CASE REPORT

Amebic and cytomegalovirus colitis mimic ulcerative colitis

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Summary Here we present a 50-year-old man who suffered from progressively bloody diarrhea for 2 months. A colonoscopy revealed pancolonic mucosal inflammation, ulceration, and spontaneous bleeding. Ulcerative colitis was initially diagnosed and sulfasalazine was prescribed. Hypoalbuminemia and renal function deterioration developed 1 year later. Steroids were prescribed for suspected nephrotic syndrome. His bloody diarrhea and abdominal symptoms worsened after steroid use. Progressive sepsis and acute renal function deterioration also developed. Positive human immunodeficiency virus (HIV) antibody was found during routine hemodialysis screening. An episode of colon perforation occurred and surgery was performed. The resected colon showed amoeba, cytomegalovirus, and fungal infection. The patient died of sepsis. In this report, we discuss how to diagnose ulcerative colitis. It is important to exclude infection before using an immunosuppressive agent.

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Introduction

The prevalence of inflammatory bowel disease (IBD) has increased in the past 10 years in Taiwan [1]. Treatment of Crohn's disease (CD) and ulcerative colitis (UC) with immunomodulators and biological (anti-TNFα) therapy is becoming more and more common; however, the use of immunosuppression agents increases the risk of opportunistic infection. Making a correct diagnosis before using these immunomodulators is important. Screening for underlying Hepatitis B virus (HBV) infection in all patients and human immunodeficiency virus (HIV) infection for adolescent and adult patients with IBD was recommended in the 2014 European Crohn's and Colitis Organization's (ECCO) guidelines [2]. Here, we present a case of bloody diarrhea initially diagnosed as UC, but which was actually found to be amebic and cytomegalovirus infections in a HIV-positive patient. This particular case highlights the importance of an accurate diagnosis when treating colitis patients.

Case report

A 50-year-old man presented with progressive bloody diarrhea for 2 months. He was a businessman without systemic diseases. He had suffered from bloody diarrhea as frequently as four to five times per day since April, 2011. He visited a local hospital where a colonoscopy revealed pancolonic ulcerations with spontaneous bleeding (Fig. 1). The pathology report showed severe colitis with ulcers and focal crypt abscesses. UC was diagnosed. Sulfasalazine was given and his symptoms gradually improved. However, hypoalbuminemia and renal function deterioration developed 6 months later. Sulfasalazine was replaced with mesalamine. A colonoscopy in May 2012 revealed multiple ulcers scattered over the whole colon and rectum. The pathology report showed nonspecific colitis. As his bloody diarrhea and abdominal fullness progressed, he was referred to our hospital. Oral and suppository mesalamine were prescribed and his symptoms improved. Hypoalbuminemia (Alb: 3.2 g/dL), proteinuria (600 mg/dL), and renal insufficiency (Crea: 1.6 mg/dL) were still noted. His autoimmune profile, including C3, C4, IgA, ANA, p-ANCA, and c-ANCA, were all normal. A renal biopsy was suggested, but the patient refused. Under the impression of nephrotic syndrome, prednisolone (50 mg/day) was used, starting in March 2013. His proteinuria and hematuria partially improved and his case was followed up at the original hospital.

However, the patient continued to suffer from aggravating bloody diarrhea and abdominal fullness from July 2013. No body weight loss was noted during this period. Sigmoidoscopy revealed severe inflammation of the rectal mucosa and deep skip ulcerations with spontaneous bleeding (Fig. 2). He was referred to the emergency department of our hospital. On physical examination, pale mucosa and periumbilical abdominal tenderness were noted. The blood test results showed hemoglobin: 6.1 g/dL, WBC: 7.06 k/μL, Seg: 76%, Band: 16%, and platelet counts: 283 k/μL. The biochemistry test showed Alb: 3.1 g/dL, BUN: 25.3 mg/dL, Crea: 1.1 mg/dL, and CRP: 14.05 mg/dL. A stool sample showed 4+ occult blood and two to five pus cells/HPF. No protozoa or parasite ova were found in the stool. Empirical antibiotics with the third generation of cephalosporin, intravenous-form steroid [methylprednisolone 20 mg/day], and mesalamine were prescribed. However, bloody stool (>5 times/day), hypotension, and anuria developed. Stool tests, including protozoa, parasite ova, and bacterial cultures, all appeared negative. As hypoxemia progressed, intubation was performed. Hemodialysis was arranged, but the routine screening revealed anti-HIV antibody positive and western blot analysis confirmed the positive status of an HIV infection. The HIV viral load was 141,000 cp/mL. His hemogram showed WBC 14.32 k/μL, seg: 89.1%, Band: 8.9%, Meta: 1%, Lymph: 0%. The CD4 count was zero. A sudden onset of severe abdominal distension with peritoneal signs occurred, and a CT scan

Figure 1 Colonoscopy image showing diffuse inflammatory mucosa associated with multiple ulcerations over the whole colon.

