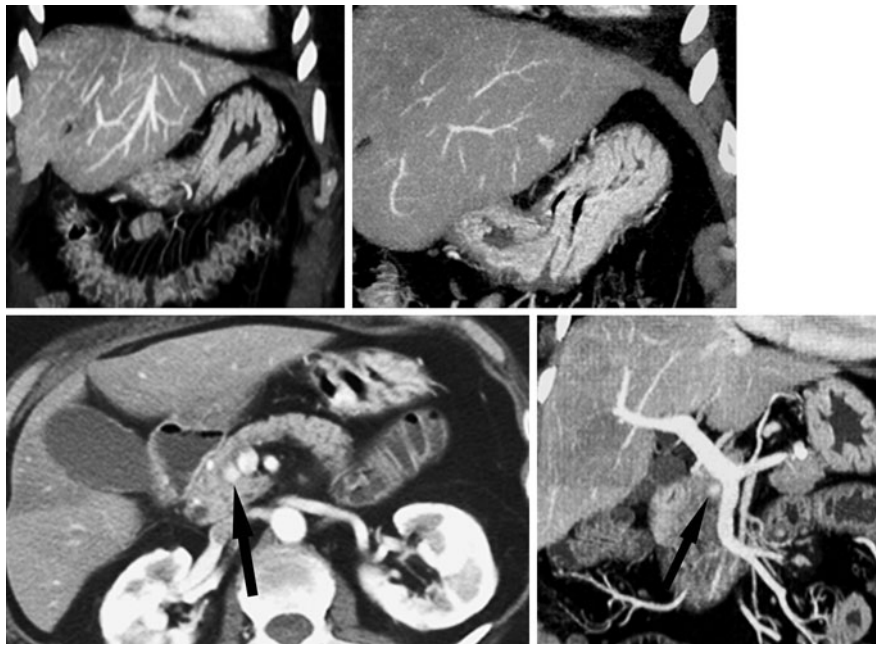

Fig. 17 Gastrinoma. Very considerable hypertrophy of the mucosal rugae over the whole stomach with major persistent enhancement. No sign of liquid hypersecretion. Presence of a small hypervascularised nodule at the isthmus of the pancreas corresponding to the gastrinoma

appearance (fat halo sign) (Fig. 19). It is much less frequent in the stomach than in the small intestine and colon where it has been observed in more than 20% of adult patients in a North American study. Obviously, it must not be confused with an acute inflammatory condition (Harisinghani et al. 2003).

2 Acute Duodenitis and Enteritis

2.1 General Characteristics (Macari and Balthazar 2001; Thoeni and Cello 2006; D'Almeida et al. 2008; Macari et al. 2007)

Acute inflammatory and infectious conditions of the small intestine may benefit from an emergency scan providing useful diagnostic data depending on the location and size of the anomalies objectified. Evidently, only severe infectious conditions that are resistant to treatment or which occur with major repercussions on the general condition of the patient necessitating hospitalisation will result in a scan being performed.

In general, these are above all localised transmural lesions which can be identified better, with Crohn's disease and its complications in the lead.

In acute diffuse conditions, whether they are infectious or inflammatory, the scan objectifies submucosal oedema with parietal stratification of the loops, producing 'target' or 'double halo' images that can be easily analysed in venous time (70 s after injection at 3 ml/s) Gore et al. 2000. In the face of this, in an acute clinical context, a number of hypotheses must be discussed (arterial ischaemia, capillary hyperpermeability, congestion by portal venous stasis, etc.), certain of which may be supported by abdominal/pelvic exploration, as a general rule complemented by thoracic exploration if there are no contra-indications for the radiation risk (young subjects and women of childbearing potential).

In all cases, the clinical context and laboratory tests are fundamental for orientating the diagnosis: a history of abdominal pain and diarrhoea, a state of acquired immunosuppression, a recent stay in a country where there are endemic parasites, a purpuric rash on the lower limbs, a marked inflammatory syndrome seen in laboratory tests, etc., are all signs providing pointers for the right direction which one needs to know how to find out by precise, directed questioning and clinical examination (Fig. 20).

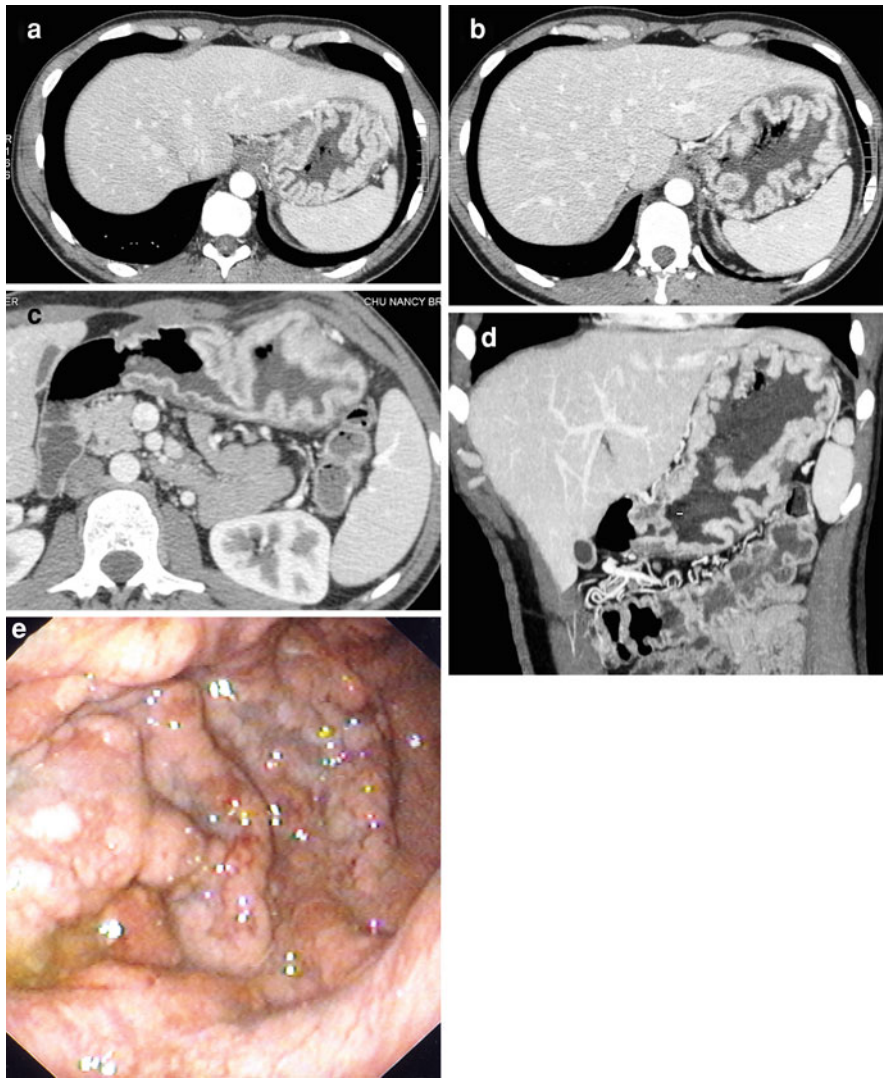

Fig. 18 Ménétrier's disease. The CT scan objectifies considerable hypertrophy with an encephaloid appearance of the rugae of the stomach, major enhancement of the mucosa and submucosal oedema (**a–c**). The stomach is hypersecretory (**c–d**) and endoscopy (**e**) confirms the encephaloid hypertrophy of the gastric rugae

2.2 Acute Infectious Conditions of the Duodenum and Small Intestine (Mazzie et al. 2007)

The aetiology of acute infections of the small intestine is very different depending whether they are occurring in an immunosuppressed context or not.

In immunocompetent subjects, acute intestinal infections are rarely investigated by a CT scan (Macari et al. 2007). The possible causes include:

- *Giardia lamblia*, a parasitosis found throughout the world, the occurrence of which can be favoured by hypogammaglobulinaemia. This parasite attaches itself to the mucosa of the proximal small intestine (duodenum and jejunum) without invading it, resulting in a local inflammatory response with parietal thickening by submucosal oedema and problems of motricity, more difficult to assess on cross-sectional images than in opacifications.
- *Ascaris lumbricoides* is responsible for one of the world's most frequent parisitoses. Its diagnosis is possible with CT scan, which shows the worms surrounded by endoluminal liquid or distended by gas and thus appearing as linear clear areas (Hommeyer et al. 1995; Sherman and Weber 2005; Rodriguez et al. 2003). Occlusion by an endoluminal ascaris cluster is a classic way of detecting the parasite in countries where it is strongly endemic. It can be accompanied by perforation revealing the condition.

