Endoscopic appearance of GI mycobacteriosis caused by the Mycobacterium avium complex in a patient with AIDS: case report and review

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Disseminated Mycobacterium avium complex (MAC) infection, an important AIDS-defining opportunistic infection in patients with advanced HIV infection (generally those with CD4 lymphocyte counts <50 cells/µL), is associated with significant morbidity and mortality, and with shortened survival.\(^1\)\(^2\) The incidence of disseminated MAC (DMAC) infection increased during the period 1982 to 1994, with the maturation of the AIDS epidemic,\(^3\) to an estimated cumulative incidence of 15% to 24% of patients with AIDS.\(^4\) With the introduction of antimicrobial prophylaxis and highly active antiretroviral therapy (HAART), the incidence of MAC infection dramatically declined and survival improved.\(^5\) According to the U.S. Centers for Disease Control and Prevention, the incidence of MAC infection decreased from 10 cases per 100 person-years in 1992 to two cases per 100 person-years in 1998.\(^6\) Survival increased from 40 days before the introduction of HAART to 398 days in the post-HAART era.\(^3\) Despite advances in the care of patients with AIDS and MAC infection, new or recurrent MAC infection continues to occur in patients with limited access to care for HIV infection and in those who do not respond to antiretroviral therapy.\(^5\)\(^7\)\(^8\) The GI tract appears to be a common portal of entry for MAC infection in patients with AIDS. Local replication may result in the formation of local foci with subsequent dissemination via lymphatics within months.\(^9\) A case of AIDS with endoscopic finding of MAC infection of the duodenum is presented. Reported cases that include an endoscopic description are reviewed, with the aim of improving early recognition of MAC infection in the GI tract.

CASE REPORT

The patient, a 42-year-old, homosexual man with HIV infection diagnosed 6 years earlier, presented with a 2-month history of epigastric pain, abdominal bloating, and intermittent fever. Two months before hospitalization, the plasma viral load had rebounded and the CD4 count was 63 cells/µL despite HAART.

At the time of diagnosis of HIV infection, antiretroviral and prophylactic antimicrobial therapy was begun, but the patient did not continue this treatment, because of severe GI symptoms. Pneumocystis carinii pneumonia (PCP) and tuberculosis developed at 4 and 5 years, respectively, after the diagnosis of HIV infection, and the patient was treated with trimethoprim-sulfamethoxazole and antituberculosis therapy. However, he was not compliant with the prescribed therapy. Eight months before presentation, nontyphoid salmonellosis and recurrent PCP developed, and blood cultures subsequently grew MAC. HAART and anti-MAC therapy (ciprofloxacin, clarithromycin, and ethambutol) were initiated.
Examination revealed mild tenderness in the right and the left upper abdominal quadrants. The CD4 lymphocyte count was 41 cells/μL; the plasma viral load was undetectable (<400 copies/mL by reverse transcriptase-polymerase chain reaction). Computed tomography of the abdomen disclosed extensive lymphadenopathy in the para-aortic region and the bilateral adrenal glands, and also localized wall thickening of the jejunum. A liver biopsy, performed because of marked elevation of the serum level of alkaline phosphatase (1595 U/L, [normal: 60-220 U/L]) and γ-glutamyl transferase (267 U/L, [<52 U/L]), revealed abundant acid-fast bacilli. No specific findings were noted at colonoscopy, but EGD disclosed diffuse, scattered white nodules and plaques in the second portion of the duodenum (Fig. 1). Cultures of blood and tissue obtained from biopsies of the liver and the duodenum all yielded MAC, despite maintenance therapy with ciprofloxacin, clarithromycin, and ethambutol. Although the symptoms gradually improved with continuation of anti-MAC treatment and HAART, the patient still complained of epigastric pain, abdominal bloating, and intermittent diarrhea. The patient developed progressive dyspnea, urinary difficulty, and retention over several days, and was admitted to our hospital. The symptoms rapidly worsened on the first hospital day, with severe respiratory distress. Lactic acidosis developed, and the patient died on the day of admission, despite resuscitation.

