REVIEW / Gastrointestinal imaging

MDCT of acute colitis in adults: An update in current imaging features

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KEYWORDS

Colitis; Acute colitis; Imaging; Computed tomography; Crohn disease

Abstract  Acute colitis is often diagnosed on multidetector row computed tomography (MDCT) because patients with this condition present with abdominal pain and a variety of nonspecific symptoms. Acute colitis has multiple causes with varying degrees of severity. Analysis of the extent of colonic involvement, presence of specific MDCT imaging features and associated signs should help radiologist narrow the diagnosis. Integrating the results of clinical examination and biological tests is mandatory, and in case of ambiguous or nonspecific MDCT findings, endoscopy and colon biopsy should always be considered for a definite diagnosis. The purpose of this review is to discuss and illustrate MDCT features that are helpful for characterizing acute colitis in adults and to provide an update in current MDCT features.

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Acute abdominal pain is one of the main causes of admission in emergency department [1] and among the various possible diagnoses, acute colitis represents a frequent cause that usually displays nonspecific clinical features. In recent years multidetector computed tomography (MDCT) has emerged as a primary screening modality for the investigation of acute abdominal syndrome. As a result, it is not rare that acute colitis be diagnosed at MDCT.

Acute colitis is characterized by an infiltration of the mucosa by inflammatory cells leading to hyperemia and edema of the submucosa. Acute colitis may involve the whole colon or be limited to colonic segments. Acute colitis has myriad causes with overlapping MDCT features, so that definite diagnosis relies on the result of endoscopy and, at a lesser degree on those of endoscopic biopsies of the colon. Consequently, the main role of the radiologist is to confirm the diagnosis, determine the extent and severity of acute colitis, and suggest a specific etiology [2].

The purpose of this review is to discuss and illustrate MDCT features that are helpful for characterizing acute colitis in adults and to provide an update in current MDCT features.

**MDCT technique**

Many patients presenting with clinical suspicion of acute colitis have nonspecific abdominal symptoms. Therefore, MDCT examination of the abdomen and pelvis should be performed with a protocol that suits for the majority of the possible diagnoses, although in a few cases the MDCT technique can be specifically tailored to the most likely diagnosis.

At our institution, when MDCT is performed in patients presenting with nonspecific abdominal symptoms in the absence of recent history of abdominal surgery, a water enema is preferably used [3]. Water enema distends the rectum and colon and adds confidence in the assessment of bowel wall and mural thickening. In addition, contrary to positive contrast agent, water enema permits to appreciate mural enhancement and does not interfere with subsequent three-dimensional reconstructions [4–6].

Although the disease can be correctly assessed without the need for intravenous administration of iodinated contrast material in a number of cases, intravenous administration of iodinated contrast material is preferable because the patterns of enhancement may provide suggestive clues for a specific diagnosis, assess severity of the disease and help depict complications, if any. We routinely administer in-patient without renal disorder 1.5 mL of contrast material per kg of body weight. Non-ionic iodinated contrast material at a concentration of 30 g of iodine per 100 mL is administered at a rate of 3 mL/s through an 18-gauge venous catheter. Scanning starts 50 seconds after the beginning of contrast material injection. This delay corresponds to the mesenteric phase of abdominal imaging [7] and allows detection of subtle colon wall abnormalities. This phase also provides a comprehensive evaluation of the abdomen and pelvis, including abdominal vessels.

Patients are positioned head first in supine position. Scanning is performed from the dome of the liver to the symphysis pubis. A collimation thickness <1-mm permits submillimeter and isotropic voxel reconstruction. An attenuation-based tube current modulation technique is used to decrease the radiation dose given to the patient [8].

A first set of images is obtained in the axial plane using a soft tissue reconstruction kernel, with a thickness of 2- to 3-mm at 2- to 3-mm intervals for analysis of axial images. A second set is obtained at 0.6-mm thickness at 0.5-mm intervals for multiplanar reconstructions as well as for maximum intensity projection (MIP) and three-dimensional (3D) views. Multiplanar reconstructions, MIP and 3D views are interpreted along with axial images to better understand involvement of colonic segments, improve localization of colonic abnormalities, and analyze the colonic vascularization.

**MDCT findings and definitions**

Acute colitis is mainly characterized by a symmetric inflammatory colon wall thickening. Of note, colonic wall is better assessed when well dilated, avoiding confusion with pseudothickening due to incomplete or poor distension or collapsed bowel wall. When correctly distended, the normal colon is characterized by a thin wall, with a thickness <3-mm [9]. Wall thickening is considered when the colon wall thickness is >5-mm [9,10]. When the colon wall thickness ranges between 3- and 5-mm, categorization is difficult. Mural thickening have been reported from 3-mm to 3 cm. Normal colonic wall usually shows homogeneous enhancement and an axial diameter of 3–5 cm and up to 6–8 cm for the cecum. Attention should be given to differentiate the usually symmetric and segmental mural thickening of acute colitis from the asymmetric and rather focal wall thickening of colorectal adenocarcinomas or those found in carcinomatosis or endometriosis [11–14].