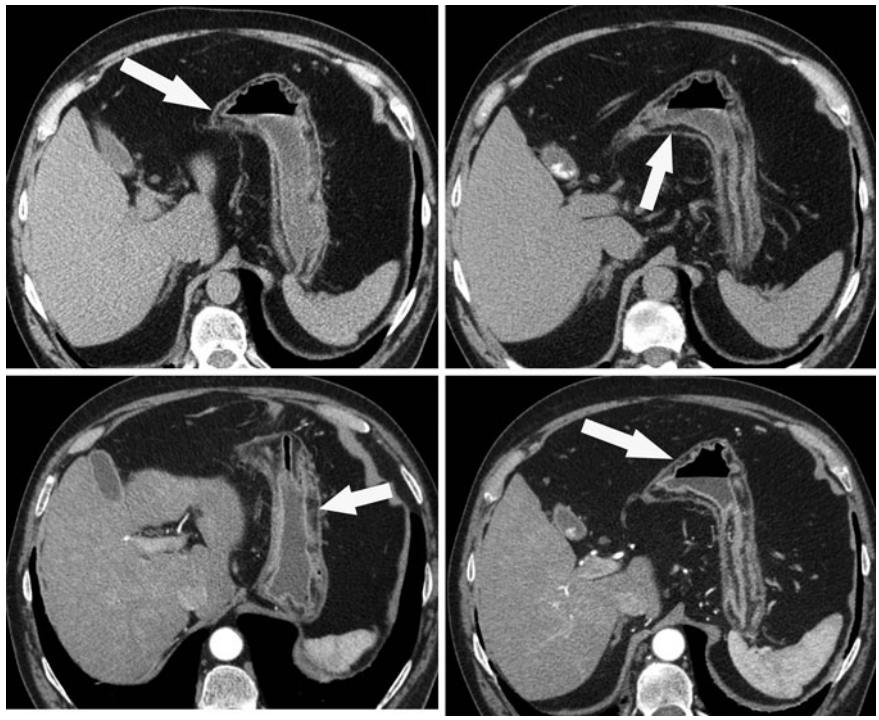

Fig. 19 Gastric parietal pseudolipomatosis. Regular submucosal thickening of fatty density has occurred over the whole gastric wall (*white arrow*). This appearance coexists with deep android adiposity. The patient is asymptomatic

- Anisakiasis is an acute infection caused by the larva of Anisakis, a marine nematode ingested with preparations made from raw fish (sushi, raw anchovies) or undercooked fish. The larva causes an inflammatory reaction of the intestinal wall, which can be of major importance and can extend over a length of about 10 cm. Lesions of the small intestine may coexist with gastric and colic involvement and can be accompanied by allergic symptoms. Occlusive forms have been reported, often combined with ascites. Blood hypereosinophilia occurs frequently. From the point of view of a CT scan, the condition is very often situated in the terminal ileum and can extend into the adjacent colon. The pathological examination confirms the presence of an eosinophilic infiltration. An increase in specific IgE can support the diagnosis and must therefore be systematically assayed when faced with any histologically diagnosed 'eosinophilic gastroenteritis' (Fig. 19). Diagnostic certainty (endoscopic and/or in laboratory tests) can avoid a surgical procedure since the condition develops favourably following drug-induced (or spontaneous) elimination of the parasite (Repiso et al. 2003; Ishida et al. 2007; Ortega-Deballon et al. 2005).
- *Strongyloides stercoralis* causes parasitosis which is particularly frequent in the tropics but also occurs in temperate regions (e.g. the southeast of the United States). The larva of the parasite penetrates the organism through the skin, migrates into the respiratory system then is coughed up and swallowed and reaches the duodenum at the adult stage. The process can repeat itself resulting in a cycle or endogenous reinfestation and visceral dissemination, particularly encephalic in immunosuppressed patients. The adult female infiltrates the lamina propria of the duodenum and jejunum causing an inflammatory reaction there of variable intensity depending on the severity of the infestation. There is generally very marked hypereosinophilia. In severe forms, the dominant feature, in CT scan, is major liquid distension of the digestive lumen giving it the appearance of paralytic ileus or occlusion combined with more or less marked thickening of the wall. The colon can be simultaneously affected with a scan appearance close to that of ulcerative colitis (Kothary et al. 1999).
- Ileitis or enteritis due to Salmonella and Shigella, the most frequent acute infectious conditions found in countries with a high standard of living, is seen as regular circumferential thickening of the last

15 cm of the ileum wall readily extending to the adjacent colon to a lesser degree. The 'target sign' appearance of the acute lesions is found particularly in Shigella infections; satellite adenomegaly may be seen. The main sources of infection are poultry meat, eggs, milk products and undercooked meat. The severity of the symptoms is very variable and can be intense in immunosuppressed subjects. The 'cholera-like' very severe forms with watery diarrhoea and the dysenteric, sometimes haemorrhagic forms are combined with hyperallergic symptoms with fever and possible multiple organ failure (Shigellosis) (Balthazar et al. 1966; Van Wolfswinkel et al. 2008).

- *Campylobacter jejuni* also affects the ileocaecal junction but it is generally a self-limiting disease. On the other hand, *Salmonella typhi* can lead to severe, sometimes haemorrhagic inflammation, essentially affecting the small intestine causing hyperaemic and oedematous changes; the depth involved can result in perforation of the intestine.
- Yersiniosis is caused by *Yersinia enterocolitica* and *Yersinia pseudotuberculosis*. They affect the right ileocolic region, presenting clinically as a pseudo-appendicitis or as acute feverish diarrhoea with terminal ileitis combined with mesenteric lymphadenitis. *Y. enterocolitica*, along with the bacteria in the Salmonella order and Campylobacter, is the cause of the majority of generally self-limiting bacterial cases of gastroenteritis. *Y. enterocolitica* above all affects young subjects. The disease is due to ingestion of contaminated food (pork meat, milk, water, tofu) or to blood transfusion. Faeces remain contaminated for 90 days after clinical resolution. Diagnosis of yersiniosis using imaging techniques is more the domain of echography than of CT scanning, considering the age of the subjects. The inflammatory circumferential thickening of the walls of the terminal ileum, the presence of regional mesenteric adenopathy, the absence of an image of appendicitis are all arguments in favour of this diagnosis. Severe septicaemia can be seen with the development of visceral abscesses specifically during treatment of iron overload with deferoxamine chelators (Abcarian and Demas 1991; Matsumoto et al. 1991; Antonopoulos et al. 2008).

In subjects seropositive for human immunodeficiency virus, opportunistic acute infections of the small intestine are now much less frequent due to the efficacy of antiretroviral treatments which reduce the viral load and the frequency of all of these complications. It is thus in cases of resistance or escape from these treatments (HAART or highly active retroviral treatment) that a CT scan may be made on a patient with an acute abdomen or a febrile diarrhoea syndrome in a seriously impaired general state of health. The degree of immunosuppression measured by the CD4 lymphocyte count is still the best guide for the radiologist, when reading CT scan images. The imaging technique only supplies a guide to a range of probable causes; it only provides characteristic features in 12% of cases, and its essential role is to contribute to evaluation of the severity of the condition, and above all to screening for surgical complications (Wu et al. 1998; Koh et al. 2002).

- A CD4 lymphocyte count >200/ml indicates relative immunocompetence and M tuberculosis is then the probable cause of the digestive disorders; with a lymphocytes count between 200 and 100/ml, there is some degree of immunosuppression and many infectious agents may be involved while below 100 CD4/ml CMV infections, infections by MAI and cryptosporidiosis must be envisaged in the first instance. The other significant elements which can be objectified and sought on CT scans include the presence of hepatic and/or splenic infectious focal lesions and light-centred adenopathies before and after injection of contrast agent, indicating tuberculosis, as well as the predominance of intestinal lesions on the terminal ileum and caecum.
- In MAI infection on the contrary, the area affected is frequently jejunal or diffuse and resembles both radiologically and histologically that seem in Whipple's disease (Figs. 20 and 21). Visceral locations are rare, and the adenopathies are homogeneous because in these patients, the very low CD4 lymphocyte count, generally <100 and 50 CD4/ml, does not allow a caseous necrosis immune response to occur.
- Cryptosporidiosis is most often seen in deeply immunosuppressed states with CD4 lymphocyte levels <50/ml but it can occur months later in the development of the disease with levels of CD4/ml <200. The area affected is generally proximal, involving the duodenum/jejunum and upper ileum, but it can affect the whole digestive tract. It is not accompanied by adenopathy. The intestinal walls

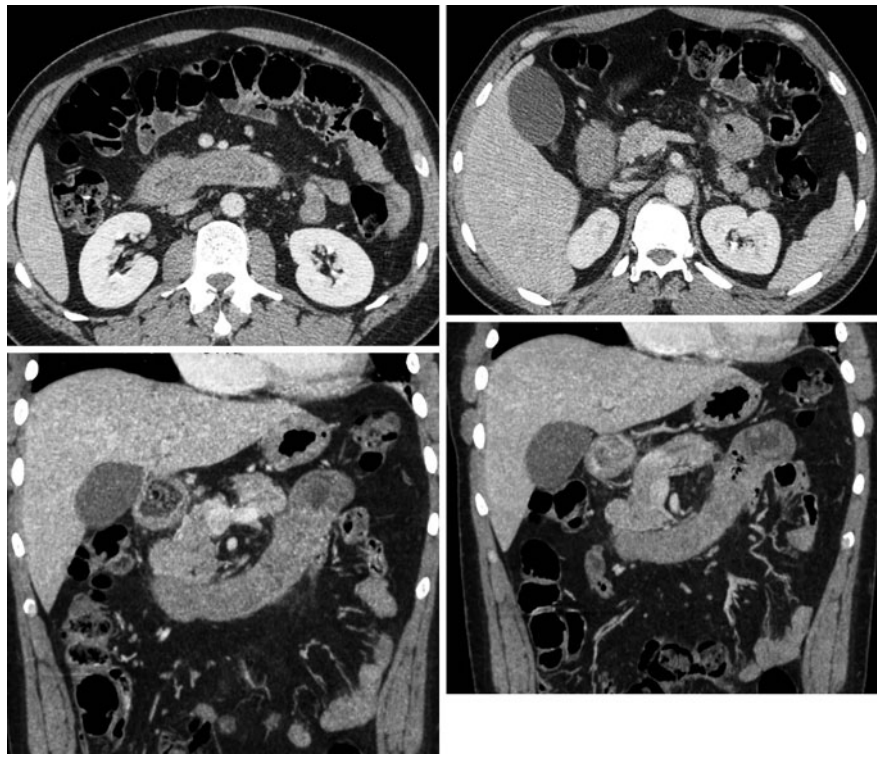

Fig. 20 Eosinophilic enteritis. Regular circumferential thickening of the walls of the duodenum and hypereosinophilyl. Biopsies confirmed the diagnosis of eosinophilic enteritis without a parasitic cause being found

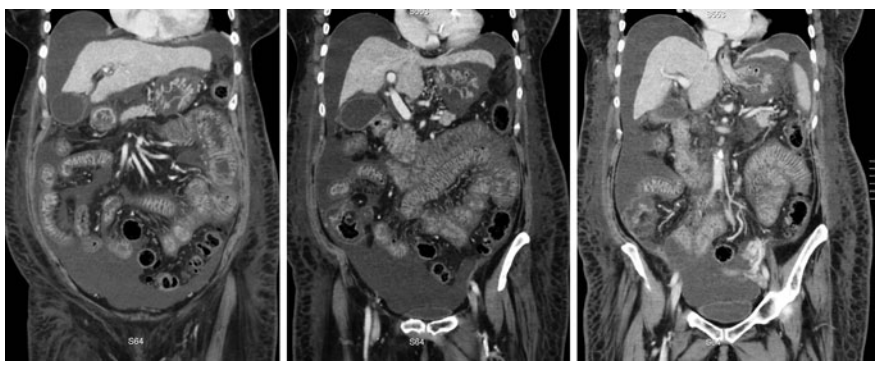

Fig. 21 Portal 'enteritis'. Ascitic oedematous decompensation of hepatic cirrhosis accompanied by diffuse submucosal oedema of the small intestine, colon and stomach. The multifactorial causes of this submucosal oedema include portal hypertension and hypoproteinaemia

are moderately and regularly thickened and the loops are the site of liquid distension which can be considerable (Fig. 22). Cryptosporidiosis, like CMV infection, can be accompanied by cholangiopathy shown on the CT scan images as a generally moderate dilatation of the intra- and extra-hepatic bile ducts. The presence of a low stenosis of the main bile duct, in the papilla region, and the predisposition for developing alithiasic cholecystitis is the other characteristic describing biliary involvement.