Figure 2 Colonoscopy image showing well-demarcated ulcerations in the sigmoid colon.
showed air in the rectum wall (Fig. 3), massive ascites, and free air in the abdomen (Fig. 4).

An emergency operation was performed, and multiple perforations from the ascending colon to the rectum, significant pus debris at the peritoneum, and massive dirty ascites were discovered. Amebic trophozoites (Fig. 5), CMV inclusion bodies (Fig. 6), and fungal hyphae (Fig. 7) were identified in the resected colon specimen. Broad-spectrum antibiotics with an antifungal agent were prescribed. Serum tests showed positive IHA for amoeba, and a CMV viral load of 272,000 cp/mL. The ascites culture yielded Enterococcus faecium, Candida albicans, and Candida krusei. The pleural effusion culture yielded Aspergillus fumigatus. Despite aggressive medical treatment, his condition deteriorated gradually, and he passed away about 10 weeks after admission.

Discussion

According to the most up-to-date ECCO guidelines, in order to make a diagnosis of UC, physicians should combine clinical evaluation, endoscopic, and histological findings, and exclude infectious causes [3]. The clinical presentation of this patient was bloody diarrhea, the most common symptom of UC in Taiwan [4]. However, bloody stool may also arise from a variety of diseases. Late onset UC (onset age: over 40) usually presents as a mild disease and requires surgery less frequently [5]. Endoscopically, continuous mucosal inflammation and confluent involvement from the rectum to the colon are typical features [3]. A pathologic diagnosis should be based on the presence of basal...
plasmacytosis, widespread crypt architectural distortion, and diffuse increased transmucosal lamina propria cells [3]. An endoscopic finding of HIV enteropathy may mimic the features of IBD and lead to misdiagnosis. In patients with advanced HIV infection, CD4 T-cell count depletion and opportunistic infections (OIs) occurs. OIs include bacterial, viral, fungal, and protozoan infections. CMV is the most common opportunistic pathogen in HIV-infected patients and can involve the whole gastrointestinal (GI) tract. Eosinophilic nuclear inclusions are characteristic of CMV-infected cells and are usually found in stromal and endothelial cells [6]. Entamoeba histolytica infections are usually asymptomatic; however, in immunocompromised patients, invasive amebiasis has mostly presented as colitis and liver abscesses. The trophozoite invades the colonic mucosa and spreads to the extra-intestinal organs. Endoscopy is useful in diagnosing amebic colitis, since amebic trophozoites can be detected from the biopsy [7]. The most common fungal infection in immunocompromised patients is candidal esophagitis; however, enteric fungal infections are unusual, even in HIV-infected patients. The diagnosis was based on fungal hyphae noted in the biopsy specimen. Highly active antiretroviral therapy (HAART) decreased the incidence and prevalence of OIs.

We reviewed the patient's previous data, endoscopic findings, and histological results. Neither typical, continuous, nor confluent mucosal inflammation, ulcerations, granularity, nor loss of visible vascular pattern of the mucosa were present. The initial pathology report revealed the presence of ulcers with focal crypt abscesses, which were nonspecific features of colitis. Furthermore, when the slides were reviewed again, the initial biopsy revealed the presence of amebic trophozoites. Therefore, if the slides had been examined in more detail, the correct diagnosis may have been made earlier.

Sulfasalazine had antiinflammatory effects through the inhibition of cytokine synthesis, inhibition of prostaglandin and leukotriene synthesis, and scavenging of reactive oxygen species [8]. His condition was relatively stable during the sulfasalazine treatment period. However, nephrotic syndrome developed 6 months later. Sulfasalazine has been reported to induce nephrotic syndrome at an extremely low frequency [9]. Prednisolone was prescribed for the nephrotic syndrome that led to rapid deterioration of his colitis. Again, making an accurate diagnosis initially and introducing effective management are the best strategies for treating these patients.

In summary, we reported a patient who presented with bloody diarrhea diagnosed as UC initially, but a detailed review of the endoscopic and histological findings resulted in the diagnosis being changed to infectious colitis. The immunosuppressive agent used aggravated his disease. While there is an increasing prevalence of IBD in Taiwan, making a diagnosis of UC should be based on medical history, clinical evaluation, endoscopic image, and histological findings, and, most importantly, the exclusion of infectious disease.

Conflicts of interest

All authors declare no conflicts of interest.

References