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# Multiple Small Bowel Perforations Secondary to Cytomegalovirus in a Patient with Acquired Immunodeficiency Syndrome

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*Cytomegalovirus gastroenteritis can be a life-threatening infection in patients with the acquired immunodeficiency syndrome. Although gastrointestinal ulcerations from cytomegalovirus have been widely reported, our patient is only the second case reported with actual perforations in the small bowel. (Henry Ford Hosp Med J 1987;35:17-9)*

Cytomegalovirus (CMV) is an important source of infectious morbidity and mortality in immunosuppressed patients. CMV has been a recurrent problem in patients with organ transplantations (1) or the acquired immunodeficiency syndrome (AIDS) (2). CMV is known to cause ulceration and bleeding in the esophagus, stomach, small bowel, and colon (3-6). A case of small bowel perforation secondary to CMV in an AIDS patient was reported in 1984 (7). We are reporting the second AIDS case with small bowel perforation caused by CMV.

## Case Report

A 28-year-old homosexual man with a four-year history of lymphadenopathy developed AIDS in December 1982. The diagnosis was based on the presence of *Pneumocystis carinii* pneumonia and a low T-lymphocyte helper/suppressor cell ratio of 0.23. CMV complement fixation antibody titer was low-normal at 1:8. Several stool examinations were negative for bacterial and parasitic pathogens. Urine and blood cultures for CMV were negative. Treatment with sulfamethoxazole-trimethoprim resolved his pneumocystosis, but diarrhea persisted.

During the next five months the patient continued to have low-grade fevers, progressive weight loss, abdominal pain, and five to seven diarrheal stools per day. Flexible sigmoidoscopy was normal. In May 1983, CMV retinitis developed, and he went blind in the right eye.

On admission in June 1983, our patient was very cachectic, weighing only 118 lbs—a 67 lb weight loss over eight months. His abdomen was markedly distended, tymphanitic, and tender. A trial of cimetidine did not relieve his diarrhea. Stool cultures on one occasion grew *Aeromonas hydrophila*, but were negative on multiple repeated cultures. On his eighth hospital day, he developed an upper gastrointestinal hemorrhage which resolved. Endoscopy with biopsies revealed acute gastritis and chronic duodenitis. The patient subsequently developed tachycardia, hypotension, and pneumoperitoneum. At exploratory laparotomy, multiple well-circumscribed perforations were present in the ileum and distal jejunum (Figs 1 and 2); the colon was grossly normal. Three feet of ileum and jejunum were resected with construction of a proximal jejunostomy and terminal ileum mucous fistula. Microscopic examination of the surgical specimen showed multiple well-defined lesions of the small bowel with punctate perforations and inclusions of CMV (Fig

3). Postoperatively, he could never be weaned from the ventilator. Bronchoscopy showed CMV, but no *Pneumocystis* in the lungs. In addition to pulmonary insufficiency, our patient developed progressive renal and liver failure and died three weeks postoperatively.

Necropsy revealed bilateral pneumonitis, an enlarged congested liver, splenomegaly with infarcts, and renal changes consistent with acute renal failure. Diffuse peritonitis and focal intraabdominal organizing abscesses were present. The distal colon had patchy ulcers with one sealed perforation. The remaining small bowel was grossly normal.

Generalized CMV infection was present on microscopic examination. CMV inclusion bodies were found in the larynx, trachea, heart, lungs, entire gastrointestinal tract, pancreas, thyroid, adrenal glands, and paratracheal lymph nodes. The liver was enlarged with chronic congestion and cholestasis with acute centrilobular hemorrhagic necrosis. The brain had acute anemic infarcts of the right and left gyri along with glial nodules and astrocystosis, Alzheimer's type II. Postmortem viral lung cultures grew CMV.

## Discussion

Although his CMV serological titers were not elevated, this patient suffered from extensive CMV infection including pneumonitis, retinitis, and complete gastrointestinal tract involvement from the esophagus to the rectum with subsequent small bowel perforation.