MDCT is very sensitive at showing inflammatory changes but has lower degrees of specificity [10]. Three different patterns of enhancement of a thickened colon wall have been described. They include homogeneous circumferential thickening, halo sign and target enhancement [15,16]. The target sign corresponds to stratification with three layers due to enhancement of mucosa and serosa while the submucosa in the middle remains hypotennuating because of edema [15]. The halo sign corresponds to stratification with two layers and consists in a marked enhancement of the mucosa and a slightly lower enhancement of submucosa and serosa related to hemorrhage or inflammation [16]. However, an impaired venous drainage due to portal hypertension or right heart failure may be responsible for halo or target sign [16].

Fatty infiltration and dilatation of mesenteric vessels are common findings in acute colitis that reflect an acute inflammatory process and can be due to hyperemia and serous fluid leakage. Similarly, ascites is a consequence of colonic wall suffering and results from leakage of edema from the colon wall to the extracellular space [17].

Mesenteric lymph node enlargement (i.e., >5 mm in short axial diameter) is similarly a common imaging finding of poor specificity. However, irregular edges or heterogeneous enhancement suggest an underlying malignant process [18].

The accordion sign, which is due to marked thickening of hastral folds, consists in broad transverse bands that may
trap orally given positive contrast material [15]. However, it has also been described in the absence of oral contrast material because of an increased mucosal enhancement after intravenous administration of iodinated contrast material [19]. The accordion sign is very suggestive for the diagnosis of pseudomembranous colitis but has also been reported in infectious colitis so that it is does not carry high degrees of specificity [19].

MDCT findings that indicate severe acute colitis include toxic megacolon, pneumatositis and perforation. Toxic megacolon consists in markedly distended colon filled of air and/or fluid, with a distorted luminal contour or an ahaustral pattern [20]. The colon wall remains thin with an ill-defined and nodular inner margin [20]. Pneumatosis coli presents as air bubbles in the colon wall arranged in a linear fashion and are best visualized with the window settings for bone or lung [20]. Bubbles may also be present in the mesenteric or portal veins and when associated with mural thickening, are highly suggestive of transmural infarction. In addition, presence of any extra colonic gas (after exclusion of special conditions of physiological pneumatoneum) should be considered as colonic perforation and lead to specific surgical management [21]. Finally, the association between acute colitis and pylephlebitis, which is a septic thrombosis of the superior mesenteric vein, has also been reported [22].

Etiologies of colitis and classification

Acute colitis refers to histopathological condition related to acute inflammatory process of the colonic wall. Although various diseases may be responsible of this inflammatory process, they can be divided into infectious and noninfectious causes. Infectious may be further divided between tuberculous and nontuberculous colitis and noninfectious acute colitis into inflammatory and noninflammatory colitis (Tables 1 and 2).

Tuberculous colitis

Clinical considerations

Tuberculous colitis is a rare condition in western countries, and its diagnosis may be difficult because it may mimic many other diseases. Colonic tuberculosis is usually acquired by ingesting contaminated milk products or, in a patient with pulmonary tuberculosis, by swallowing tracheobronchial secretions [20]. Optical colonoscopic features in colonic tuberculosis include mucosal nodules and ulcers, which may be occasionally associated with strictures, pseudopolyoid folds, or fibrous bands [23]. The diagnosis is usually established by positive cultures for acid-fast bacillus and acid-fast bacilli staining of tissue samples obtained during colonoscopy or sometimes laparoscopy in association with the presence of caseating granulomas. However, caseating granulomas, which are the hallmark of tuberculosis, are found in only one third of the cases. Location of the disease is ubiquitous, but the ileocecal valve is often involved. The lesions tend to be transmural with marked desmoplastic reaction and a large amount of fibrous tissue. The lesions characteristically produce deep transverse ulceration of an irregular or “geographic” contour. Inflammation may produce a masslike lesion called tuberculosis, which may mimic colon cancer [24]. Occasionally, enteroenteric fistula or mixed tuberculous and bacterial abscesses are encountered. MDCT can be helpful for the diagnosis and consequently, knowledge of specific MDCT findings may avoid unnecessary invasive procedure.

