Fulminant amebic colitis is an uncommon manifestation of amebiasis. The computed tomography (CT) appearance of fulminant amebic colitis (FAC) is rarely reported. Understanding the pathologic change of FAC may help recognize the characteristic CT findings of FAC. The classic pathologic feature of FAC is the macroscopic appearance. FAC usually shows multiple sites of necrosis and perforation of the colon, ranging from superficial ulcer, flask-shaped ulcer to transmural necrosis. Extended submucosal ulcer with intramural dissection is the characteristic CT finding of FAC. Other important CT findings include pancolitis with multiple foci of target sign and discontinuous bowel necrosis.

Entamoeba histolytica is an enteric protozoan that infects 6% of the world’s population [1]. Only 3% of afflicted patients have their clinical course complicated by fulminant colitis [2]. These patients are often toxic, secondary to toxic megacolon, perforation and peritonitis. Once perforation of a transmural necrosis has occurred, the mortality rate is extremely high, ranging from 55% to 92% [3]. The high mortality rate may be related to lack of clinical suspicion of FAC, severity of the disease, and its nonspecific CT findings [3-5]. The rate of correct preoperative diagnosis of FAC is low [6, 7]. Early diagnosis of FAC may lower the mortality rate. We describe here the characteristic CT findings of FAC.

CASE REPORT

A 72-year-old male was admitted to our hospital due to fever, diarrhea and painful swallowing for one week. Nasopharyngeal carcinoma (NPC) was diagnosed 6 weeks ago and post incomplete radiation therapy (20 days/25 days) which was stopped 7 days before admission. Subtotal gastrectomy due to perforated peptic ulcer was performed 10 months ago. He had no traveling history in the past few years. Physical examination revealed erythematous change and scaling of the neck. Injected oral mucosa with multiple ulcerations was found. The abdomen was soft, nondistended and without peritoneal sign. NPC post radiotherapy status and enterocolitis were our initial impression. The stool examination and serologic test for amebiasis were not performed. On the sixth day after admission, progression of shortness of breath, diarrhea and abdominal distension with peritonitis were noted. The laboratory data showed pancytopenia, hyperglycemia, an elevated C-reactive protein level and elevation of prothrombin time and activated partial prothrombin time. Septic shock due to hollow organ perforation was suspected. An emergent triple contrast (oral, rectal and intravenous) enhanced CT (4 detector-row) was performed. It revealed pneumoperitoneum, ascites and pancolitis with dilatation of the colon and terminal.
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ileum. Pericolic infiltration was minimal. There was colonic wall thickening (ranging from 8 mm to 14 mm), from cecum to rectum, with multiple foci of target sign appearance and intramural contrast medium accumulation (Fig. 1a, 1b, 2a). Imaging findings of bowel necrosis such as intramural air, no enhancement of the bowel wall and absence of the bowel wall were identified scattering in the cecum, ascending, transverse and descending colon. Multiple foci of discontinuous enhanced and nonenhanced colonic wall were noted (Fig. 2a, 2b). In the sigmoid colon, there were two large areas showing extended submucosal contrast medium accumulation with intramural dissection (Fig. 2c). Pancolitis with perforation was our initial diagnosis. Emergent laparotomy was performed. The laparotomy showed nearly total gangrenous change of the colon. Discoloration of the terminal ileum was also noted. Total colectomy with ileostomy was performed. The histological examination showed transmural gangrenous necrosis, flask-shaped ulcers (Fig. 3a, 3b) and perforations in the colon. Gangrenous necrosis in the mucosa and submucosa of the terminal ileum and appendix was noted. Numerous amebic trophozoites were seen in colon, terminal ileum, and appendix and pericolonic lymph nodes. Fulminant amebic colitis with gangrenous change and perforation was diagnosed. The patient’s condition got worse gradually, though Metronidazole was given intravenously after surgery. The patient expired on the thirteenth day after admission.

DISCUSSION

Amebiasis may present in a noninvasive or invasive form. The invasive trophozoite of ameba has the capacity to invade the colonic mucosa [7]. Right side colon and sometimes the whole colon are commonly involved by FAC [8]. The terminal ileum may be involved up to 10% [5]. Coexistence of an amebic liver abscess was reported in about 53% of cases [1]. Early preoperative diagnosis of FAC is difficult [6, 7]. Sigmoidoscopic or colonoscopic examination is difficult and also risky [3]. The positive rate of stool examination for amebic trophozoites is about 40% [7]. Diagnosis of amebiasis is most specifically and rapidly made by serologic test. About 85% sensitivity has been reported [7, 9]. But a false negative rate up to 41% has been mentioned [1].

In 1985, Luvuno et al. suggested that amebic invasion of the arteries supplying a segment of colon, consequent thrombotic occlusion and ischemic necrosis result in the progression from superficial ulcerations to transmural necrosis [8]. In 1994, Chun et al. described the morphologic change of FAC. It begins as a mucosal ulcer which extends and expands within the submucosa, undermining the adjacent mucosa [2]. In 1996, Li et al. also described the classic flask-shaped ulcer of amebiasis [10]. In 1996, Takahashi et al. described multiple sites of necrosis and perforation of the colon in all their 55 cases of FAC [1]. The CT findings of these above pathologic changes sometimes can be recognized.

