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Case 1

A 21-year-old monk presented with chronic diarrhea and significant weight loss for 8 months. Stool exam revealed numerous RBC and WBC. Stool test for acid fast bacilli was negative. Colonoscopy showed multiple large transverse ulcers with exudates from the ascending colon to descending colon (Figure 1). Barium enema revealed multiple segments of mucosal irregularity and ulceration of the entire colon, with extension to involve ileocecal valve and terminal ileum (Figure 2). Chest radiograph showed reticulonodular infiltration of the right lung, predominantly on the upper lobe. Sputum for acid fast bacilli stain was positive. Computed tomography of the abdomen revealed multiple segments of thickened bowel wall, involving sigmoid colon up to cecum and terminal ileum (Figure 3). The degree of wall thickening is more on the right side. Several necrotic mesenteric nodes were present.

Colon biopsy showed numerous acute and chronic

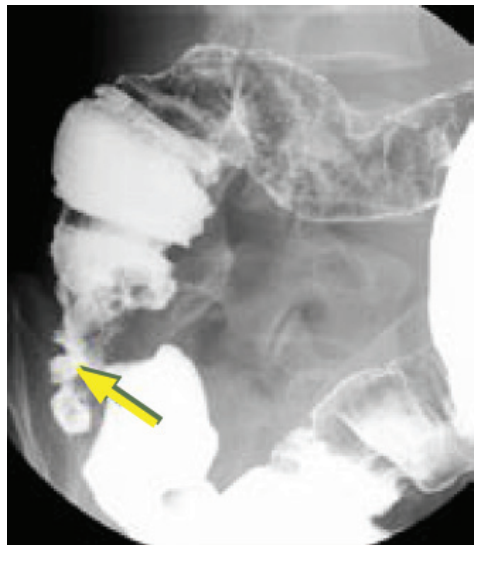

Figure 2. BE showed mucosal irregularity of cecum and IC valve.

Figure 1. Descending colon (Transverse ulcer).

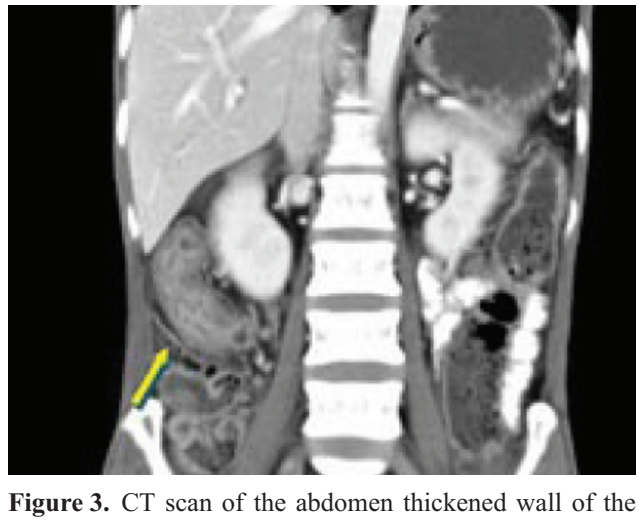

Figure 3. CT scan of the abdomen thickened wall of the ascending colon (yellow arrow).

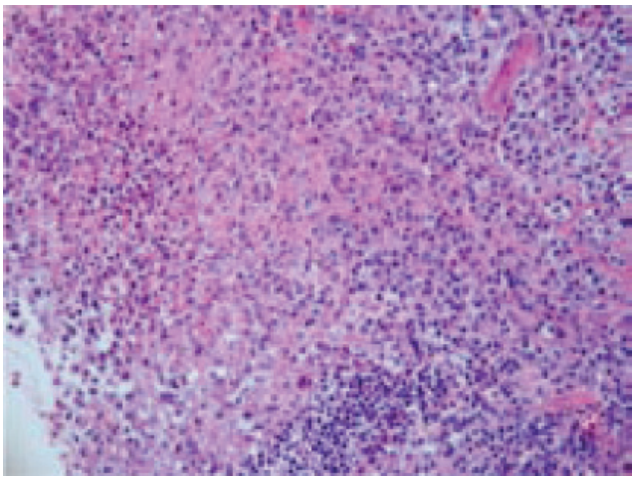

Figure 4. Vague granulomas the lamina propria, and ulcerated surface.

inflammatory cells infiltrating the lamina propria, forming vague granulomas with ulcerated surface (Figure 4). Tissue for acid fast bacilli stain and PCR for tuberculosis were negative.