Microscopic examination of the small bowel revealed heavy infiltrate of histiocytes and other mononuclear cells adjacent to the point of perforation. The cells of this infiltrate as well as vascular endothelium were enlarged and contained intranuclear and intracytoplasmic inclusions consistent with CMV infection.

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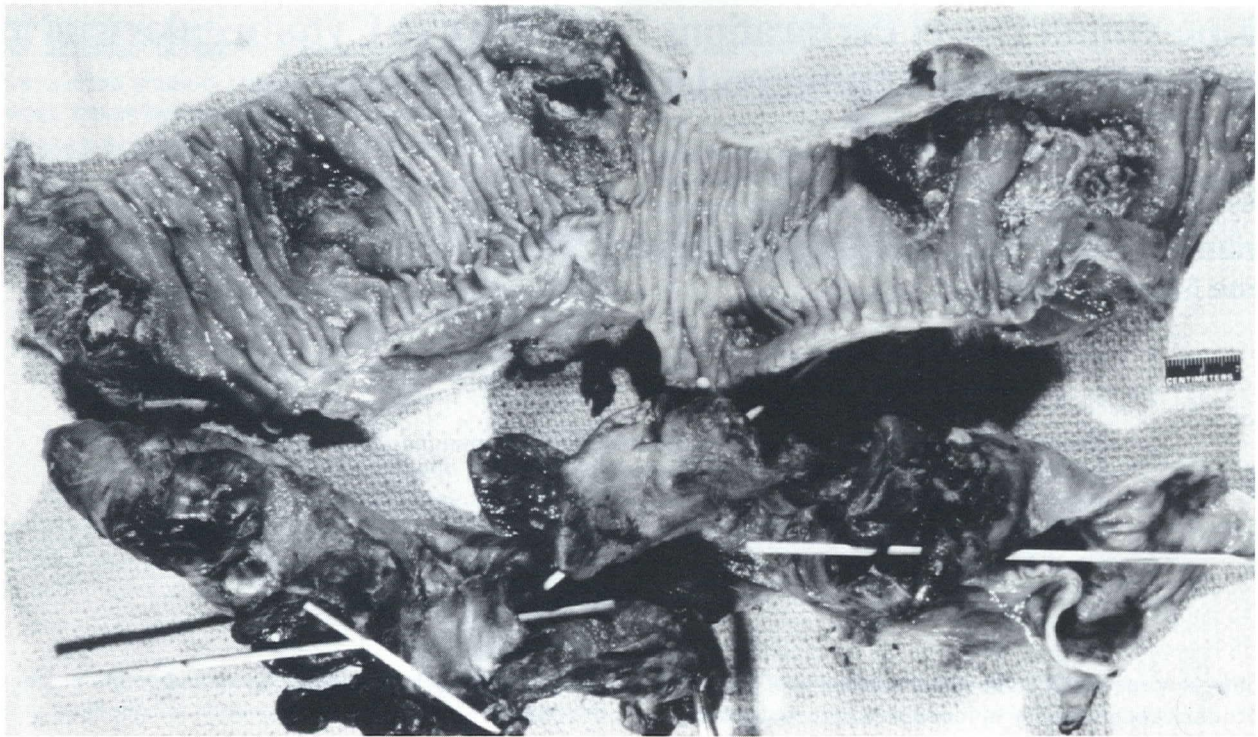


Fig 1—Excised segment of the small bowel displays multiple areas of focal necrosis and perforations.



Fig 2—Closer view of an area of developing necrosis in the small bowel.

The blood vessels were partially or completely obstructed as a result of CMV vasculitis, either from enlarged endothelial cells or from thrombosis.

In areas of the bowel distant from the perforations, the vasculitis and cellular infiltrate were confined mainly to the mucosal villi, resulting in edema and necrosis of villi. Progression of the vasculitis subsequently to the submucosa and muscularis apparently led to further necrosis and perforation.