Figure 1. a. Non-enhanced CT shows pneumoperitoneum (white arrow) and circumferential intramural contrast medium accumulation in the proximal descending colon (black arrow). b. A big ulcer with an extended intramural air collection and dissection in the descending colon is noted (white arrows).
Figure 2. a. Enhanced CT shows extended submucosal ulcer (black arrows) with intramural contrast medium dissection in the transverse and proximal descending colon. Target sign appearance and discontinuous enhanced and nonenhanced wall of the transverse colon (white arrows) are seen. b. Discontinuous bowel necrosis in the middle descending colon (white arrows) and absence of the bowel wall of the ascending colon with detached mucosa (black arrows) are seen. c. Two large extended submucosal ulcers (black arrows) with minimal pericolic infiltration in the sigmoid colon are noted.

Figure 3. a. Low power view of one segment of sigmoid colon shows a bowl-shaped necrotizing ulcer (U, arrow) extends from mucosa (M) to submucosa (SM). The submucosal layer shows necrosis, inflammation and with granulation tissues. Numerous amebic trophozoites are seen in the necrotic tissue and in the inflammatory submucosa (not shown). b. Deeper ulceration extending to the submucosa (arrows), then a subsequent lateral extension through the submucosa with undermining the normal mucosa, gives rise to the classic flask-shaped ulcer of amebiasis.
Understanding the morphologic change of FAC, from superficial ulceration to transmural necrosis and flask-shaped ulcer, may be helpful to recognize the CT imaging features of FAC.

CT is the established gold standard for assessing the presence and complications of amebic colitis and solid organ involvement. It is a highly sensitive method for the detection of intramural abnormality and extraluminal extension of colonic disease. Triple contrast enhanced CT was performed in our patient. The phenomenon of intramural contrast medium (CM) density is brighter than the intraluminal CM density, which may be related to the dilution effect of intraluminal pus and the higher intraluminal pressure that forced the rectal CM into the intramural space through a colonic ulcer. The submucosal ulcer with focal contrast medium accumulation on image Fig. 2a & c is more circumferential, showing curvilinear band in shape and nondependent. The intraluminal CM-fluid level seen in the normal colon is horizontal and dependent. In the absence of rectal contrast enema preparation, intravenous enhanced CT images may help to demonstrate the submucosal ulcer with focal CM accumulation. The focal or extended submucosal CM accumulation with intramural dissection reflects the underlying pathologic morphology change of flask-shaped ulcer and deeper ulceration. Though flask-shaped ulcer could be noted in moderate amebic colitis (without complication) and chronic amebic colitis, extensive intramural dissection of these conditions is unusual. From the pathologic point of view, this finding doesn't occur in other infectious colitis, inflammatory bowel disease and ischemic bowel disease. The characteristic CT finding in our patient which suggested the possibility of FAC is extended submucosal ulcer with intramural dissection. To the best of our knowledge, this is the first documented CT finding of FAC.

Another important CT finding is pancolitis with multiple foci of target sign appearance and alternating enhanced and nonenhanced bowel wall (discontinuous bowel necrosis). It reflects the microscopic and macroscopic feature of vascular occlusion and transmural necrosis due to multiple different sites of trophozoites colonic invasion [1, 2, 8]. Ulcerative colitis, typhilitis, pseudomembranous colitis, ischemic bowel disease, and cytomegalovirus (CMV) colitis could present as pancolitis with target sign appearance. However, multiple different sites and discontinuous bowel necrosis are very unusual in these diseases. The presence of ascites, is less common associated with ulcerative colitis.

Typhilitis occurs most frequently in patients with acute leukemia who are receiving chemotherapy. Pseudomembranous colitis is usually related to the use of broad-spectrum antibiotics. Ischemic bowel disease has its characteristic vascular distribution, in which simultaneous involvement of superior and inferior mesenteric arteries and rectum is very rare. Neither colonoscopic nor CT signs of colitis can reliably distinguish FAC and CMV colitis. CMV colitis often presents with longer duration of diarrhea, deeper colonic mural ulcer, prominent perirectal infiltration and sometime associates with small bowel involvement [11]. On the other hand, FAC, Crohn disease, pseudomembranous colitis and CMV colitis may present as skip lesions [2]. They should be included in the differential diagnoses.

In conclusion, understanding the pathologic change of FAC from superficial ulceration to transmural necrosis and flask-shaped ulcer may be helpful to recognize the CT imaging features of FAC. Extended submucosal ulcer with intramural dissection is the characteristic CT finding of fulminant amebic colitis. Pancolitis with multiple foci of target sign appearance and discontinuous bowel necrosis is another important CT finding. When the CT finding of extended submucosal ulcer with intramural dissection is identified, the possibility of FAC should be considered.

◆

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猛暴性阿米巴大腸炎之電腦斷層表徵：病例報告

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猛暴性阿米巴大腸炎，是阿米巴痢疾感染的一種不尋常表現。其電腦斷層表徵很少在文獻上發表過。了解其在病理上的形態學變化，可以使我們更容易了解並偵測到其在電腦斷層上之特徵。它的典型病理特徵，就在肉眼即可看出之外觀：在大腸上可以看到多處組織壞死及穿孔。猛暴性阿米巴大腸炎是先由淺層之腸壁黏膜潰瘍發展至全層腸壁之壞死，並合併有典型的燒杯狀潰瘍。由黏膜潰瘍至黏膜下潰瘍所發展出的擴張性黏膜下潰瘍合併腸壁內之剝離延伸，是猛暴性阿米巴大腸炎之典型電腦斷層特徵。另泛大腸炎合併有標靶樣之腸壁增厚及多處非連綿性之腸壞死也是其電腦斷層之重要發現。