MDCT presentation

The ileocecal area is the portion of the gastrointestinal tract that is most frequently involved by tuberculosis. Characteristic MDCT features include asymmetric thickening of the ileocecal valve and medial wall of the caecum, hypertrophic folds, pericolic fat stranding, exophytic extension engulfing the terminal ileum, and large lymph nodes with central, hypoattenuating portions (Fig. 1) [25]. Lymph nodes are markedly enlarged, often hypoattenuating with a peripheral rim of enhancement and may also contain calcifications. Fistulas and sinus tracts can be seen but are less common than in Crohn’s disease. Segmental colitis, diffuse colitis, and short strictures that mimic tumor may be seen [20]. A cone-shaped cecum caused by scarring, as well as hypertrophy of the ileocecal valve (i.e., the Fleischner sign) can be seen on rare occasions [20]. At an advanced stage, MDCT may show peritoneal thickening, ascites and a retracted cecum together with smooth symmetrical stenosis of the transverse colon [26].

Crohn disease is the main differential diagnosis of tuberculous colitis. Differentiation between these two entities is crucial because of differences in treatment strategies. Indeed, treatment of Crohn disease requires high dose of corticoids that may dramatically worsen tuberculosis. Distinction between these two entities can be problematic without laparotomy [23]. However, some helpful clues may suggest one diagnosis rather than the other. Thickening of the colonic wall may be more prominent in tuberculous colitis than in Crohn disease. In tuberculous colitis, lymph nodes, when present, are large, necrotic and adjacent to the colon whereas in Crohn disease they often show diffuse and homogeneous enhancement. Pericolic fibrofatty proliferation and a long involvement of the distal ileum are more suggestive for Crohn disease. In tuberculous colitis mural stratification is absent whereas it is common finding in active Crohn disease [23,27]. Occasionally, a short thick-walled stricture due to tuberculosis may be seen on MDCT and can be mistaken for colonic adenocarcinoma [24]. In case of tuberculous involvement, the cecum is usually retracted and the ileocecal valve is rigid and incompetent.

Infectious colitis

General considerations

Generally, the diagnosis of infectious colitis is based on clinical symptoms and does not require MDCT. Patients with infectious colitis usually present with an acute onset of dysenteric symptoms, consisting of fever, crampy abdominal pain, abdominal tenderness, tenesmus and diarrhea with bleeding. However, infectious colitis may be detected incidentally at MDCT or in patients for whom the clinical symptoms are equivocal.
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MDCT: multidetector row computed tomography; IBD: inflammatory bowel diseases; CMV: cytomegalovirus.
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MDCT: multidetector row computed tomography; IBD: inflammatory bowel diseases; p-ANCA: perinuclear anti-neutrophil cytoplasmic antibodies; ASCA: anti-saccharomyces cerevisiae antibodies.
Diagnosis of infectious colitis may be difficult with MDCT because the findings are not specific. Bacterial causes include *Shigella*, *Salmonella*, *Yersinia*, *Campylobacter*, *Staphylococcus*, and *Chlamydia trachomatis*. Viral causes include cytomegalovirus and rotavirus [6]. In immunodeficient patients, cytomegalovirus causes more frequently colitis than ileitis. The definite diagnosis of infectious acute colitis is difficult with MDCT alone and is confirmed on the basis of the results of stool analysis and/or those of optical colonoscopy.

**MDCT presentation**

In patients with infectious colitis, MDCT uniformly shows colon wall thickening in association with target sign. Pericolonic fat stranding, free fluid effusion or lymphadenopathy may also be observed [10,29]. Multiple air-fluid levels may be seen in the colon due to increased fluid production and accumulation as well as spontaneous colonic distension. Pancolitis at MDCT is due to infection in 50% of the cases [10]. Depending on the portion of abnormal colon, a specific cause may be suggested. In case of infection due to *Yersinia*, *Salmonella*, or *C. Jejuni* abnormalities are limited to the ascending colon with an associated ileitis in a number of cases (Fig. 2). Conversely, a diffuse involvement is mostly due to cytomegalovirus and *Escherichia Coli* [37]. *N. gonorrhoea*, Herpes virus, and *C. trachomatis* typically involve the rectosigmoid. In acute colitis due to Schistosomiasis and *Shigella*, inflammation is confined to the descending colon [39]. Colitis due to cytomegalovirus usually mimics ulcerative colitis with diffuse mucosal ulceration or Crohn colitis with aphthous ulcerations and skip areas [38]. *Pneumatosis intestinalis* can be observed in severe infectious colitis due to *E. Coli* [78,79].

**Pseudomembranous colitis**

**Clinical considerations**

Pseudomembranous colitis is a complication of broad-spectrum antibiotic therapy and is commonly observed in neutropenic patients. Alteration of normal gastrointestinal...
flora by antibiotics allows colonization by *C. difficile*, which is a strictly anaerobic organism that produces enterotoxins A and B, resulting in inflammation of the colon, diarrhea and pseudomembranous exudates [31–33]. Over the last decade, the incidence of *C. difficile* associated disease has progressively increased and is now a frequent clinical problem in North America and Europe [30].