Diagnosis:

Disseminated mycobacterium tuberculosis

Discussion:

Digestive system is one of the sites for extrapulmonary tuberculosis. The digestive system is involved in 66% of patients with abdominal tuberculosis[1]. The ileocecal region is the most commonly affected within the gastrointestinal tract followed by the colon. In colonic involvement, the cecum and ascending colon are most commonly affected, followed by the transverse and descending colon[2]. Autopsies of patients with pulmonary tuberculosis before the era of effective treatment demonstrated intestinal involvement in 55-90% of fatal cases[3]. Direct infection from the wall of the gut is highly possible after drinking unpasteurized milk or swallowing a large number of bacilli from the pulmonary cavity. Reactivation from the body within few years after hematogenous spreading is also a possible mode[2]. Tripathi and Amarapurkar studied 110 cases of TB in the GI tract to identify the morphologic spectrum of the disease. The most common clinical presentations were abdominal pain (82.7%), fever (58.2%), weight loss (53.6%), and diarrhea (29.1%). Concurrent involvement of the ileum, cecum and ascending colon were seen most frequently in 56 cases (50.9%). The next most common site of involvement was the terminal ileum alone, in 43 cases (39.1%)[4]. Common colonoscopic findings were ulcers (70%), nodules (56%), deformed cecum and ileocecal valve (40%), strictures (23%), polypoid lesions (14%), and fibrous bands forming mucosal bridges (7%)[5].

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CASE 2

A 19-year-old woman presented with chronic watery diarrhea and significant weight lost for 6 months. She had been in excellent health until 6 months ago. Stool ova and parasite examinations revealed no pathogen. Colonoscopy was performed and showed multiple small whitish mobile thin worms with thread-like anterior half, coiled and straight posterior end and penetrating mucosa of cecum (Figure 1-3).

Figure 1. A whitish whipworm with obtuse posterior end in cecum (red arrow); was recognized to be female worm.

Diagnosis:

Trichuris trichiura infestation

Discussion:

Trichuris trichiura (whipworm) infestation is an endemic in tropical and temperate countries, including Southeast Asia. Most patients are asymptomatic, especially if less than 10 worms or if only males are present, whereas, infestation with larger numbers of worms may cause abdominal pain, diarrhea, weight loss, and anemia[1]. Heavy colonic infection causes syndrome named Trichuris dysentery syndrome. Those patients mainly children presented with mucoid diarrhea, rectal bleeding, rectal prolapsed, iron deficiency anemia and clubbing of fingers[2].

Whipworm is transmitted by feco-oral route and inhabits the human cecum and proximal large bowel. The adult worms have a thin, tapered anterior region. The female worm is 30 to 50 mm in length, has an

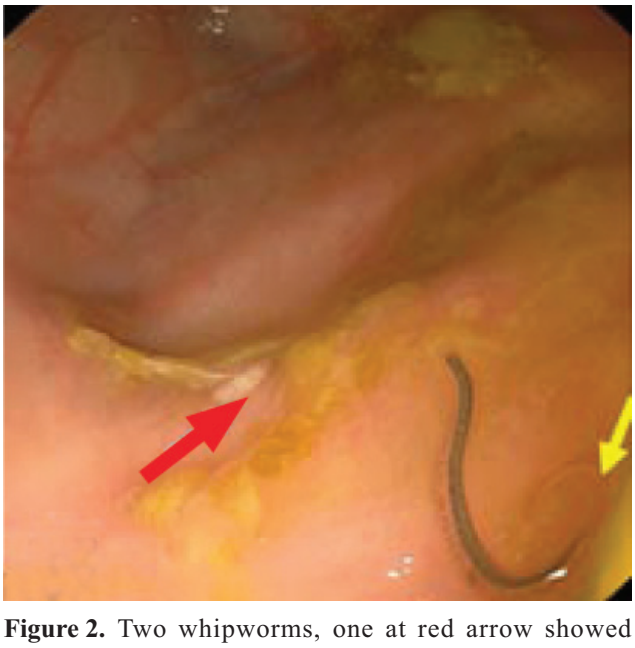

Figure 2. Two whipworms, one at red arrow showed straight posterior end, to be female whipworm and another one at yellow arrow showed coiled posterior end, to be male whipworm.