- Gastrointestinal infections with *Isospora belli* cannot be distinguished clinically and radiologically from cryptosporidiosis; only microscopic examination of the faeces can provide an exact diagnosis.
- CMV intestinal infections preferentially concern the ascending caecum or the whole colon

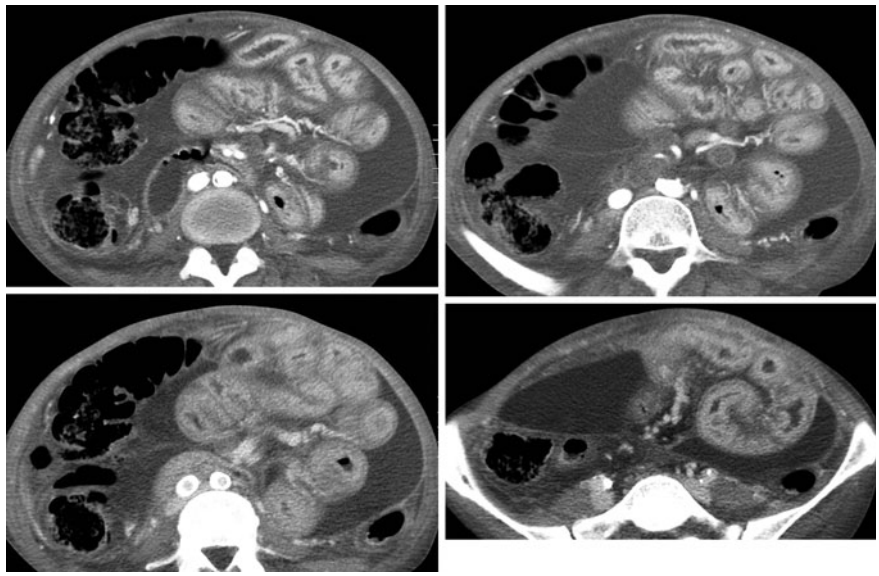

Fig. 22 Enteritis due to MAI in an HIV+ patient. Considerable submucosal thickening of the walls of the small intestine with thickened and enhanced mucosa and serosa, abundant ascites. Laboratory investigations confirmed infection by atypical mycobacteria

(pancolitis); the small intestine is rarely affected. The regular parietal thickening can be massive in severe forms, or even have a pseudotumoral polypoid appearance. It does not occur with adenopathy. The coexistence of biliary involvement is a diagnostic argument.

- Histoplasmosis can, in countries where it is endemic, give rise to digestive involvement when the CD4 count is < 100/ml. As with tuberculosis, it can be a primary infection or a reactivation. In the disseminated forms of histoplasmosis, the intestine is involved in 75% of cases. The aspects seen on a CT scan are identical to those encountered in tuberculosis: localisation on the terminal ileum and the ascending colon, generally regular concentric parietal thickening, sometimes with pseudotumoral stenosis, infiltration of the adjacent peritoneum, hypodense regional adenopathy in the mesentery and retroperitoneum and possible hepato-splenic, adrenal glands and peritoneal nodular infectious focal lesions.
- In neutropenic subjects, in particular during aplasia induced to treat malignant haemopathy by stem cell transplantation, cancer chemotherapy or after organ transplant, it is essential to have good knowledge of the factors orientating the diagnosis of acute gastrointestinal events after allogeneic haematopoietic stem cell transplantation, and in particular, the length of time since the patient became aplasic (Schmit et al. 2008; Kirkpatrick and Greenberg 2003; Beckett and Olliff 2005).
- Chemotherapy-related mucositis is the inevitable consequence of lesions of the mucosal barrier because of the toxicity of the aplastic drugs used. The initially inflammatory epithelial lesions ulcerate (approximately at D 15) then heal. In the CT scan, diffuse or segmental circumferential thickening of the wall of the digestive tube is seen with mucosal hyperaemia (alternation of hypoperfused and hyperperfused areas within the thickened walls). In general, there is little or no infiltration of the mesentery, nor is there adenopathy. The gastrointestinal tract is generally affected diffusely, and the clinical symptoms are moderate secretory diarrhoea and/or abdominal pain.
- Neutropenic enterocolitis most often involves the caecum (typhlitis or caecitis) but it is not unusual for the terminal ileum to be affected which is a good argument in favour of the diagnosis and against that of infectious colitis, in particular of pseudomembranous colitis due to *Clostridium difficile*. The affects are multifactorial combining mucositis lesions, deep neutropenia, disturbances induced in the microbial flora and a haemorrhagic necrosis component of ischaemic origin. The CT scan shows circumferential thickening of the walls of the caecum (typhlitis), of the ascending colon and the terminal ileum, combined with moderate inflammatory infiltration of the adjacent peritoneum. In general, there is little ascites and no adenopathy. In severe cases, signs of ischaemia

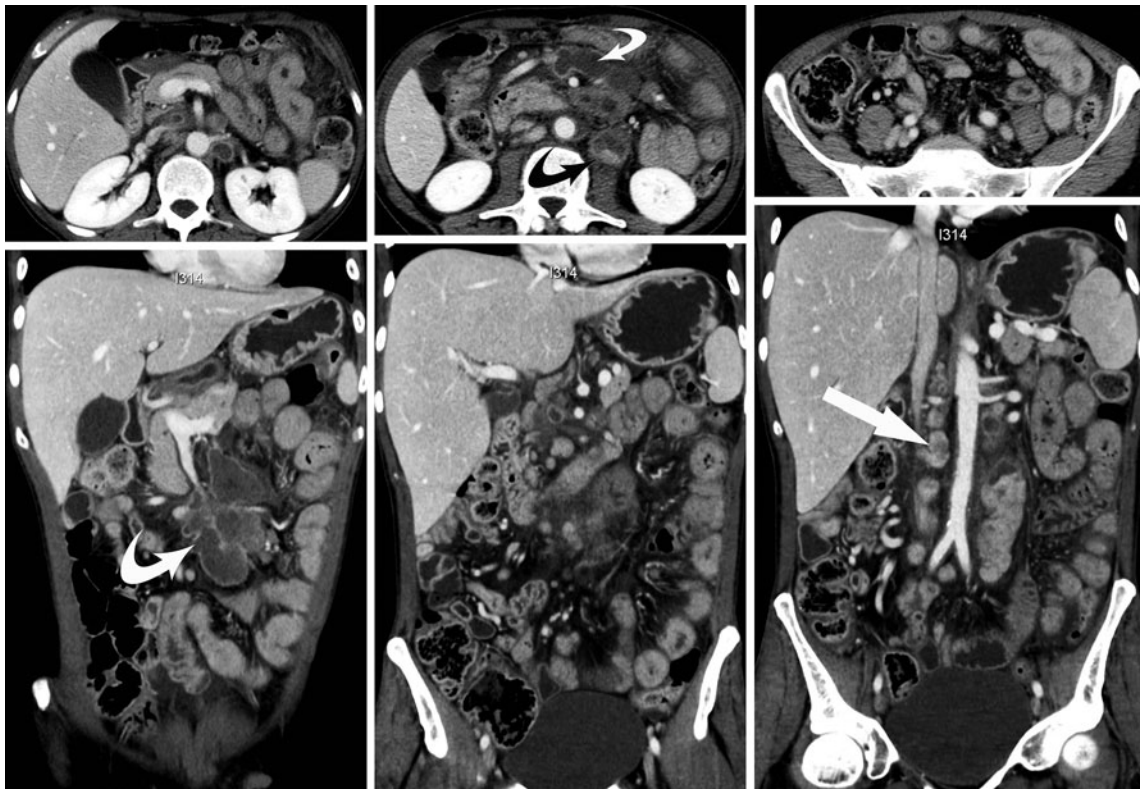

Fig. 23 Enteritis due to MAI in a young HIV+ patient. Jejuno-ileal parietal abnormalities (thickening, hyper-enhancement) accompanied by hypodense retroperitoneal mesenteric adenopathies, suggestive of tuberculosis, but the clinical context and in particular the very strong immunosuppression suggest more a MAI infection which will be confirmed by laboratory investigations

appear (parietal pneumatosis, lack of mucosal enhancement) or local complications: abscess, pneumoperiteum.