The diagnosis of pseudomembranous colitis is usually based on clinical history that includes a recent treatment with antibiotics and confirmed by the results of stool assay for the *C. difficile* toxin. However, the clinical presentation can be misleading and stool assays for *C. difficile* toxin have a substantial rate of false-negatives so that optical sigmoidoscopy is required for a rapid identification of *C. difficile*-associated pseudomembranes, which are made of necrotic mucosal cells. Oral administration of metronidazole is the first-line therapy whereas the use of oral vancomycin is restricted to patients who have experienced a relapse of the disease after a course of metronidazole. Probiotic therapies based on administration of Saccharomyces *boulardii* represent another treatment option [80]. However, some patients, if not treated appropriately or presenting with a fulminant form, may experience toxic megacolon and perforation, thus resulting in a fatal outcome [81,82]. Those patients may thus require emergency colectomy for cure [33, 83, 84]. As a consequence, radiologists should be familiar with the MDCT findings of pseudomembranous colitis. As a limitation, however, MDCT findings alone do not help predict which patients with pseudomembranous colitis will require surgery [85].

**MDCT presentation**

Marked circumferential or eccentric thickening of the colonic wall is the most common CT finding in pseudomembranous colitis with a mean value between 10.7 mm and 14.7 mm [range: 3 to 32 mm] [15, 25, 29]. The degree of thickening in pseudomembranous colitis is greater than that observed in any other inflammatory or infectious disease of the colon so that thickening is a helpful differentiating feature [34, 39]. On MDCT, wall thickening in pseudomembranous colitis is often more irregular and shaggy than in Crohn disease [15]. In patients with pseudomembranous colitis, the target and halo sign may be present. In addition, the colon is often dilated secondary to transmural inflammation. The most suggestive feature of pseudomembranous colitis is the accordion sign (Fig. 3) [15]. This sign has a sensitivity comprised between 38% and 73% and a specificity of 61% for the diagnosis of *C. difficile* colitis [19, 28]. Approximately three-fourths of pseudomembranous colitis present as pancolitis [28] but it one fourth of the cases, the disease starts as a proctitis and progresses towards the descending colon. It can also be limited to the ascending colon in up to 30%–40% of cases [35]. Limited involvement to portions of the colon and rectum has also been reported [15]. Ascites can be observed in approximately one third of patients with pseudomembranous colitis [35, 36].

Although, pericolic fat stranding can be observed in many other inflammatory and infectious diseases of the colon, in pseudomembranous colitis it is mild and contrasts with the relatively marked colonic wall thickening since the condition predominantly affects the mucosa and submucosa [34].

![Figure 3](Image 358x620 to 586x785)

**Figure 3.** Forty-two-year-old man with pseudomembranous colitis. Multidetector row computed tomography image in the axial plane shows thickened, hypoattenuating submucosa of the transverse colon (arrows). Hyperattenuating mucosa is visible (arrowheads). The combination of hypoattenuating submucosa with hyperattenuating mucosa results in the accordion sign.

The target sign, which has been originally described in Crohn disease and ulcerative colitis, can be associated with pseudomembranous colitis. The accordion sign can be found in cryptosporidiosis, ischemic colitis, lupus vasculitis, ulcerative colitis, Crohn disease, and infectious colitis due to *Salmonella* or cytomegalovirus [19]. Ascites may be a helpful feature for differentiating between pseudomembranous colitis and Crohn disease. However, ascites can also be present in ischemic and infectious colitis.

**Graft vs. host disease**

**Clinical considerations**

Graft vs. host disease is a severe complication of allogeneic bone marrow transplantation [76]. This condition occurs when the donor T-lymphocytes attack the transplant recipient’s body. The gastrointestinal tract (predominantly the ileum and colon), skin, and liver are the most frequently affected organs. Graft vs. host disease usually happens during the first 3 months after transplantation and affects approximately 15 to 50% of patients treated with allogeneic bone marrow transplantation [76]. Clinically, this condition manifests as fever, diarrhea, vomiting and sometimes gastrointestinal bleeding. Pathologically, graft vs. host disease is characterized by a diffuse destruction and replacement of glandular structures by macrophages, lymphocytes and plasmocytes. Glandular crypts of the colonic mucosa are involved by extensive lesions of necrosis [86].