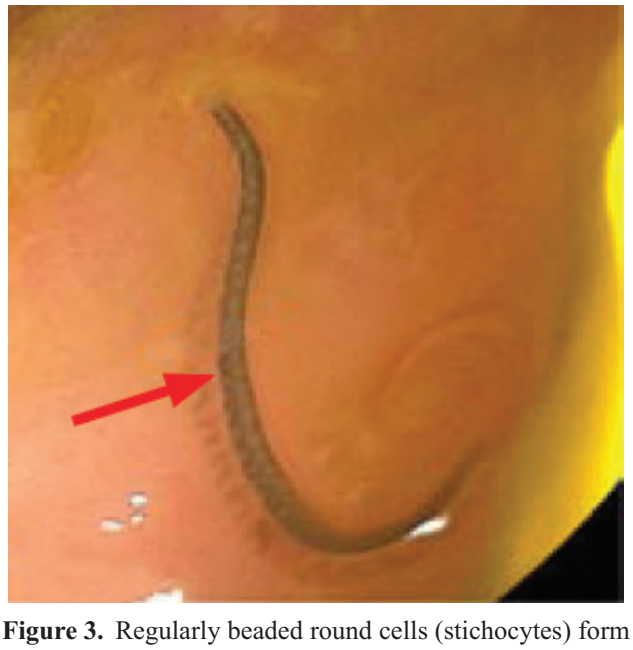

Figure 3. Regularly beaded round cells (stichocytes) form the stichosome (red arrow) in male whipworm.

uncoiled posterior extremity and lays 3,000 to 20,000 eggs per day. The male is slightly smaller, and has a coiled caudal extremity with a copulatory spicule2. At the esophageal part of whipworms, there are stichocytes made of number of stichosomes and stichocytes exhibit exocrine granules that contain a variety of excretory and secretory products that may alter host cell physiology to allow the worm to establish parasitism in the host[3].

Diagnosis of trichuriasis is by the demonstration of brown, barrel-shaped ova in feces. However, in some patients, stool examination could not show ova while colonoscopy could demonstrate whipworm infestation[1,4]. Colonoscopy usually demonstrates the mobile whitish worm; 30-50 mm in length with threadlike anterior end, which penetration in the mucosa. The worms are most common found in cecum. Surrounding colonic mucosa usually appeared edematous and erythematous but ulceration was not common1.

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CASE 3

A 66-year-old man with no previous medical illness presented with a 3-day history of bloody diarrhea and lower abdominal pain. One day prior to admission, he developed rectal bleeding. His stool examination demonstrated Entameba histolytica cysts. Sigmoidoscopy showed multiple discrete small ulcers with thick yellowish exudates and erythema rim. There was normal intervening mucosa along the rectosigmoid colon (Figure 1-2). Colonic biopsy revealed multiple foci of erosion with acute and chronic inflammatory infiltration in the lamina propia. No organism was seen in the submitted tissue. He was treated with 10-day metronidazole. Complete colonoscopy was performed 2 weeks later and it showed a markedly improvement of the lesions (Figure 3-4).

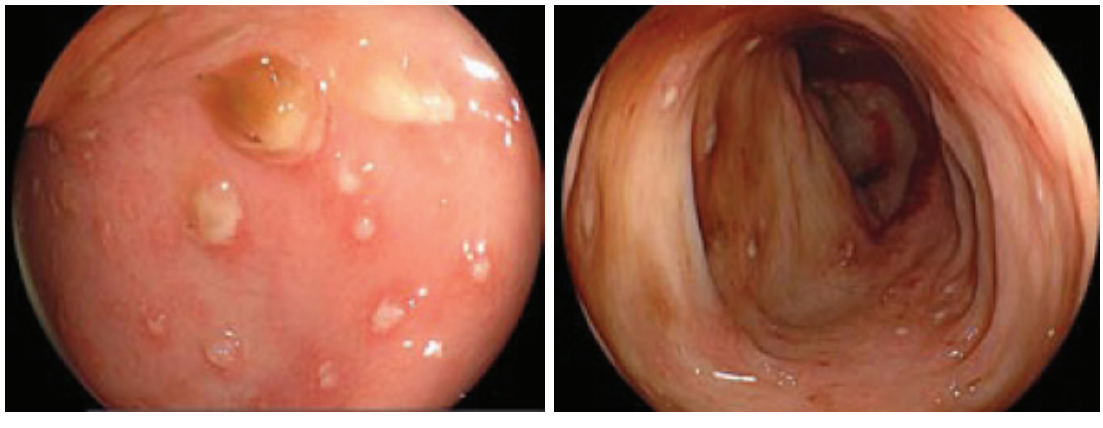

Figure 1-2. Multiple discrete small ulcers with thick yellowish exudates and erythema border.

