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Peritonitis due to *Mycobacterium avium* complex in patients with AIDS: report of five cases and review of the literature

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Summary
Background: *Mycobacterium avium* complex (MAC) often causes disseminated infection in patients at an advanced stage of HIV infection; however, peritonitis associated with disseminated MAC infection is rare.
Methods: In this report, we describe five cases of MAC peritonitis in AIDS patients at our hospital, and analyze these cases alongside 11 previously published cases found in the literature.
Results: Most of the AIDS patients with MAC peritonitis had CD4 counts of <50 cells/μl and elevated alkaline phosphatase levels. Other than ascites, MAC was also isolated from other sterile and non-sterile sites. Of note, six patients (37.5%) presented with chylous ascites.
Conclusions: Despite therapy, MAC peritonitis is associated with a grave outcome.

Introduction

Mycobacterium avium complex (MAC) infection is a common complication of advanced-stage HIV-1 infection, which often occurs in patients with CD4 lymphocyte counts of <50 cells/μl.[1] Before the introduction of highly active antiretroviral therapy (HAART) and primary prophylaxis, MAC infection in those patients at an advanced stage of HIV infection was often associated with significant mortality and morbidity. Although the incidence of MAC infection has declined substantially in the era of HAART, it continues to occur in patients without access to HAART or in patients who develop virological and immunological failure to HAART due to drug intolerance or the emergence of antiretroviral resistance.[2]

MAC can be isolated from non-sterile sites, such as specimens of the gastrointestinal and respiratory tracts, which may represent colonization or disseminated infection. In disseminated MAC infection, the organs most commonly involved are the spleen, lymph nodes, liver, intestines, and bone marrow; the lungs, adrenals, stomach, and central

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nervous system are less commonly involved.[3] Although MAC is a well-described pathogen in AIDS patients, peritonitis due to MAC has rarely been described.[4] In this report we describe five cases of peritonitis due to MAC and review the literature.

Methods

Between June 1994 and February 2008, 1778 non-hemophiliac HIV-infected adult patients who sought medical care at the National Taiwan University, the largest referral hospital for HIV inpatient and outpatient care in Taiwan, were consecutively enrolled in an observational study to describe the clinical spectrum of HIV-related opportunistic illnesses. Over the 14-year study period, 88 patients (4.9%) were diagnosed as having MAC infection; among these patients, 46 (52.3%) had disseminated MAC infection that was diagnosed based on the isolation of MAC from sterile sites. Five patients had MAC isolated from ascites and presented with clinical manifestations consistent with peritonitis. A standardized case record form was used to collect clinical information on these five patients.

We also reviewed the literature available on this subject by performing a PubMed search using the terms '*Mycobacterium avium* complex and peritonitis', 'AIDS and peritonitis', 'HIV and peritonitis', '*Mycobacterium avium* complex and ascites', and '*Mycobacterium avium* and ascites'. The abstracts of the articles selected in each of these multiple searches were identified, and those dealing particularly with the subject were recorded and reviewed in full form. Clinical presentations, relevant laboratory results, treatment course, and overall outcome were recorded.

The diagnosis of MAC peritonitis required the isolation of the organisms from ascites fluid either by culture or molecular biology assays. Disseminated MAC infection was defined as isolation of MAC from the blood, bone marrow, liver biopsy specimen, or specimens from two or more non-contiguous sites. Patients with MAC isolates recovered from specimens of respiratory tract (sputum, bronchoalveolar lavage, or sinus aspirate), gastrointestinal tract (gastric lavage, colon biopsy, or stool sample), or wound were not considered to have disseminated MAC infection when cultures of sterile sites were negative. Chylous ascites was defined as a milky-appearing peritoneal fluid that was rich in triglyceride ($\geq$200 mg/dl).

Results

Case report

A 42-year-old homosexual man with a CD4 count of 13 cells/μl, who had failed to respond to HAART, sought medical care at an outside hospital because of intermittent high fever, diffuse abdominal pain, watery diarrhea, and weight loss for 6 months. He had no history of liver disease or alcohol abuse. A diagnosis of sclerosing mesenteritis was presumptively made according to the findings of computed tomography (CT) of the abdomen. He later presented to our hospital with aggravation of the above symptoms after several months of conservative treatment.

On physical examination, the patient's temperature was 38 °C. He was cachectic, with temporal wasting. His abdomen was markedly distended with a doughy consistency on palpation and dullness on percussion. There was diffuse abdominal tenderness with mild muscle guarding and rebound tenderness. The liver and spleen could not be palpated.

Initial laboratory data included: white blood cell count (WBC) of 3.73×10^9 cells/l with 85.2% neutrophils, 7.8% lymphocytes, 6.7% monocytes, and 0.3% basophils, hemoglobin 12.2 g/dl, platelets 194×10^9 cells/l, aspartate aminotransferase 50 U/l, alanine aminotransferase 21 U/l, alkaline phosphatase (ALP) 125 U/l, and C-reactive protein 11.5 mg/dl. CT of the abdomen showed dirty fat stranding with increased density of the central mesentery with enlarged lymph nodes and wall thickening of the small bowel (Figure 1). Diagnostic laparoscopy revealed severe adhesion of the omentum to the small intestine and massive turbid ascites. Further analysis yielded lymphocytic ascites with a WBC count of 0.7×10^9/l, glucose 118 mg/dl, lactate dehydrogenase 787 U/l, and a serum-to-ascites albumin gradient (SAAG) of 1.6 g/dl. Acid-fast stain of the omentum specimen was strongly positive for mycobacteria, while that of the ascites specimen was negative. Anti-tuberculous therapy with rifabutin, isoniazid, ethambutol, and pyrazinamide were initiated along with antiretroviral therapy. Cultures of the patient's sputum, ascites, and omentum tissue subsequently yielded MAC. Although mild alleviation of abdominal symptoms was soon observed after his regimen was changed to clarithromycin, ethambutol, amikacin, and levofloxacin, relapse occurred several weeks later because of intolerance to the antiretroviral therapy.

Literature review

A total of five published reports of 11 cases of MAC peritonitis were identified in the literature,[5–9] and alongside the five cases diagnosed at this hospital, 16 cases of MAC peritonitis in patients with HIV infection were reviewed. Table 1 summarizes the clinical presentations of these 16 patients. The majority were male, and the median age was 36 years (range 23–50 years). Liver cirrhosis was documented in two of them. Most of the patients had CD4 counts below 50 cells/

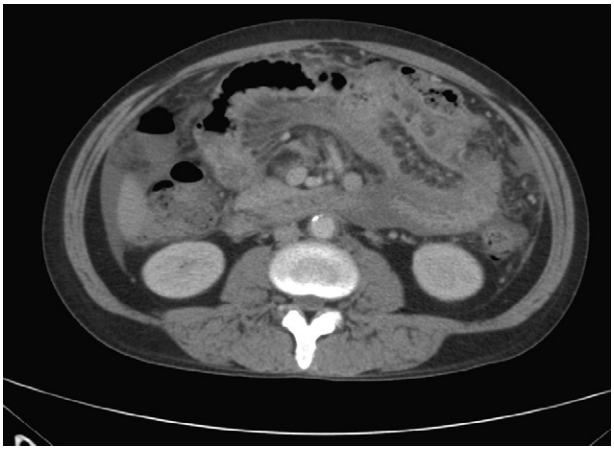

Figure 1 Computed tomography of the abdomen after administration of contrast medium showing thickened bowel wall and mesentery in an AIDS patient with *Mycobacterium avium* complex peritonitis.