**MDCT presentation**

MDCT findings include colonic wall thickening that may result in luminal narrowing. The terminal ileum can also demonstrate parietal thickening [76]. Prolonged coating of colon with contrast material has been reported in patients with severe mucosal disease [76]. In these cases, the diluted barium can become trapped into the bowel wall because of mucosal healing due to superficial ulcers [77]. This intramural dissection of barium at MDCT is not pathognomonic for
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graft vs. host disease and has been also described in other conditions that cause severe mucosal ulceration, such as ischemic colitis. Other findings have been reported such as a halo and target sign [76], pericolic stranding with accompanying lymph nodes resembling mesenteric necrotic lymphadenitis [44] and in some cases, distension of the colon [44].

Colonic involvement in graft vs. host disease is similar to that observed in radiation-induced colitis except for the length of involvement, which is usually less extensive in radiation-induced colitis. Although the colon wall can be thickened, thickening is less marked than in typhlitis and pseudomembranous colitis. In this regard, a colon wall thickness >7 mm virtually excludes graft vs. host disease [40]. However, in ambiguous cases, clinical history helps reach the specific diagnosis.

Typhlitis

Clinical considerations

Typhlitis, which is also called neutropenic enterocolitis, occurs almost exclusively in neutropenic patients under treatment for hemopathy [45]. Indeed, typhlitis has been reported after bone marrow transplantation [44] and in patients with aplastic anemia, lymphoma, or acquired immunodeficiency syndrome [37]. Patients with this condition present with fever, watery or bloody diarrhea, gastrointestinal bleeding and abdominal pain in the right lower quadrant.

Histopathologically, typhlitis is characterized by marked edema and inflammation of the cecum, the ascending colon, and rarely the terminal ileum. The inflammation can lead to transmural necrosis and perforation. In addition, some patients may experience severe or even life-threatening hemorrhage [87]. Pathophysiology of this condition has not yet been fully established, but it is hypothesized that it is the result of a combination of neutropenia, ischemia, opportunistic infection (cytomegalovirus or adenovirus), mucosal hemorrhage, and at some degrees neoplastic infiltration [37]. Neutropenia is considered as the main factor because it generates an overgrowth of colonic germs. Treatment consists of bowel rest, total parenteral nutrition, broad-spectrum antibiotics, and massive fluid and electrolyte replacement. Surgery can be needed in patients with transmural necrosis, perforation, or uncontrolled sepsis. At surgery, all necrotic colonic tissues must be resected [88–90].

MDCT presentation

MDCT is the best imaging modality in patients with typhlitis because of the risk of bowel perforation and severe bleeding due to videocolonoscopy. MDCT demonstrates cecal distention with marked circumferential thickening, which may be associated with a target sign [41]. The terminal ileum can be involved and free fluid effusion can be present [45,91]. Pneumatosis of the cecal wall is a suggestive sign although it is rarely visible and can be seen in other conditions such as cecal ischemia [40]. Although typhlitis has often been described as a disease of the ascending colon (Fig. 4), it is to be noted that other portions of the colon can be involved [40,78]. Pericolic inflammatory stranding is uncommon. Detection of pneumatoasis, pneumoperitoneum, and pericolic fluid collections is important because they require urgent surgical management [42,43,91,92]. MDCT is also helpful in assessing favorable response to treatment [37]. Of interest, there have been cases of patients with neutropenic colitis and colonic pneumatosii who recovered after conservative therapy only [40].

Distinction of typhlitis from other causes of colitis such as Crohn disease or other infectious diseases may be difficult but the clinical history helps to make a correct diagnosis [46,47]. Clues to the diagnosis include presence of marked thickening, which is localized to the ascending colon in association with severe neutropenia.

Ischemic colitis

Clinical considerations

Ischemic colitis represents 1/1000 hospitalizations [93] but its incidence is underestimated because of its mild or transient nature [63]. Clinical presentation ranges from transient limited ischemia involving the mucosa and submucosa to transmural infarction responsible of bowel necrosis and death [61]. Clinical symptoms include crampy abdominal pain, diarrhea and mild rectal bleeding. In cases of severe ischemia, peritoneal inflammation may be present in association with metabolic acidosis and septic shock [63].

Figure 4. Thirty-seven-year-old man with typhlitis. a. Multidetector row computed tomography (MDCT) image in the axial plane shows marked, irregular, circumferential thickening of the right colon (arrows) and pericolic fat stranding (arrowhead). b. MDCT image in the coronal plane shows marked thickening of the right colon (arrows). The right colon and the sigmoid (not shown) are not involved by the disease.
The most common cause of ischemic colitis is an acute, self-limited compromise in intestinal blood [61]. The left colon is involved most of the time in elderly patients. Although MDCT may have suggestive findings, videocolonoscopy is the diagnostic modality of choice and is safe when performed carefully.