- The graft-versus-host reaction; After haematopoietic stem cell allotransplantation, the return to normal in immunological terms occurs in three phases:
  - During the first 10–30 days, the immune system is severely impaired by pancytopenia; local defences are weakened by mucitis.
  - Between 30 and 100 days after transplantation, the neutropenia corrects itself but with a delay for the lymphocytes, the cause of continued cellular and humoral immune deficiency. Infectious complications are thus frequent. An acute form of graft-versus-host reaction appearing as abdominal pain, and secretory diarrhoea can develop once the graft is functional. On CT scan, abnormally intense enhancement of the mucosa of the digestive tract is seen very clearly predominating on the small intestine. This corresponds histologically to a destroyed mucosa, replaced by richly vascularised granulation tissue. The intestinal loops are generally distended with liquid with a circumferential submucosal oedema and a comb sign in the corresponding mesentery; on the other hand, there is only rarely infiltration of the mesentery. The alternation of healthy areas with pathological areas and the clinical context of haematopoietic stem cell transplantation are obviously major factors for making a diagnosis (Fig. 23).
  - After 100 days, a chronic form of graft-versus-host reaction can be seen which occurs mainly as a malabsorption syndrome without expression in imaging techniques.
- Acute fungal infections, in particular the disseminated invasive forms of aspergillosis which may be the origin of necrotic ulcerative enteritis particularly concerns the ileum, the walls of which are

circumferentially thickened and the neighbouring mesentery infiltrated. There is a very high risk of perforation and the symptoms are often not very specific, sometimes limited to a fever and abdominal pain without clear guarding. A CT scan is therefore of fundamental importance to confirm the parietal and peritoneal abnormalities of the ileocolic junction (Tresallet et al. 2004).

- Mucormycosis. This is seen in subjects treated for a malignant haemopathy, in immunosuppressed transplant patients, keto-acid diabetics, patients with cardiac and renal impairment and during chelator treatment for iron overload. Gastrointestinal involvement is uncommon, affecting the stomach more that the small intestine and colon. It is seen on CT scan as a circumferential diffuse thickening of the walls of affected digestive segments, which often coexist with hepatic focal localisations (Suh et al. 2000).

2.3 Vascular Acute Inflammatory Conditions of the Duodenum and Small Intestine

2.3.1 Acute Manifestations of Vasculitis on the Small Intestine (Ha et al. 2000; Ahn et al. 2009)

The majority of forms of vasculitis, in any case those concerning small- and medium-sized vessels, may affect the small intestine. We should think of them each time we see images in a young patient suggesting ischaemic lesions in an unusual site, for instance in the stomach, above all the duodenum (ischaemia in the duodenum is practically always related to vasculitis), or the rectum, or when the small intestine and colon are simultaneously diffusely affected or when other viscera are affected, e.g. the uro-genital system.

- Henoch-Schönlien purpura (sometimes called rheumatoid purpura) is vasculitis of small vessels characterised by the presence of serum IgA and the precipitation of immune complexes in the arterioles, capillaries and venules. Most frequent in children, where it usually develops benignly, a quarter of cases are seen in adults, more often in men (sex-ratio 2/1), with a poorer prognosis because of more frequent and more severe renal involvement. The clinical triad of acute abdominal pain, arthralgia and palpable purpura must bring this diagnosis to mind. Abdominal pain occurs in 44% of cases of Henoch-Schönlien purpura in adults, preceding the skin lesions in 10–15% of patients. The CT scan shows an inflammatory thickening with double halo signs of the digestive segments affected, loss of mesenteric fat transparency with vascular engorgement, and adenopathy; ascites is present in the severest forms (Figs. 24 and 25). Complications due to perforation or occlusion by invagination are less frequent in children and the digestive lesions generally evolve favourably; the prognosis for the disease depends on the kidneys (Chung et al. 2006).
- Periarteritis nodosa (PAN) is a necrotising inflammatory vasculitis which affects the wall of small- to medium-sized muscular arteries leading to the formation of micro-aneurysms and stenoses. It particularly affects middle-aged men (in their 40 or 50 s; sex-ratio 2–3/1) who, in 36% of cases, are carriers of the hepatitis B virus. cANCAs are found in laboratory tests in a large majority of cases but they are not specific (since they are also found in Wegener's granulomatosis, in Churg–Strauss syndrome and microscopic polyangiitis). The digestive tract is affected in 50–70% of PAN cases, essentially the jejunum which is the site of extensive submucosal oedema which can be spontaneously hyperdense when it is haematic, with a double halo image on biphasic acquisitions after contrast medium injection. Perforations and stenoses are complications of the vascular lesions. The scan, even with the spatial resolution of current machines, cannot objectify the microaneurysms that are easily shown by angiography (Rhodes et al. 2008; Jee et al. 2000).
- Microscopic polyangiitis (hypersensitivity vasculitis; leukocytoclastic vasculitis) is a condition identical to PAN which affects small diameter vessels (arterioles, venules, capillaries). cANCAs are present in the majority of cases. Renal involvement is found in 90% of cases in the form of a necrotising glomerulonephritis. The effects on the digestive tract are identical to those seen in the other types of vasculitis: regular inflammatory thickening of the (generally ileal) walls, vascular engorgement, loss of mesenteric fat transparency, and ascites.

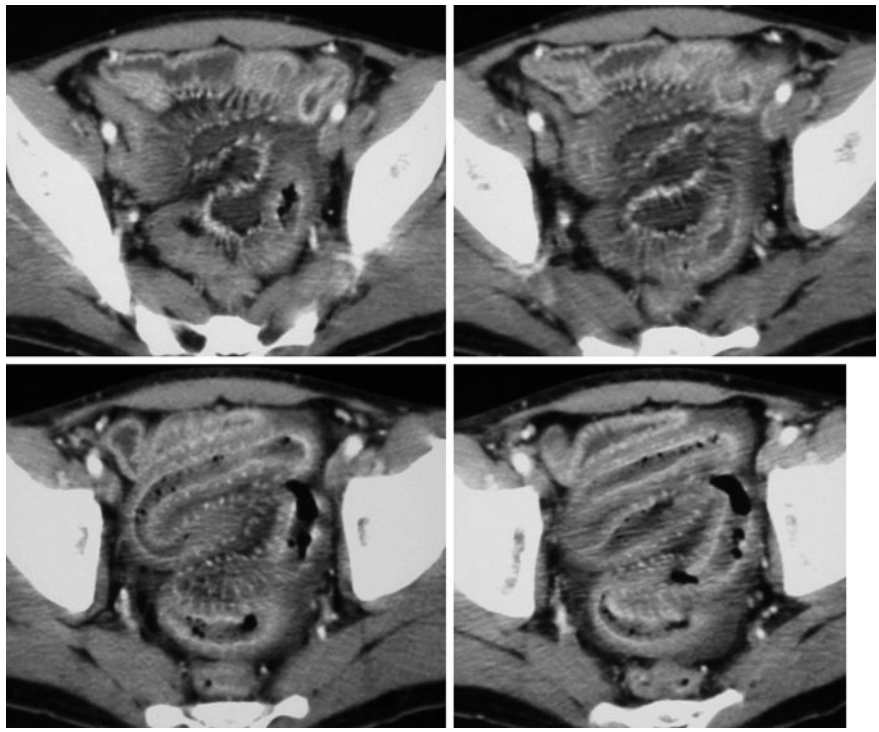

Fig. 24 Cryptosporidiosis and choleriform diarrhoea syndrome in an HIV+ patient with a CD4 level below 50/ml. Diffuse submucosal thickening of all the ileal loops with increased mucosal contrast, hypervascularisation with the vasa recta showing a comb sign. Diagnosis of cryptosporidiosis was confirmed by the laboratory tests

- Wegener's granulomatosis histologically affects the intestinal tract in 24% patients but is only expressed clinically in 10%. It affects the small intestine and colon. The appearance on CT scan is identical to that seen in other forms of vasculitis. Acute forms are linked to intestinal perforations or extensive massive ischaemic necrosis (fulminating necrotising enterocolitis) (Pickhardt and Curran 2001).
- Churg–Strauss syndrome can exceptionally occur on intestinal-mesenteric structures (20% of cases), either in the form of ischaemic lesions secondary to vasculitis, or by eosinophilic infiltration of the wall which can be the cause of an occlusion or haemorrhagic diarrhoea (Rha et al. 2000).
- Acute-disseminated lupus erythematosus (ADLE) is a necrotising vasculitis of autoimmune origin which preferentially affects young female subjects. The digestive effects are ischaemic, often haemorrhagic lesions consecutive to a real obliterating endarteritis. In physiopathological terms, the hypercoagulable state linked to the circulating antiphospholipid antibodies (anticardiolipin antibodies and lupus anticoagulant which are encountered in 30–40% of patients with ADLE, as against 2% in the general population) appears to be of decisive importance in the occurrence of digestive symptoms. We speak of an antiphospholipid antibody syndrome when there are not enough diagnostic criteria for ADLE. Perforative and occlusive necrotic complications are classic. The whole digestive tract can be affected but the predilection is for the territory of the superior mesenteric artery. In acute situations, the CT scan shows circumferential thickening of the walls of affected digestive segments, engorgement of mesenteric vessels with a particular arrangement of the vasa recta of the ileum related to its shortening (comb sign), and adenopathy. Polyseritis is frequent (peritoneal, pleural, pericardial liquid effusions, etc.) and evokes the diagnosis. A high incidence of urogenital tract lesions has been reported (lupus glomerulonephritis, cystitis, hydronephrosis by fibrous changes to the ureterovesical junction or following vesicoureteral reflux secondary to a detrusor spasm). In everyday practice, these data are of very limited interest because of their rarity (Pagnoux et al. 2003; Kaushik et al. 2001).
- *Behcet's disease*. The gastrointestinal tract is affected in 10–50% of patients with Behcet's disease. The lesions affect the ileo-caecal junction with a predilection for the terminal ileum and are

