CASE REPORT

RECTAL PROLAPSE ASSOCIATED WITH CYTOMEGALOVIRUS PSEUDOMEMBRANOUS COLITIS IN A CHILD INFECTED BY HUMAN IMMUNODEFICIENCY VIRUS

Chitsanu Pancharoen¹, Sasithorn Likitnukul¹, Voranush Chongsrisawat¹, Boosba Vivatvekin¹, Parvapan Bhattarakosol², Pongsepeera Suwangool³ and Usa Thisyakorn¹

¹Department of Pediatrics, ²Department of Microbiology, ³Department of Pathology, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand

Abstract. We report a newly recognized presentation of cytomegalovirus (CMV) enterocolitis in a 4year-old girl with newly diagnosed HIV disease who presented with rectal prolapse. Gross findings showed multiple whitish punctate lesions. An endoscopic examination revealed multiple shallow ulcers and pseudomembranes along the colon. A biopsy from colonic tissues demonstrated CMV-like inclusion bodies. A direct immunofluorescence assay using specific CMV monoclonal antibody was positive for CMV-infected cells in specimens from the rectal smear.

The gastrointestinal manifestations of HIVinfected children are related to opportunistic infections, lymphoproliferative disease and cancer. Gastritis and enteritis are commonly caused by cytomegalovirus (CMV), a potentially virulent pathogen resulting in necrosis, obstruction, hemorrhage and perforation, with significant morbidity and mortality (Haller and Cohen, 1994). However, rectal prolapse, as a presenting feature of CMV enterocolitis has never been mentioned. We herein report a case of a 4-year-old HIV-infected girl who presented with CMV enterocolitis, causing pseudomembranes and rectal prolapse.

A 4-year-old previously healthy Thai girl was admitted to King Chulalongkorn Memorial Hospital, Bangkok, Thailand, on January 15, 1997 because of chronic bloody diarrhea and rectal prolapse. Physical examination revealed a chronically ill patient with cervical lymphadenopathy, hepatomegaly, generalized distension and tenderness of the abdomen. A large-sized rectal prolapse, 10 centimeters in diameter, with multiple whitish punctate lesions and serous discharge was seen (Fig 1). Stool culture was negative for enteric pathogens. Culture obtained from the rectal lesions grew Enterococcus fecalis. The abdominal roentgenographic examination revealed thickening of the bowel wall at the left side of the abdomen. An endoscopic examination showed multiple shallow ulcers with mucus and pseudomembranes covering the mucosa, and a biopsy was performed. The tissue pathology disclosed colonic mucosa covered with mucin, fibrin, red blood cells and polymorphonuclear cells, and few glandular epithelial cells containing CMV-like inclusion bodies (Fig 2). The antibody to human immunodeficiency virus (HIV) was positive. The CD4+ lymphocyte count was 64 cells/mm³ and 8%. Co-existing CMV retinitis was diagnosed by an ophthalmologist. The diagnosis of CMV colitis was confirmed by positive CMV-infected cells in specimens from the rectal smear, using a direct immunofluorescence assay and a specific CMV monoclonal antibody. Immunoglobulin G (IgG) and immunoglobulin M (IgM) for CMV were 1:800 and negative, respectively, and 1:400 and negative, respectively, in the second specimen. Isospora belli and Cryptosporidium parvum oocysts were recovered from wet preparation of stools and stools stained by modified Kinyoun acid-fast technique, respectively. Entamoeba histolytica titer was negative and the titer was 1:256 in the second blood specimen.

Antimicrobial therapy included four antibiotics (ceftriaxone, metronidazole, cotrimoxazole,

Correspondence: Dr Chitsanu Pancharoen, Department of Pediatrics, King Chulalongkorn Memorial Hospital, Rama IV Road, Bangkok 10330, Thailand. Tel/Fax: 66 (0) 2256-4930 E-mail: chitsanu.p@chula.ac.th



Fig 1– The prolapsed rectum showed diffused ulceration with mucosal friability and plaque-like pseudomembranes.



Fig 2–Histology of colonic tissue showing inflammation with intranuclear inclusion body in the glandular epithelial cells (arrow) (H & E x 400).

azithromycin), an antiviral agent (ganciclovir) and antiretroviral agents (zidovudine, didanosine). The prolapsed rectum was reducible after a few days of ganciclovir therapy. Repeated endoscopic examination with biopsy done after three weeks of ganciclovir usage demonstrated multiple aphthuslike ulcers around the bowel wall and microscopic examination showed characteristic CMV intranuclear inclusion bodies in glandular epithelial cells. The patient was discharged after completing five weeks of ganciclovir treatment without a longterm prophylaxis, with a total of seven weeks of hospitalization. Unfortunately, the patient was readmitted twice and died of possible septic shock in the last admission, on September 15, 1997.

CMV seems to be a common opportunistic pathogen in immunodeficient patients, especially in HIV-infected persons with severely immunologic suppression (Frenkel *et al*, 1990). Although CMV disease is less frequent among children, this infection still contributes significantly to morbidity and mortality among this population (Kitchen *et al*, 1997). Presentations of CMV enterocolitis include chronic diarrhea, bleeding, obstruction and perforation of the intestines (McLoughlin *et al*, 1987; Haller and Cohen, 1994; Mustafa, 1994; Kitchen *et al*, 1997). Even though HIV/AIDS is epidemic in Thailand, there have been few reports concerning CMV enterocolitis in HIV-infected children (Sirisanthana, 1995; Chiraguna and Ruangvirayudh, 1997). Our patient presented with rectal prolapse, which may be due to CMV itself, severe and prolonged course of diarrhea, malnutrition and other co-existing OIs.

Diagnosis of CMV enterocolitis includes pathologic findings *ie* pseudomembranes and inclusion bodies in colonic tissues, polymerase chain reaction (PCR) and viral isolation (Mustafa, 1994; Hanshaw, 1995). In our patient, besides the pathologic findings, the positive test using direct immunofluorescence assay and demonstration of CMV retinitis helped confirm the diagnosis of CMV enterocolitis.

In conclusion, CMV enterocolitis should be considered as a cause of pseudomembranes or rectal prolapse in HIV-infected patients.

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