**MDCT presentation**

MDCT shows suggestive findings in up to 89% of patients with ischemic colitis [94]. Two main patterns of ischemic colitis have been described, including a wet and a dry form [62]. In reversible non-transmural ischemic colitis, MDCT demonstrates homogeneously enhancing colon wall thickening, thumbprinting, and pericolonic stranding with or without peritoneal fluid (Fig. 5). After reperfusion (also called the "wet" form), MDCT usually demonstrates a halo or target sign. In case of infarction, that is a total vascular occlusion without reperfusion, the colonic wall remains thin and unenhancing, associated with luminal dilatation. This condition is also called the "dry" form. In these cases, MDCT may demonstrate a thrombus in the corresponding mesenteric vessel [43]. If ischemia is transmural, strictures may form and occasionally, a toxic megacolon develops.

Pneumatosis may be associated with gas in the mesenteric or portal vein, and thus suggests advanced stage bowel infarction (Fig. 6) [20]. In contrast to mesenteric ischemia, a focal defect on MDCT angiography is unusual and when present often mismatches with the segment of the colon involved.

Ischemic colitis usually appears in elderly patients with polyvascular disease and demonstrates a vascular distribution pattern just proximal to a normal colon and rectum. MDCT presentation is therefore nonspecific and even *P. coli* can be a benign condition and may also be associated with any severe colitis [78]. In addition, early MDCT findings cannot be considered as prognostic indicators for the presence or later development of infarction [94]. Thus, MDCT findings should routinely be confirmed by colonoscopy. Patient history, clinical presentation and the results of laboratory tests are mandatory to confirm the diagnosis.

**Phlebosclerotic colitis**

**Clinical considerations**

Phlebosclerotic colitis is a rare form of ischemic colitis that usually affects the right colon [64]. Patients with

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**Figure 5.** Fifty-year-old woman with reversible, non-transmural ischemic colitis. a. Multidetector row computed tomography (MDCT) image in the axial plane shows circumferential, homogeneous thickening of left colon (arrow). No abnormalities of the left colon (arrowhead) are visible. b. MDCT in the coronal plane shows circumferential thickening of left colon (arrows). There is no visible enhancement of the submucosa. The mucosa has an irregular surface displaying a thumbprinting pattern (black arrowheads). c. MDCT in the coronal plane using a maximum intensity projection (MIP) algorithm shows paucity of distal branches of left colic artery.

**Figure 6.** Sixty-two-year-old male with irreversible, transmural ischemic colitis. a. Multidetector row computed tomography (MDCT) image in the axial plane shows linear pneumatosis of right and left colon wall (arrowheads) and gas within mesenteric venous branches (arrow). b. MDCT in the coronal plane confirms the linear form of parietal pneumatosis (arrowheads).
phlebosclerotic colitis usually present with recurrent right abdominal pain, nausea, vomiting and chronic diarrhea with melena. Ischemia is due to phlebosclerosis that impairs venous drainage of the colon [65]. Venous obstruction is caused by fibrotic sclerosis and calcification of the wall of the mesenteric veins. The clinical course is different from that of the more common type of ischemic colitis caused by arterial obstruction [65]. The etiology of phlebosclerotic colitis is unknown, however, coagulation disorder, hemodialysis and cirrhosis are considered as favouring conditions [95–97].

**MDCT presentation**

The characteristic imaging features of phlebosclerotic colitis consist of dilated venous collaterals with calcifications and/or obstructions of the veins of the colonic wall and adjacent mesentery associated with a marked edematous thickening of the colonic mucosa and an increased attenuation of adjacent fat [68,69].

Ischemic colitis is the main differential diagnosis of phlebosclerotic colitis because of segmental involvement of the colon. Homogeneous marked thickening of the colonic wall is a nonspecific finding, and may be associated with numerous types of colitis or intra-abdominal infections with pyelophlebitis that may mimic phlebosclerotic colitis [66,67]. Depiction of calcifications on mesenteric veins originating from the affected colon segment is a major clue to help suggest diagnosis.

**Inflammatory colitis**

**Clinical considerations**

Patients with Crohn disease and ulcerative colitis usually present with similar clinical symptoms, including nonspecific abdominal pain, cramping, tenesmus or hematochezia [29]. In case of rectal involvement, patients have frequent but small-volume stools with a sensation of incomplete evacuation. Patients with Crohn disease frequently complain from acute pain in the ileocecal area and fever that clinically mimic appendicitis [29]. Biologically peri-nuclear anti-neutrophil cytoplasmic antibodies, (p-ANCA) are present in patients with ulcerative colitis but this feature is not sensitive enough for the diagnosis of ulcerative colitis. Anti-saccharomyces cerevisiae antibodies (ASCA) are more frequently observed in Crohn disease. Although clinical history and endoscopic findings help discriminate between Crohn disease and ulcerative colitis, MDCT may help differentiate between these two entities when clinical, endoscopic and histopathological findings are equivocal. In addition, MDCT plays an important role for the detection of a variety of complications of inflammatory bowel diseases. Accurate identification of complications is important because they have impact on patient’s management and outcome because undetected complications may have severe consequences.