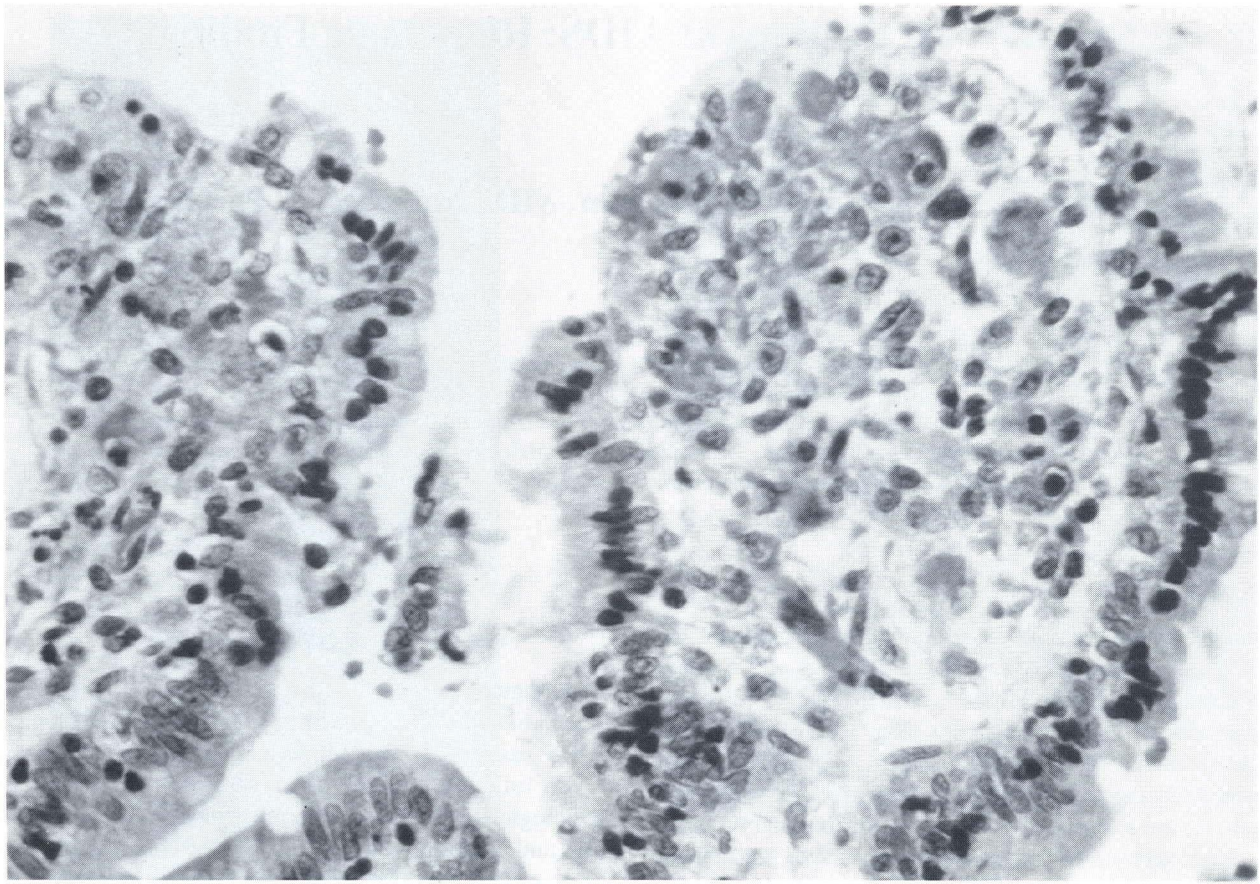
Although we were unable to definitively correlate gastrointestinal CMV infection with our patient's diarrhea and weight

loss, CMV must be suspected when treating AIDS patients with diarrhea of unknown etiology (8). Prior to the case of terminal ileum perforation in an AIDS patient as described by Frank and Raicht (5), all reported gastrointestinal perforations secondary to CMV had been limited to the large bowel. Clinicians treating AIDS patients should recognize that small bowel perforations may accompany aggressive CMV infections and that serologic tests for CMV are often not helpful.

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*Fig 3—Small bowel mucosa shows loss of epithelium and massive infiltrate of histiocytes. Histiocytes and endothelial cells are enlarged and display intranuclear and intracytoplasmic inclusions (hematoxylin-eosin stain) (X320).*