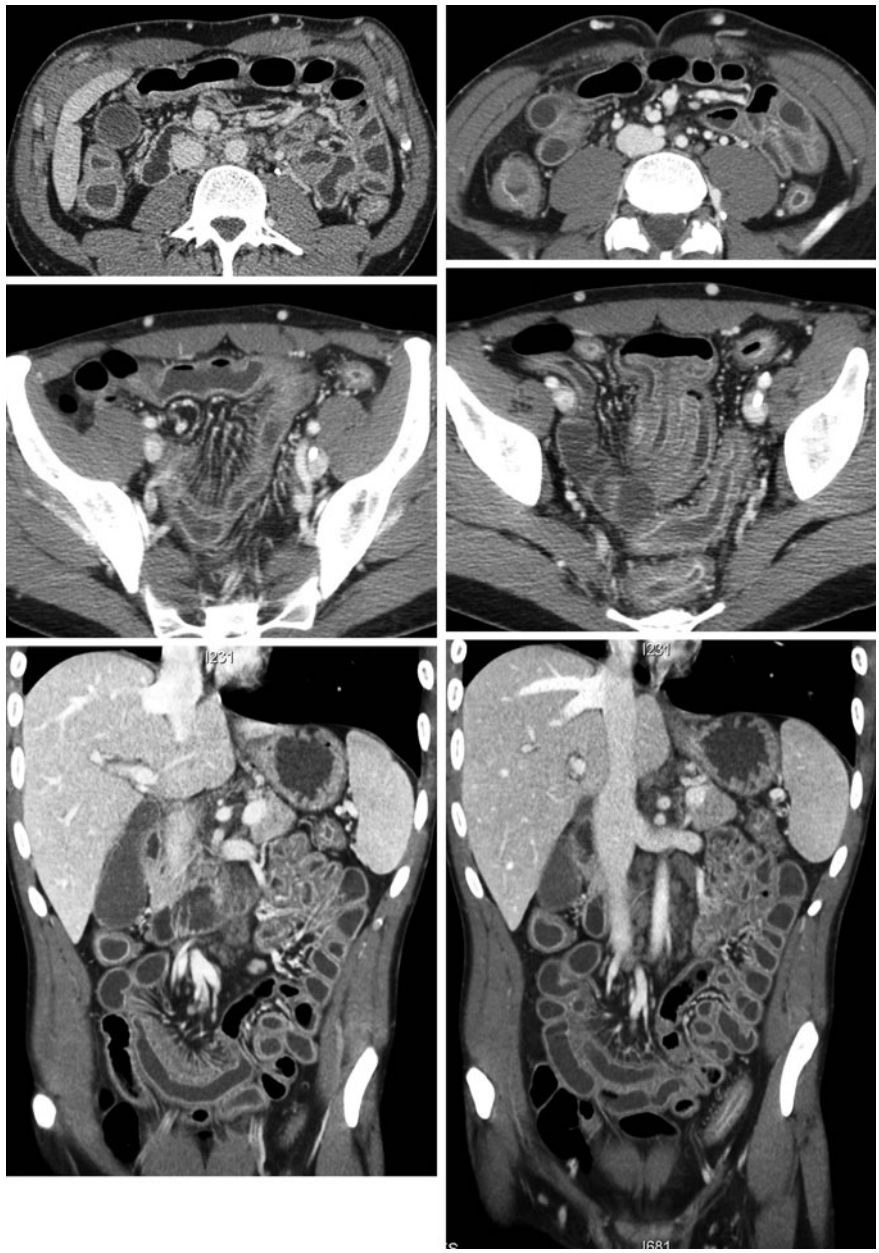

Fig. 25 Graft-versus-host reaction in a young patient who had received a transplant of haematopoietic stem cells. Diffuse thickening due to submucosal oedema with increased contrast in the mucosa of all the small intestine loops and the colon, in a graft-versus-host reaction

the consequence of an inflammatory vasculitis preferentially affecting the venules. The deep ulcerations typical of the disease may be transmural, which explains the frequency of local complications such as haemorrhage, perforation, fistulae, localised or diffuse peritonitis, for the diagnosis of which the CT scan is particularly valuable. Parietal thickening can be massive, localised, asymmetric with a polypoid appearance, pseudotumoral, ulcerated in the centre (Chung et al. 2001).

- *Rheumatoid vasculitis*. In longstanding severe rheumatoid arthritis, with marked disorders in laboratory tests and in particular in male patients, acute intestinal inflammatory symptoms may be seen related to leukocytoclastic vasculitis of the small venules. The diffuse thickening of walls of the small intestine and colon with double halo enhancement, the possible infiltration of the mesentery and the peritoneal liquid reaction are identical to what is seen in the other forms of vasculitis.

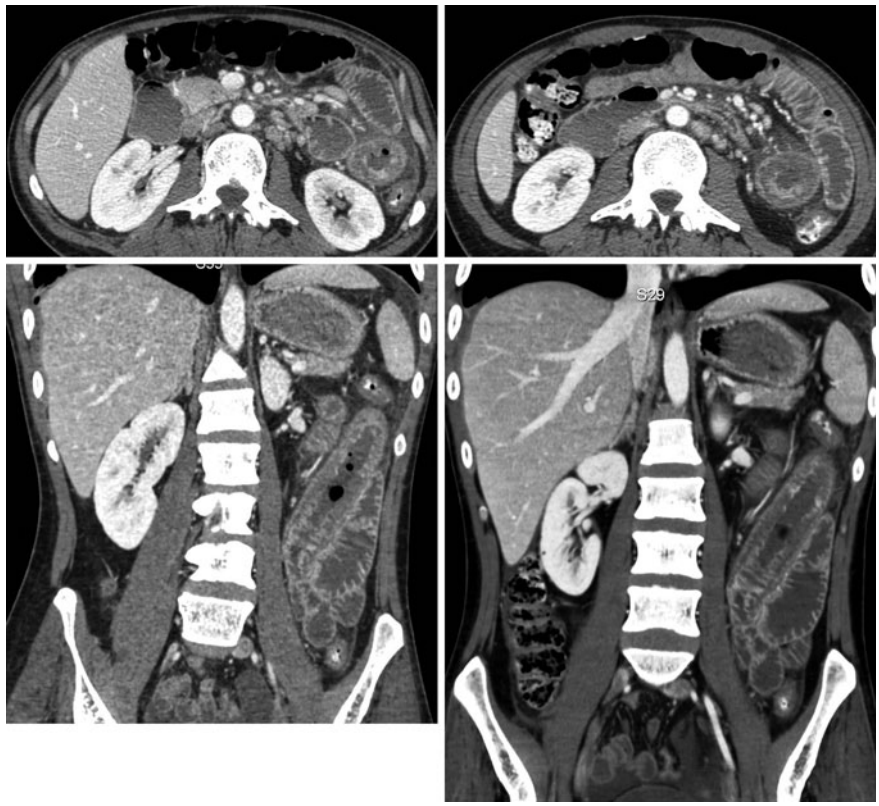

Fig. 26 Acute painful epigastric symptoms in a 29-year-old patient. The scan objectifies oedematous circumferential thickening of the proximal jejunal loops with discrete peritoneal fluid effusion accompanied by small mesenteric and peritoneal adenopathies. Purpura eruptions were seen on the lower limbs within hours of the examination confirming the diagnosis of Henoch-Schönlein purpura

2.3.2 Inflammatory Conditions with a Vascular Origin Excluding Forms of Vasculitis

- *Acute radiation enteritis of the small intestine (*Lyer et al. 2001*)*. This is seen during the weeks immediately following pelvic irradiation, most often after doses higher than 45 Gy for uterine cervical cancers. It appears as an oedematous parietal thickening of the loops of the small intestine in the pelvis and lower abdomen. Limitation of the lesions to the field of irradiation with clear demarcation is highly evocative for this diagnosis (Fig. 26). The condition is usually resolved in a few weeks with symptomatic treatment. This acute effect, related to the direct action of ionising radiation on the lining epithelium is different from chronic radiation enteritis which is related to an obliterating endarteritis, the cause of fibrosing inflammation producing stenoses and fistulae, generally appearing several months to several years (2 months to 30 years) after irradiation.
- *Angio-oedema (or angioneurotic oedema) of the small intestine*. Angioneurotic oedema is a non-inflammatory condition characterised by acute episodes of capillary hyperpermeability with oedema of the skin of the face and of the respiratory and intestinal mucosae. The gastrointestinal condition is expressed as an acute abdomen, generally resolving within 24–72 h but there is a potential risk of death by hypovolaemic shock. The appearance of watery diarrhoea in the final phase of the attack is good diagnostic evidence. CT scan images combine extensive inflammatory thickening of the walls of the digestive tract (stomach and small intestine) with liquid distension of the digestive lumens, congestion of mesenteric vessels and peritoneal fluid effusion. The cause of this disease is a hereditary or acquired lack of C1-esterase inhibitor, which should be determined by laboratory investigations. A personal or familial history of Quincke's oedema or minor forms (episodes of urticaria, transitory dyspnoea) should be sought but the digestive events may precede cutaneous and respiratory symptoms by several years. There are paraneoplastic forms associated with lymphoproliferative syndromes, autoimmune

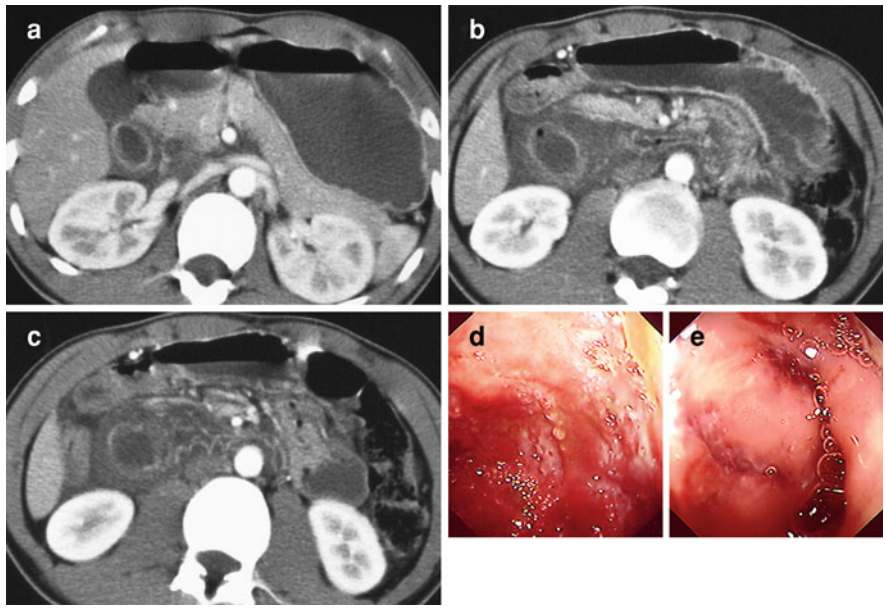

Fig. 27 Acute painful epigastric symptoms in a 21-year-old female patient. Considerable circumferential submucosal (**a–c**) oedema of the duodenum confirmed by endoscopy (**d**) which shows the hyperaemic (**d, e**) and haemorrhagic character of lesions due to Henoch-Schönlein purpura

diseases or cancers. Certain cases of this condition may be seen during treatment with conversion enzyme inhibitors (ACE inhibitors) (De Baker et al. 2001).