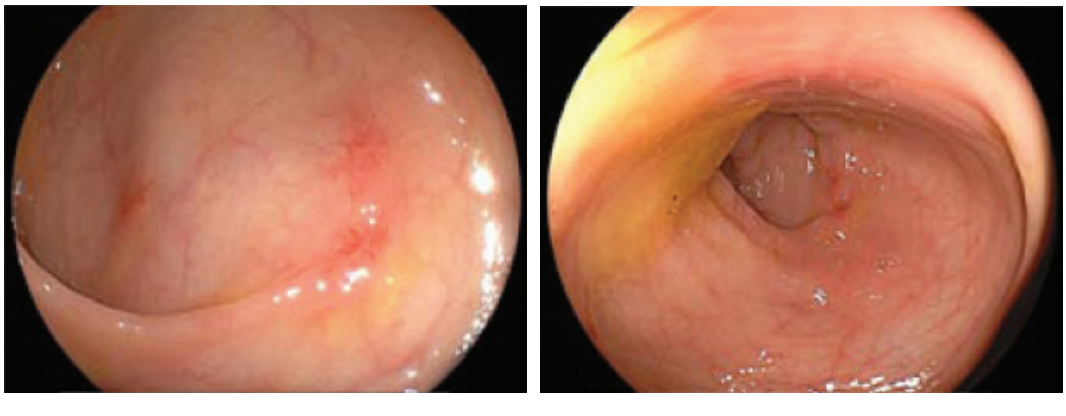

Figure 3-4. Follow-up colonoscopy.

Diagnosis:

Amoebic colitis

Discussion:

E. histolytica can infect people of both genders and all ages; however, populations at risk may vary with geographic location, host susceptibility, and differences in organism virulence. The simple life cycle of *E. histolytica* begins when infectious cysts are ingested in fecally contaminated food or water. After ingestion and passage through the stomach, the organism excysts and emerges in the large intestine as an active trophozoite. Trophozoites multiply by simple division and encyst as they move further down the large bowel. Cysts are then expelled with the feces and may remain viable in a moist environment for weeks to

months[1].

Amoebic colitis may occur days to years after initial infection and is characterized classically by abdominal pain and bloody diarrhea. Watery or mucus containing diarrhea, constipation, and tenesmus may also occur. Complications of intestinal disease include stricture, rectovaginal fistulas, formation of an annular intraluminal mass (amoeboma), bowel obstruction, perianal skin ulceration, toxic megacolon, perforation, peritonitis, shock, and death[1].

Colonoscopy is useful for the diagnosis of amoebic colitis but is not required if stool antigen detection or PCR is positive. Amoebic colitis can appear as punctuate hemorrhagic areas or small ulcers (up to centimeters in diameter) with exudative centers and hyperemic borders. The cecum and ascending colon are affected most commonly, although in severe disease the entire colon may be involved. In addition, early in the infection process, endoscopy results may be entirely normal. As disease progression occurs, mucosa may become hyperemic due to inflammatory changes, and pseudomembranes can occur, resembling inflammatory bowel disease. Aspirates content from colonic ulcers should be examined immediately microscopically for motile trophozoites[2].

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CASE 4

A 56-year-old man presented with fever and bloody diarrhea for 2 weeks. He had a history of kidney transplantation 2 months ago. He received immunosuppressive drugs; mycophenolate soium, tacrolimus and prednisolone 10 mg/D. Colonoscopy was performed. It revealed diffuse subepithelial hemorrhage and multiple shallow ulcers extend from sigmoid to terminal ileum (Figure 1-4). Biopsy showed erosive surface and edematous lamina propia. Numerous neutrophils and lymphoplasmacytic cells infiltrated in lamina propia. Many endothelial cells of vessels in lamina propia showed large cells with intranuclear inclusions (Figure 5). The diagnosis was CMV colitis. CMV immunohistological stain was positive in several cells. His serum CMV viral load was 51,800 copies/mL.