DISCUSSION

Although opportunistic infection with MAC is common in patients with advanced HIV infection, there are few detailed endoscopic descriptions of MAC infection of the GI tract. A search of the MEDLINE database (English language publications) identified 54 reported cases of DMAC infection with involvement of the GI tract in patients with AIDS in which an endoscopic description was included.8,10-24 The demographic, clinical, and endoscopic features, and the outcome for the 55 cases (including the present case) are shown in Table 1. Most reported cases predate the HAART era; only 3 patients received HAART.8,24 The median age of the patients was 32 years (range 14 months to 50 years). The common presenting symptoms included diarrhea (93%), fever (89%), weight loss (87%), and abdominal pain (82%). GI manifestations included gastric ulceration, enterocolitis, enteric fistulas, intra-abdominal abscess, and hemorrhage.1,21 Anemia, neutropenia, elevated alkaline phosphatase, hepatosplenomegaly, lymphadenopathy, and pneumonia also were observed, because dissemination to multiple organ systems was present in most of the patients. In the 55 reported cases of MAC infection of the GI tract, the duodenum (76%) was the most common site, followed by the rectum (24%), the ileum (6%), the colon (4%), the esophagus (4%), the jejunum (2%), and the stomach (2%). The CD4 count at presentation was available in only 4 cases; the median CD4 count was 41 cells/μL (6-69 cells/μL).8,15,23,24 Most patients died soon after the diagnosis of DMAC infection, despite antimycobacterial therapy. The high mortality rate for patients with GI DMAC infection is probably explained by the fact that only 3 patients received macrolide-containing treatment regimens and HAART.

Because patients with DMAC infection are in an advanced stage of HIV infection, concurrent GI infections may be present. Multiple pathogens, such as microsporidia, Cryptosporidium, Giardia, cytomegalovirus (CMV), and Isospora, have been identified by EGD in the duodenum of HIV-infected patients with chronic unexplained diarrhea.25 EGD findings may be nonspecific, with the exception of ulcerative lesions, which are frequent in CMV gastro-duodenitis.26 In our review, the most common endoscopic finding was multiple raised nodules (38%) in the involved sites. These nodules could be yellow, white, yellow-whitish, or pink. Although nodular lesions are frequent,9 endoscopy in patients with MAC infection of the GI tract may disclose normal findings (36%), ulcerations (11%), erythema (13%), edema (11%), friability (11%), a reduced mucosal vascular pattern (9%), erosions (4%), confluent nodules (4%),

Figure 1. A, Endoscopic view showing scattered white nodules and plaques in second portion of duodenum. B, Magnified endoscopic view.
<table>
<thead>
<tr>
<th>Reference</th>
<th>Age/gender</th>
<th>Clinical presentation</th>
<th>Involved GI tract</th>
<th>Endoscopy/autopsy finding</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Strom et al&lt;sup&gt;10&lt;/sup&gt;</td>
<td>30/M</td>
<td>Spiking fever, diarrhea, weakness, abdominal pain secondary to a massively enlarged spleen</td>
<td>Small bowel</td>
<td>Prominent folds in the mucosa with elevated yellowish grains and nodules on the surface of the villi</td>
<td>Died</td>
</tr>
<tr>
<td>Gillin et al&lt;sup&gt;11&lt;/sup&gt;</td>
<td>39/M</td>
<td>Diarrhea, fever, weight loss</td>
<td>Duodenum</td>
<td>Minute superficial ulcerations</td>
<td>Died</td>
</tr>
<tr>
<td>Caya et al&lt;sup&gt;12&lt;/sup&gt;</td>
<td>32/M</td>
<td>Diarrhea, weight loss, fever</td>
<td>Duodenum</td>
<td>Diffuse and raised whitish lesions, 2-4 mm in diameter</td>
<td>Died</td>
</tr>
<tr>
<td>Wolke et al&lt;sup&gt;13&lt;/sup&gt;</td>
<td>40/M</td>
<td>Abdominal pain, bloody diarrhea</td>
<td>Rectum and sigmoid colon</td>
<td>Edematous, erythematous, and friable, with multiple linear and oval erosions</td>
<td>Survived</td>
</tr>
<tr>
<td>Roth et al&lt;sup&gt;14&lt;/sup&gt;</td>
<td>30/M</td>
<td>Fever, diarrhea, weight loss</td>
<td>Duodenum</td>
<td>Erythematous macular mucosal lesions</td>
<td>Died</td>
</tr>
<tr>
<td>Schneebaum et al&lt;sup&gt;15&lt;/sup&gt;</td>
<td>38/M</td>
<td>Diarrhea, anorexia, malaise, low-grade temperature, weight loss</td>
<td>Terminal ileum</td>
<td>Strictures with considerable compromise of the intestinal lumen, mucosal ulcerations, and thickening of the bowel wall (resection specimen)</td>
<td>Alive</td>
</tr>
<tr>
<td>Vasquez-Iglesias et al&lt;sup&gt;16&lt;/sup&gt;</td>
<td>24/F</td>
<td>Diarrhea, weight loss, pruritis, mild asthenia, anorexia</td>
<td>Duodenum</td>
<td>Several isolated, yellow, and raised nodules of 2 x 4 mm; more numerous and confluent distally</td>
<td>Survived</td>
</tr>
<tr>
<td>Gray and Rabeneck&lt;sup&gt;17&lt;/sup&gt;</td>
<td>35 cases; mean 35.1/M</td>
<td>Weight loss (30/35); fever, sweat, or chills (21/35); diarrhea (17/35); abdominal pain (7/35)</td>
<td>Duodenum (30), esophagus (2), rectum (7)</td>
<td>Duodenum: normal (17), fine white nodules on the mucosa (12) Rectum: normal (3), perianal fistula (1), reduced vascular pattern (2), rectal ulcer (1) Esophagus: ulcers (2)</td>
<td>Died (32/35)</td>
</tr>
<tr>
<td>Connolly et al&lt;sup&gt;18&lt;/sup&gt;</td>
<td>4 cases; NA</td>
<td>Persistent diarrhea, weight loss</td>
<td>Rectum</td>
<td>Loss of vascular pattern, generalized inflammation and mucosal friability;</td>
<td>NA</td>
</tr>
<tr>
<td>Monsour et al&lt;sup&gt;19&lt;/sup&gt;</td>
<td>36/M</td>
<td>Fever, left upper quadrant pain, explosive diarrhea, weight loss</td>
<td>Duodenum</td>
<td>Diffuse white nodules 2-3 mm in diameter; superficial erosions</td>
<td>Died</td>
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<tr>
<td>30/M</td>
<td></td>
<td>Fever, malaise, anorexia, hepatosplenomegaly, epigastric burning, melena, weakness, confusion</td>
<td>Duodenum</td>
<td>Small, white nodules with surrounding erythema</td>
<td>Died</td>
</tr>
<tr>
<td>Cappell and Gupta&lt;sup&gt;20&lt;/sup&gt;</td>
<td>27/F</td>
<td>Watery diarrhea, fever, abdominal pain, weight loss</td>
<td>Stomach, small and large intestine</td>
<td>Diffuse ulceration</td>
<td>Died</td>
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(continued)
expression (2%), and aphthous erosions (2%). Thus, for definitive diagnosis in symptomatic patients, especially those without typical endoscopic findings, it is essential to obtain tissue specimens for histopathologic examination and microbiologic culture.