- *Buerger's disease (thromboangiitis obliterans)*. In young subjects who are usually heavy smokers this condition affects the vasa vasorum of arteries of intermediate size, leading to thrombosis and severe parietal inflammatory reactions. Digestive involvement is infrequent. Intestinal parietal symptoms are of the same type as those seen in vasculitis but an angioscan shows the segmental thromboses of the medium-sized arteries. The context (young subject, heavy smoker) is fundamental to the diagnosis (Marder and Mellinghoff 2000).
- *Vasculitis linked to cocaine*. After just one inhalation of cocaine, the picture of an acute abdomen can be seen on a CT scan with images analogous to those seen in acute or subacute ischaemia of the digestive tract (Fig. 27). The colon is most often the site but the small intestine alone can be affected, essentially the ileum. The histological similarities between Buerger's disease and vasculitis due to cocaine have led some authors to suggest that there are close links between these two conditions (Herrine et al. 1998; Hagan and Burney 2007).

2.3.3 Drug-Induced Enterocolitis (Chatelain et al. 2007)

Drug-induced enteropathies, particularly colic conditions, are frequent but in general are not investigated using imaging techniques because of the usually minor character of their clinical symptoms.

- *Drug-induced Ileitis*
  - Ulcerated ileitis lesions can be seen after ingestion of NSAIDs, but are much less frequent than colic conditions. One of their particularities as they develop is the formation of diaphragm-like stenoses (Fig. 28).
  - Corticosteroids, tablets of iron salts or potassium chloride may lead to lesions similar to those caused by NSAIDs.
  - In all cases, the clinical picture can be acute with bloody diarrhoea, and peritonitis through perforation may be seen. The severity of the condition is not related to how long treatment has been administered.
- *Enteropathy due to NSAIDs.* Diaphragm disease is a rare but specific complication of chronic prolonged use of NSAIDs. It is seen above all in middle-aged women taking NSAIDs over the long term for a rheumatic condition. It may be revealed clinically by an acute occlusive syndrome but usually attention is drawn to the condition by subocclusive episodes, chronic diarrhoea and

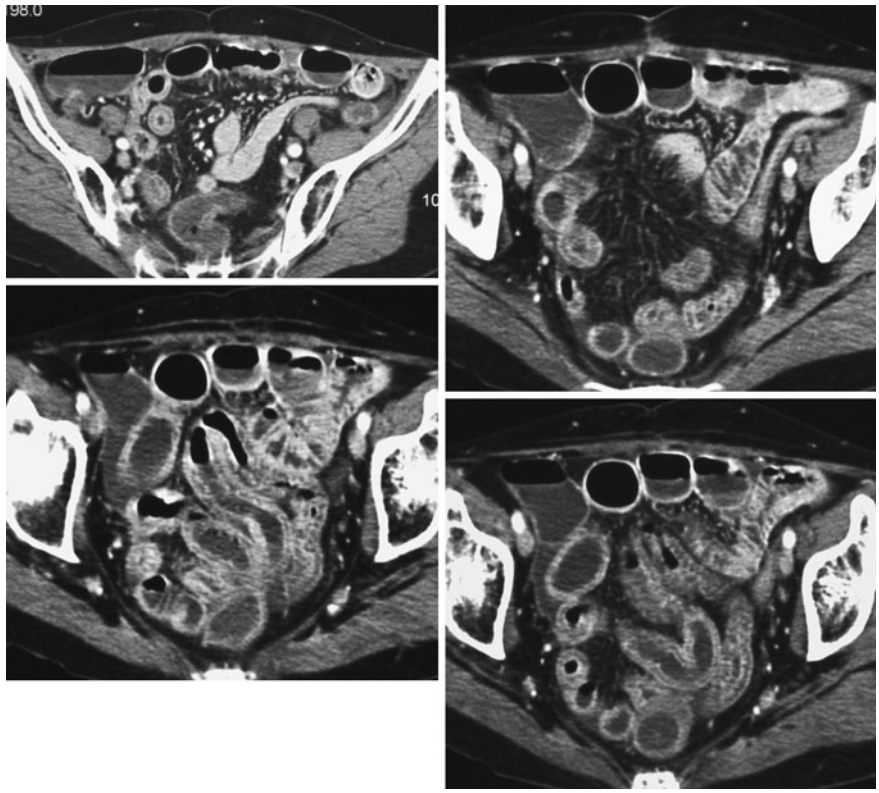

Fig. 28 Acute radiation enteritis. Regular moderate circumferential submucosal oedema of all the pelvic loops of the small intestine in a female patient irradiated during the previous 3 weeks for a neoplasia of the cervix of the uterus

weight loss. Fine fibrous circular membranes, perforated in the centre, develop in the ileal lumen, opposite valvules with which they interact. Histologically the submucosa is the site of fibrous transformation which would seem to correspond to the development of the ulcerous lesions caused by the NSAIDs (Zalev et al. 1996).

- *Necrotising ileocolitis due to treatment with kayexalate and sorbitol.* An ulcerated condition of the distal ileum and the right colon, often accompanied by partial or transmural necrosis, which can then be complicated by perforation with peritonitis, may be seen in renally impaired subjects treated with kayexalate for hyperkalaemia. In fact it is the sorbitol, a hypertonic solution administered to counteract the constipation induced by kayexalate, which would seem to be the cause of the necrotising ileocolic lesions, through its direct toxic action on the mucosa and the local hypovolaemia that it induces due to its hypertonicity, and which causes a low local flow that can lead to parietal necrosis.
- *Ileal melanosis*. Like the much more frequent colic melanosis with which it can be associated, this is the consequence of long-term treatment for constipation by laxatives of the anthraquinone group. It is a feature of pathological anatomy which is not visible using imaging techniques.
- *Clofazimine enteropathy*. Clofazimine belongs to the phenazine group used in the treatment of leprosy, atypical mycobacterial infections and sometimes in graft-versus-host reactions, and after being used for several months can accumulate in the mucosa of the small intestine, the mesenteric ganglia and the greater omentum, giving them a brown-black or orangey colouration by which the condition can be identified in a pathological anatomy examination. The clinical picture is a combination of abdominal pain, vomiting and diarrhoea.

2.4 The Acute Appearance of Crohn's Disease in the Small Intestine (Madureira 2004; Gourtsoyianni et al. 2009)

Crohn's disease can be revealed by an acute painful feverish episode with diarrhoea, or by an acute complication, such as an intestinal occlusion, deep

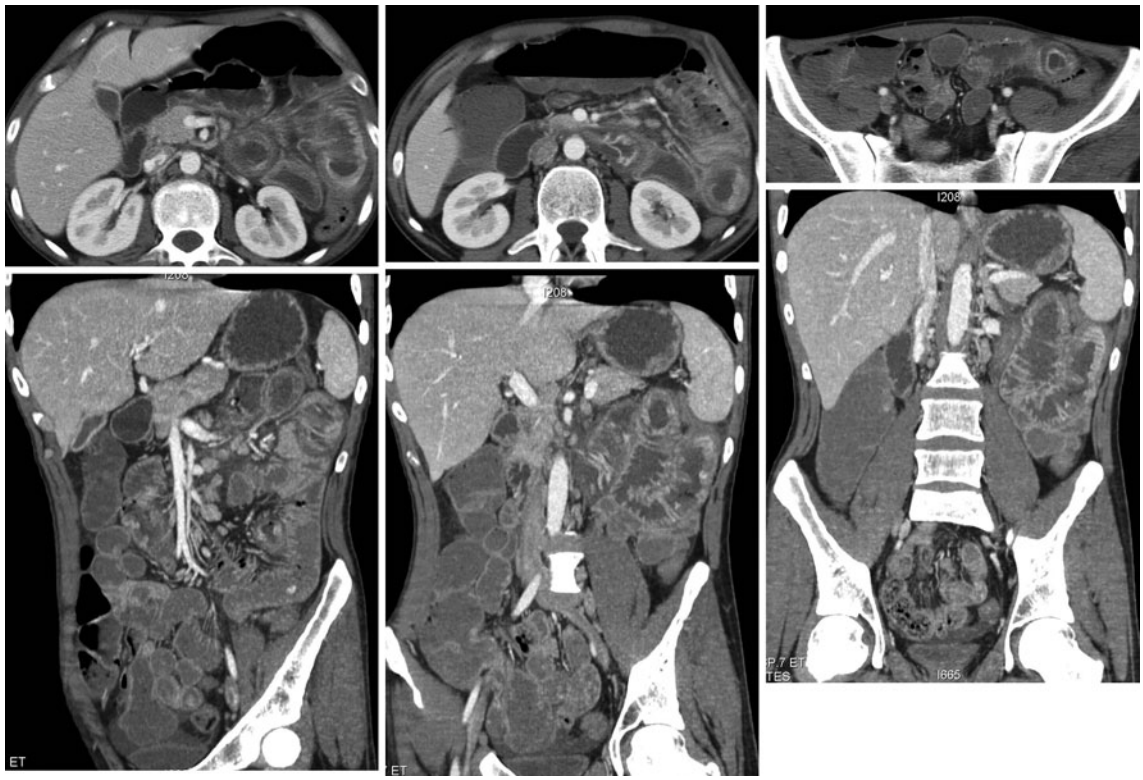

Fig. 29 Vasculitis in a heroine addict. The scan was performed for acute hyperalgesic pain which shows acute lesions predominantly on the proximal jejunum and characterised by considerable circumferential submucosal oedema with target sign enhancement, accompanied by small quantities of peritoneal fluid effusion

abscess, fistula, perforation or digestive haemorrhage. In all these circumstances, a CT scan is often performed quite quickly, and one must be able to identify the disease on the images produced, and its possible complications (Meyers et al. 1995; Lee et al. 2002).