Figure 1. Diffuse subepithelial hemorrhage of the colon.

Figure 2. Multiple ulcers with edematous rim.

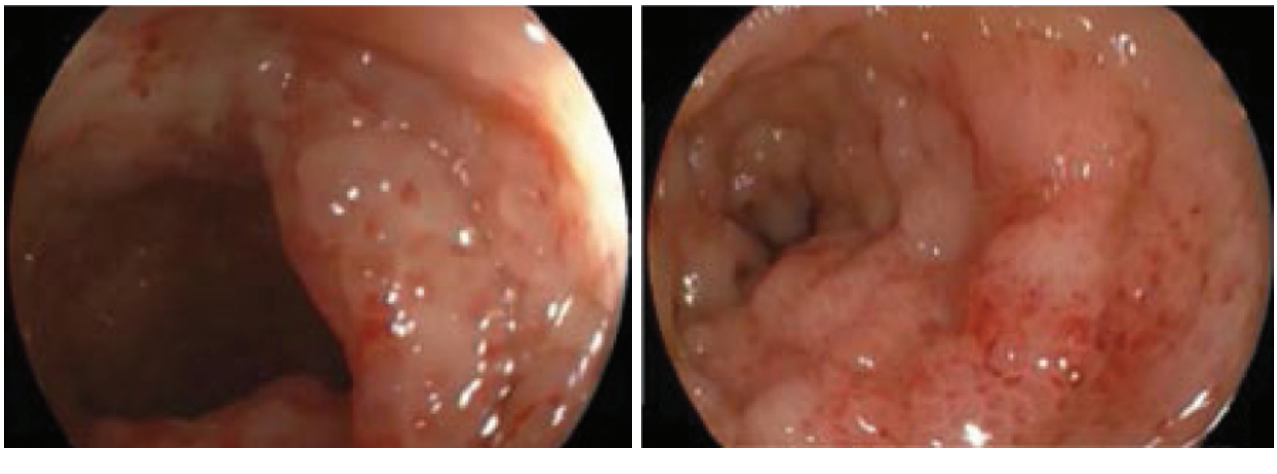

Figure 3-4. Severe edematous colonic mucosa with subepithelial hemorrhage.

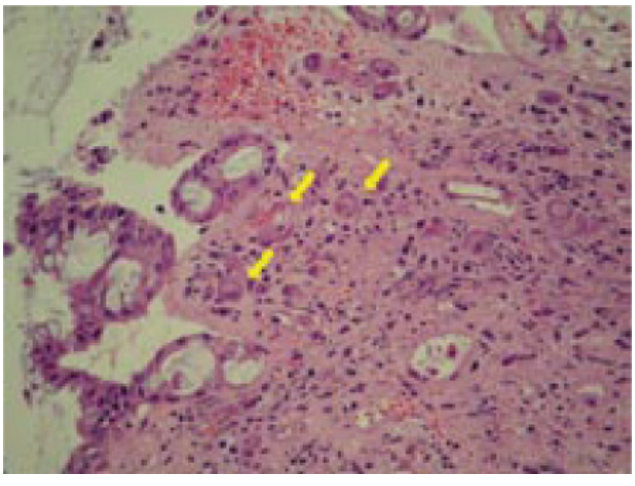

Figure 5. CMV infected cell with intranuclear inclusion (yellow arrows).

Diagnosis:

Cytomegalovirus colitis

Discussion:

CMV remains the single most important pathogen affecting the outcome of solid organ transplantation. CMV has the direct effects of morbidity and mortality related to infection, but also contributes to a multitude of short and long-term indirect effects mediated by its modulation of the immune system. Luminal tract disease is the most common manifestation. Esophagitis and colitis are the most frequently observed luminal syndromes, usually characterized by ulcerative lesions[1].

CMV colitis usually manifests with abdominal pain, persistent small-volume diarrhea, and rectal bleeding. Bloody diarrhea or hematochezia are the most common symptoms in immunocompetent patients with CMV colitis. Although a wide spectrum of findings can occur, typical endoscopic findings are mild and patchy to include erythematous colonic mucosa with edema and subepithelial hemorrhage. Less commonly endoscopic findings are discrete ulceration surrounded with normal colonic mucosa, colitis with ulceration, and pseudomembrane formation (very rare). The gold standard for diagnosis remains histopathology and immunohistochemical staining for CMV is the best confirmation test[2].

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