**MDCT presentation**

Considerable overlap exists between the MDCT findings of Crohn disease and those of ulcerative colitis. However, some features may help distinguish between the two conditions. Colonic involvement by Crohn disease affects the ascending colon and terminal ileum. Severe involvement with marked inflammation results in stenosis of the ileocecal valve and upstream dilatation of the ileum. Diffuse involvement of the colon may occur in Crohn disease but involvement of the descending colon as the single location is rare [6]. By contrast, ulcerative colitis is typically left sided or diffused, and rarely involves exclusively the ascending colon [29]. In addition, in ulcerative colitis, the rectum is virtually always abnormal whereas, in Crohn disease, the rectum may be spared although other colonic segments are involved [6]. Luminal narrowing along with mural thickening of the rectum are often associated with perirectal fatty proliferation with enlargement of the presacral space. This latter association of findings is very suggestive for the diagnosis of ulcerative colitis (Fig. 7) [29].

Wall thickening is the most frequent MDCT finding in Crohn disease and ulcerative colitis [16,54,55]. The mean wall thickness in Crohn disease (11 mm) is usually greater than that in ulcerative colitis (7.8 mm) [29,98]. Wall thickening in ulcerative colitis is typically diffuse, symmetric and continuous, whereas in Crohn disease it is typically eccentric, asymmetric and segmental with apparently disease-free areas (the so-called ‘’skip regions’’). The asymmetric pattern, which typically occurs along the mesenteric border of the intestine, can result in the formation of pseudodiverticula along the antimesenteric border.

In patients with idiopathic inflammatory bowel diseases, thickening of the colonic wall is found in association with mural stratification. The halo and target signs most often indicate an acute stage of the disease but are not specific. The water halo sign indicates acute stage and can be observed in both Crohn’s disease and ulcerative colitis. The fatty halo sign indicates chronic disease and is more frequent in ulcerative colitis [99]. By contrast, homogeneous enhancement (i.e., *en bloc*) is consistent with an inactive or a chronic fibrous disease (Fig. 8) [48]. Other features must be searched for to heighten confidence in diagnosing acute
disease. In this regard, enlarged or engorged pericolic vessels strongly suggest active disease [49].

Proliferation of mesenteric fat is very suggestive for Crohn disease [57,58]. By contrast, proliferation of perirectal fat is less specific, and can be observed in both Crohn disease and ulcerative colitis [29]. Mesenteric and pericolic lymphadenopathy suggests Crohn disease rather than ulcerative colitis (Fig. 9), although this finding is certainly not specific for inflammatory bowel disease.

A main role of MDCT in inflammatory bowel diseases is to detect severe complications that require urgent therapy [18,98,100]. Of these, toxic megacolon is a severe complication of ulcerative colitis, which must be ruled out [101]. When present, pneumatosis intestinalis suggests an extremely severe form of the disease. Prompt therapy is mandatory to avoid severe or even life-threatening complications such as colon perforation and peritonitis [102,103]. Other complications can be observed in patients with Crohn disease. They include pericolic phlegmon and abscess [104,105]. MDCT allows differentiating phlegmon, which is an ill-defined inflammatory mass that usually requires antibiotics only (Fig. 10), from abscesses, which is a well-defined collection with peripheral rim that often requires a more aggressive treatment such as percutaneous drainage or surgery [106,107]. Abscesses are present in 15% to 20% of symptomatic patients with Crohn disease [59]. Abscesses are detected almost exclusively in Crohn disease and not in ulcerative colitis [29,56]. They usually result from sinus tract, fistula or perforation. They are often confined to the bowel wall but they may extend to

Figure 8. Twenty-two-year-old woman with Crohn disease. a. Multidetector row computed tomography (MDCT) image in the axial plane shows thickening of sigmoid wall with poor, en bloc enhancement indicating chronic disease. Moderate stenosis (arrow) of the sigmoid colon is visible. Fatty proliferation adjacent to sigmoid colon is visible (arrowheads). Vessel engorgement is visible (curved arrows) displaying a feature similar to that observed in the so-called ‘‘comb sign’’. b. At a different level of slice, MDCT in the axial plane shows mild distension of right and left colon (arrowheads). c. MDCT in the oblique plane confirms luminal stenosis of sigmoid colon (arrow) and upstream dilatation (arrowhead). d. Photograph shows gross specimen after surgical resection and confirms stenosis of sigmoid colon and adjacent fatty proliferation.
adjacent structures such as the bladder, psoas muscle, and pelvic wall. MDCT can also be used for image guidance when percutaneous drainage of abscess is required [107].