It is obvious that, given an acute picture, even if questioning the patient or the clinical examination points towards Crohn's disease, the use of enteroclysis should be avoided and the examination performed usually without modifying the content of the loops or by performing enterography without a catheter, by ingestion of water or opacifying agent and in all cases taking care to minimise the dose of ionising radiation delivered to these patients, who are often young and who will be repeatedly exposed to CT scans as the condition develops.

Crohn's disease in the small intestine preferentially involves the terminal ileum, but, particularly in acutely revealed forms, it can affect almost the whole ileum or a shorter or longer segment of the jejunum and evidently the whole or part of the colon.

Positive CT scan diagnosis of Crohn's disease in the small intestine is based on the criteria for transmural inflammation seen as:

- more than 3 mm of parietal thickening;
- parietal stratification visible with double halo images in the active inflammatory forms;
- the presence above all, of the classic modifications of vascularisation on the mesenteric side of the loops affected: vascular jejunisation of the ileum (i.e. increase in the number of vasa recta per unit length, tortuosity and dilatation of these vessels), and a comb sign, which essentially reflect the shortening of the digestive segments involved and their mesentery which is, moreover, the site of fibrofatty infiltration (sclerolipomatosis);
- the presence of mesenteric adenopathy.

The mesenteric vascular abnormalities indicate an active, developed, widespread condition seen in an acute clinical context. There is close correlation with the clinical Crohn's disease activity index.

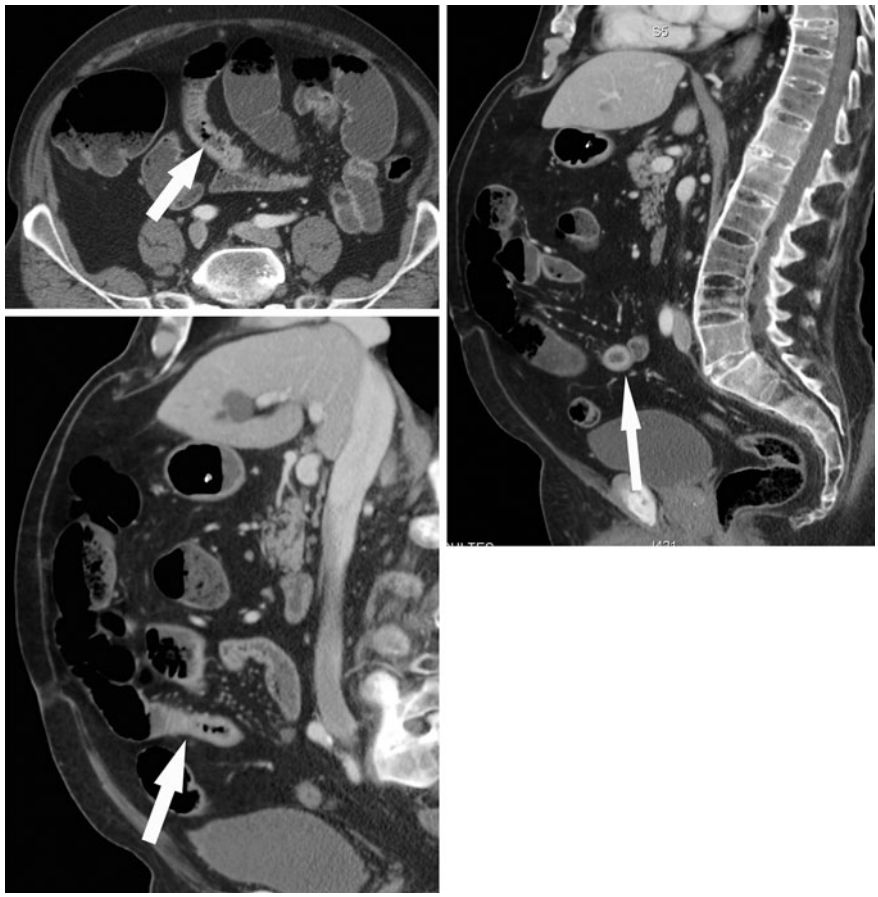

Fig. 30 Acute enteropathy due to non-steroidal anti-inflammatory drugs. Moderately stenosing parietal circumferential thickening of the jejunum, with considerable lasting enhancement (*white arrow*). Double balloon enteroscopy confirmed the diagnosis of enteritis due to NSAIDs

In contrast, fibrostenotic lesions appear in the scan as:

- regular uniform thickening, with no or little enhancement after injection of a contrast agent. However, it must be emphasised that the fibrosis can be enhanced, sometimes considerably, on delayed acquisitions. Delayed enhancement is therefore not a decisive predictive element for the efficacy of the medical treatment;
- stenosis of the lumen and upstream distension ~~it~~ with regular infundibuliform connection of the normal wall;
- ~~absence of mesenteric vascular modifications, in particular the comb sign.~~

Target or double halo enhancement images are also not themselves specific, even if this feature was initially described in Crohn's disease. Differential diagnosis should consider ischaemia, vasculitis, infectious enteritis, haematoma (Fig. 29), radiation enteritis and graft-versus-host reaction. Submucosal fatty metaplasia (or submucosal pseudolipomatosis) can lead to hesitation even more here than in the stomach. Its typical appearance of fatty density (the fat halo sign) and its existence in an overweight patient with excessive deep abdominal fat makes its easy to identify (Fig. 30).

An emergency CT scan, even if it lacks precision and does not allow as fine an analysis as CT enteroclysis (Kohli and Maglinte 2009), does permit Crohn's disease to be diagnosed and shows the activity of the disease (Minordi et al. 2009). Obviously, it highlights local complications: inflammatory or fibrous stenosis, abscesses, fistulae, perforations and any possible remote lesions such as urinary calculi, venous and more rarely arterial thrombosis, spondyloarthropathy, sclerosing cholangitis, etc. (Figs. 31, 32, and 33).

Meta-analyses have shown that there are no significant differences in diagnostic accuracy between CT and MRI (Horsthuis et al. 2008a, b). Given the necessity of limiting irradiation as much as possible,

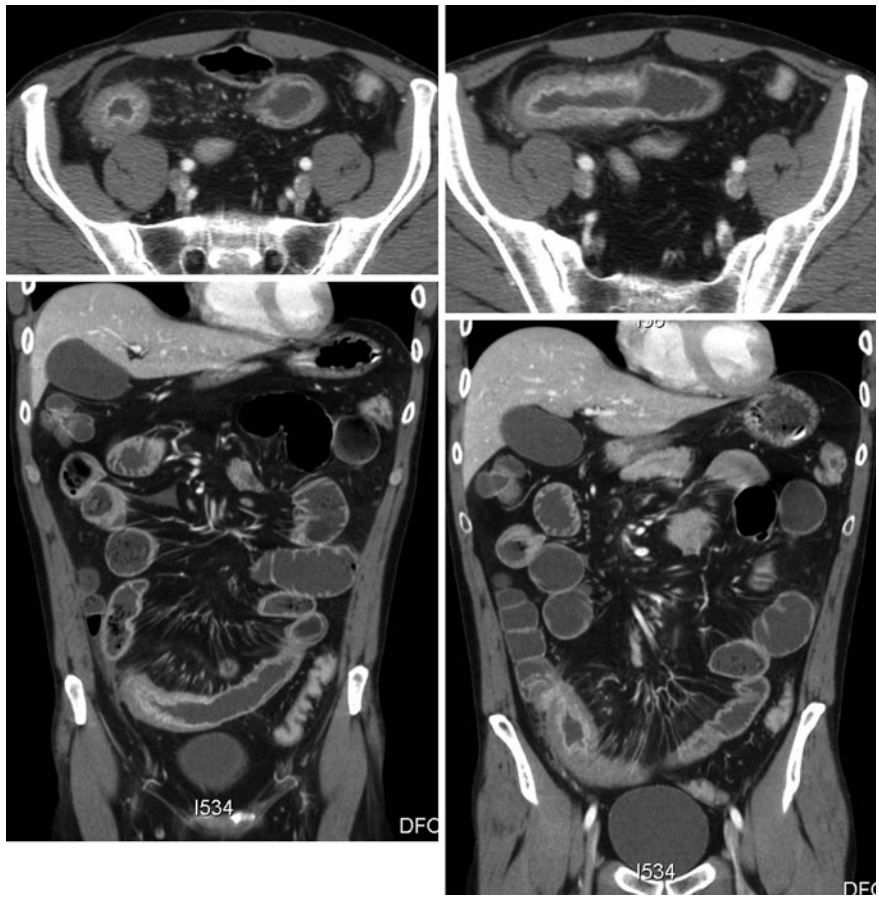

Fig. 31 Acute attack revealing Crohn's disease. Regular circumferential thickening of the final ileal loops with target sign image by enhancement of the mucosa and signs of transmural inflammation with comb sign appearance of the vasa recta