The introduction of effective antiretroviral therapies for HIV infection and antimicrobial prophylaxis against MAC infection have had a major impact on the incidence and the clinical course of MAC infection in patients with AIDS, but patients with new or recurrent MAC infection are still encountered in clinical practice. This may occur for several reasons: the patient may be unaware of the HIV infection; lack of access to care for HIV infection; poor compliance with treatment, including prophylaxis, for MAC infection; and poor adherence to HAART, resulting in virologic failure and suboptimal immunologic recovery. Because GI MAC infection may be the first presentation of AIDS-associated opportunistic infection, it is essential to be alert for endoscopic findings, even in the era of effective antiretroviral therapies. Early recognition of GI MAC infection by endoscopy in HIV-infected patients and initiation of anti-MAC therapy and HAART may reduce morbidity and mortality.

REFERENCES


Double-balloon enteroscopy for diagnosis of a Meckel’s diverticulum in a patient with GI bleeding of obscure origin

Antonio Gasbarrini, MD, Simona Di Caro, MD, Massimiliano Mutignani, MD, Giovanni Cammarota, MD, Lucia Fini, MD, Fabio Pacelli, MD, Paolo Pola, MD, Giovanni Doglietto, MD, Guido Costamagna, MD, Giovanni Gasbarrini, MD

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Standard EGD, colonoscopy, and radiologic imaging studies fail to demonstrate a source in about 10% of patients with GI bleeding. Because the small bowel is only partially accessible with endoscopy, it is the site of most undiagnosed lesions. Meckel’s diverticulum is the most common congenital anomaly of the GI tract (approximately 2%-3% of the population). It is the result of improper closure and absorption of the omphalomesenteric duct and usually is located within 100 cm of the ileocecal valve. There is a strong male preponderance, and 60% of patients with Meckel’s diverticulum come to medical attention before 10 years of age. Heterotopic gastric or pancreatic mucosa frequently is found within the diverticulum in patients with symptoms because of the anomaly. Patients with a Meckel’s diverticulum that contains heterotopic gastric mucosa (30% of cases) are more likely to develop symptoms than those with a diverticulum that contains only intestinal mucosa. The most common complications are hemorrhage as a consequence of peptic ulceration, obstruction, intussusception, volvulus, and diverticulitis. Although the clinical, histopathologic, and radiologic features of the complications of Meckel’s diverticulum are well known, the diagnosis may be difficult before surgery.²