Fistulas are frequent complications of Crohn disease. They most often originate from the rectum rather than the colon. Perianal and rectovaginal fistulas can be detected with MDCT although MR imaging is more effective for their depiction [60].

Because of a possible association between intra-abdominal venous thrombosis and ulcerative colitis, MDCT, which provides a comprehensive evaluation of intra-abdominal veins, including portal and hepatic veins, is of value. MDCT helps detect unsuspected thrombi in patients in ulcerative colitis (Fig. 11) [52,53].

Proliferation of perirectal fat can be observed in Crohn disease, ulcerative colitis, pseudomembranous colitis and radiation colitis [29]. The fatty halo sign can be observed in patients who had received external radiation therapy [50,51]. Intramural fat can be also present in patients who are free of colonic disease but in such cases, the fatty layer is thinner by comparison with the thickness observed in patients with inflammatory colon disease [99]. Toxic megacolon can be observed in infectious colitis. Fistulas can be due to infectious diseases such as tuberculosis or actinomycosis.

**Radiation-induced colitis**

**Clinical considerations**

External radiation therapy can result in severe injury of the colon and rectum [70]. Acute radiation-induced proctitis occurs during or within 2–4 weeks after the treatment [70–72]. Approximately 75% of patients who receive external radiation therapy for pelvic or genitourinary tract tumors and all patients who received 60 Gy or more will experience acute proctitis symptoms [108]. Acute proctitis manifests as pain, self-limited diarrhea, tenesmus, and rectal bleeding [70,108,109]. Endoscopically inflammation of the mucosa, edema and ulcerations may be found [70]. This form of acute radiation injury is usually recognized clinically and treated symptomatically with *restituto ad integrum* within 4 weeks after therapy. It is self-limited and usually does not require imaging for diagnosis. However, 5 to 15% of patients experience severe radiation toxicity that may require temporary interruption of the treatment and may need MDCT to depict complication [70,108,110]. Prevention during external radiation therapy is the key of management. Control of radiation dose and local treatments along with endoscopic management are always preferred to surgical procedures [111–113].

**MDCT presentation**

During the acute phase of radiation injury, MDCT shows nonspecific regular and symmetric wall thickening with halo sign and inflammatory stranding adjacent to the affected region, typically the sigmoid colon and the rectum for prostate or cervical cancer [51]. MDCT appearance is nonspecific, but the clinical history helps suggest the correct diagnosis [114]. Complications, such as fistulas, strictures and abscesses, may also be demonstrated by MDCT.

MDCT findings in active phase of radiation-induced injury of the colon are similar to those observed in graft-versus-host disease and in acute infectious rectitis [73]. MDCT findings in the chronic phase of radiation-induced injury of the rectum raise the problem of tumor recurrence in the pelvis. In the other segments of the colon, MDCT findings...
such as strictures and fistulas may mimic chronic inflammatory diseases at a fibrous or chronic stage [73].

**Toxic colitis**

Clinical considerations

Chemotherapy-induced colitis has been reported with taxan and docitaxel [75]. Patients usually present with acute abdominal pain with rebound tenderness early after the beginning of the treatment. Fever and neutropenia may also be present in association with negative blood and stool cultures.

MDCT presentation

Toxic colitis may be limited to a single segment of the colon but may also be more extensive up to pancolitis. Mural thickening is usually moderate and usually the colon thickness is < 12-mm [75]. Mucosal enhancement, stranding of the peritoneal fat and limited ascites have also been described [75].

MDCT findings observed in patients with toxic colitis overlap with those of colitis of other origins, so that there are no findings specific for this entity. Infectious colitis should be considered as differential diagnosis in immune competent patients. Typhlitis and graft vs. host disease are differential diagnoses in immune deficient patients [74].

**Conclusion**

MDCT is the first-line imaging modality in patients with severe or atypical abdominal pain with biological inflammatory changes. MDCT helps suggest the positive diagnosis of acute colitis with high degrees of sensitivity and assess the extent and the severity of the disease. However, MDCT findings have low specificity because of marked overlap in presentation of different types of acute colitis. The radiologist should always keep in mind the major role of integrating the clinical context for a specific diagnosis. In case of equivocal diagnosis or atypical MDCT findings, the final diagnosis should be based on the results of endoscopic examination and result of colonic biopsy. However, knowledge of MDCT features of the various causes of acute colitis and findings suggestive for a severe form of the disease should help apply the most appropriate therapeutic option, thus avoiding delay in management.
Disclosure of interest
The authors declare that they have no conflicts of interest concerning this article.

References


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