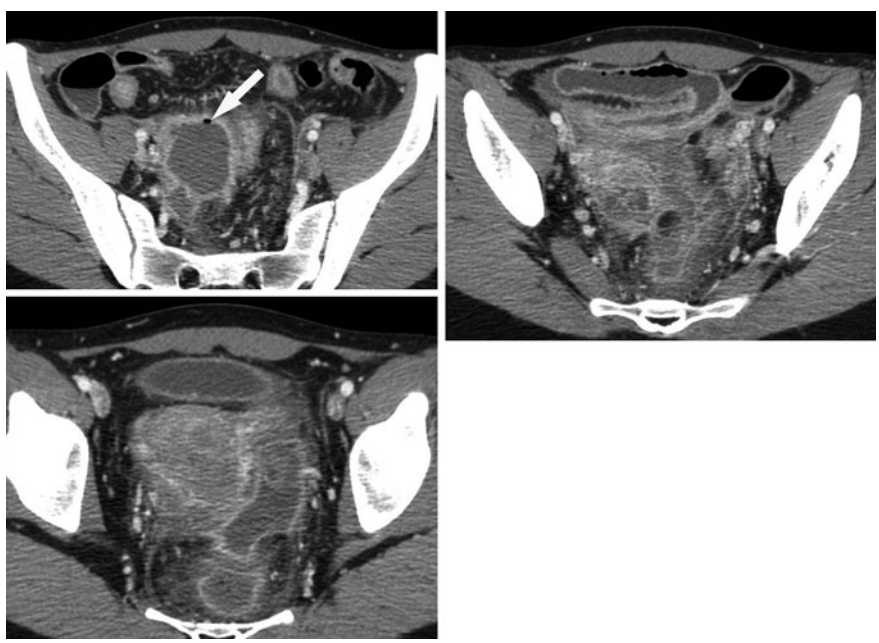

Fig. 32 Acute attack of Crohn's disease with pelvic peritonitis. Presence of images of acute inflammatory thickening of the ileal wall with signs of transmural inflammation. Multiple pelvic collections with one larger organised collection with thick wall and gas bubbles (*white arrow*)

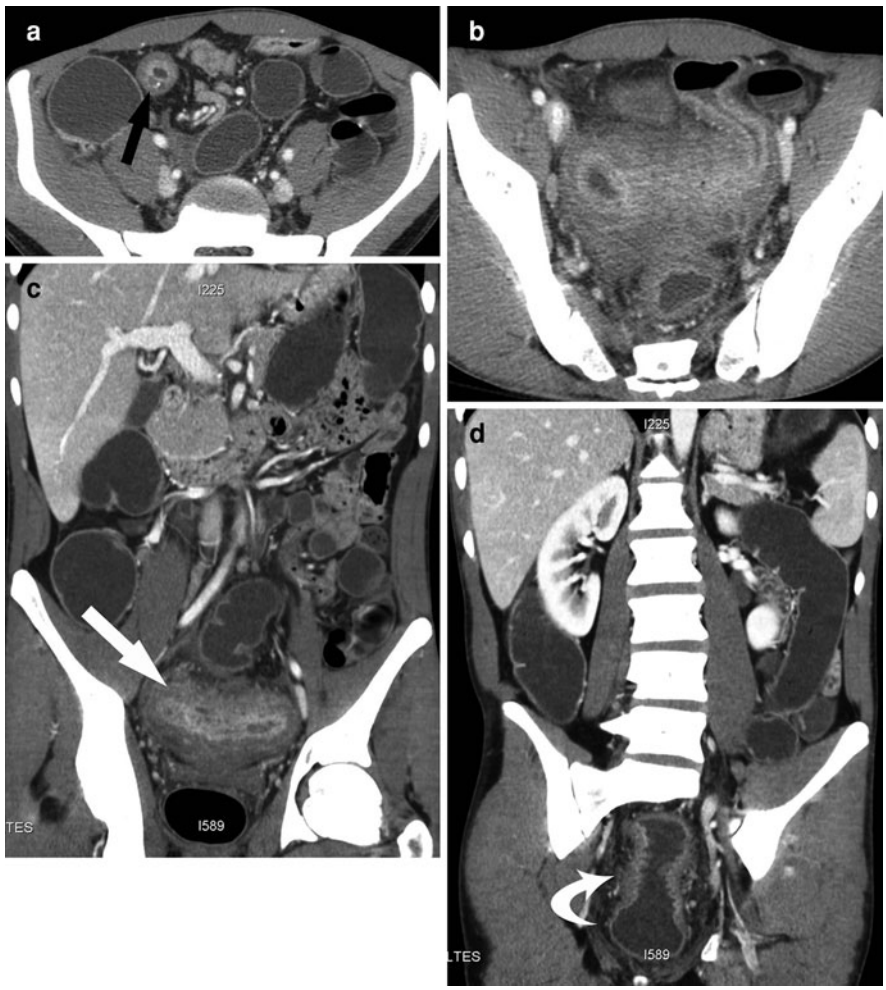

Fig. 33 Phlegmon complicating an ileal Crohn's disease extending to the rectosigmoid junction. The CT scan shows the typical images of ileal Crohn's disease (*black arrow*). In the pelvis there is massive inflammatory infiltration of the adjacent mesenteric structures (*straight white arrow*) (**a–c**) with continuous extension at the rectosigmoid junction (*white curved arrow*) (**d**)

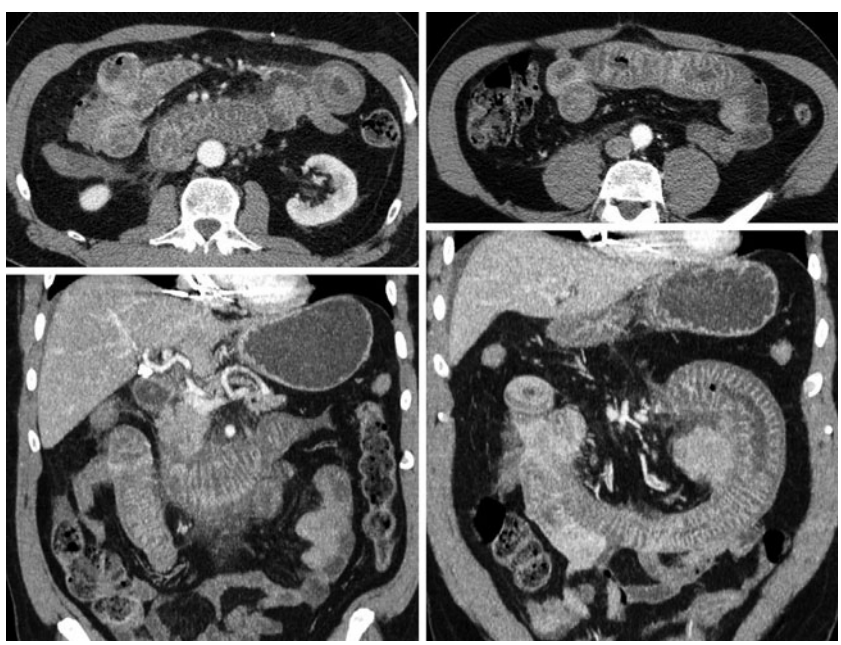

Fig. 34 Duodenal-jejunal parietal haematoma after ingestion of large dose of Aspegic®. Presence of considerable circumferential thickening extending to the wall of the proximal jejunal loops. The patient had ingested relatively large quantities of Aspegic® during the days prior to the acute incident which revealed this

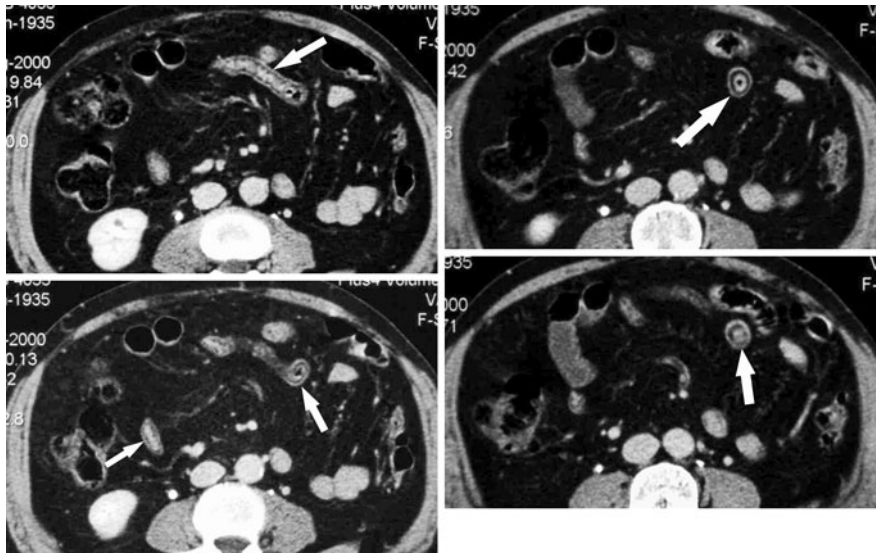

Fig. 35 Jejunal segmental submucosal metaplasia in an overweight patient. These images of parietal thickening with a fatty density seen in the context of painful abdominal symptoms led wrongly to the diagnosis of ischaemia of the small intestine and a misunderstanding of the real origin of the condition, which was biliary

particularly in young patients, it therefore seems best to direct these patients wherever possible to MRI, all the more so as the additional information from diffusion sequences allows very effective assessment of the degree of activity of the disease, correlated perfectly with clinical/laboratory activity indices. Injected sequences can thus be avoided and T2-weighted sequences used, at the equilibrium state ~~and in diffusion,~~ to effectively follow the spontaneous development of the disease and/or during treatment.

2.4.1 To summarize

The appearance in a CT scan of localised or diffuse hypodense parietal thickening of the stomach and/or small intestine, with visible stratification after injection, in the context of an acute abdomen, should mean that several hypotheses need to be considered for which exploration of the rest of the abdominal cavity may provide pros and cons of use for the aetiological diagnosis. It is usually, above all, the clinical history and carefully searching the patient's medical history which will guide this aetiological orientation. A radiologist is a clinician who must go well beyond the description of images if he wishes to provide the medical community with the services which the latter has the right to expect (Figs. 34 and 35).

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