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A Submucosal Antral Mass Caused by Cytomegalovirus Infection in a Patient with Acquired Immunodeficiency Syndrome

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A 29-yr-old homosexual man with acquired immunodeficiency syndrome presented with watery diarrhea and fever. Upper gastrointestinal endoscopy was performed to obtain duodenal aspirates and biopsies. A 4-cm submucosal mass in the gastric antrum was identified. Subsequent abdominal CT scan confirmed the presence of this antral mass. An attempt at CT guided needle biopsy was nondiagnostic. Because the mass possibly represented a Kaposi's sarcoma or lymphoma, exploratory laparotomy and open biopsy was performed. Examination of the biopsy specimen showed inflammatory debris with multiple intranuclear cytomegalovirus inclusions. This report describes a case of a submucosal antral mass caused by localized cytomegalovirus infection in a patient with acquired immunodeficiency syndrome.

INTRODUCTION

Cytomegalovirus (CMV) is an infectious agent that is particularly widespread in the homosexual community (1, 2). While this agent usually causes little morbidity in normal immunological hosts (3), its virulence is quite different in immunosuppressed patients. Severe CMV infection of the esophagus, stomach, duodenum, and colon has been described in patients with acquired immunodeficiency syndrome (AIDS) (4, 5). An immunocompromised host with apparent CMV involvement of the biliary tree and gallbladder has also been reported (6). This report describes an endoscopically diagnosed submucosal antral mass in a patient with AIDS. Open surgical biopsy showed this to be a collection of inflammatory cells, edema, and numerous stromal and epithelial cells with CMV inclusion bodies.

CASE REPORT

A 29-yr-old homosexual man was well until November 1983 when he developed right upper quadrant abdominal pain and a 10–lb weight loss. His only laboratory abnormalities were a mildly elevated SGOT and a positive VDRL and FTA. The remainder of his evaluation consisting of a chest x-ray, upper gastrointestinal series, liver–spleen scan, intravenous pyelogram, ultrasound of the biliary tree and liver, and chest CT scan was entirely normal. He received oral tetracycline for treatment of syphilis. In January 1984, he was admitted to a community hospital for progressive weight loss and persistent right upper quadrant pain. Laboratory studies included an elevated SGOT and alkaline phosphatase. Fiberoptic esophagogastroduodenoscopy (EGD) showed diffuse gastritis. Biopsy was nonspecific. Endoscopic retrograde cholangio-pancreatography (ERCP) showed intra– and extrahepatic biliary ductal irregularities. Abdominal CT scan was normal. Laparotomy and cholecystectomy were performed for what was described as an inflamed gallbladder. Examination of the gallbladder specimen showed severe chronic cholecystitis with CMV infection. Intraoperative liver biopsy also showed mild CMV hepatitis. Symptoms improved postoperatively, and the patient was discharged.

In February 1984, he again presented with right upper quadrant pain and weight loss. Evaluation consisted of a normal intravenous pyelogram, abdominal CT, bone scan, HIDA scan, and cervical lymph node biopsy. Flow cytometry showed reversal of the normal helper-suppressor T cell relationship with a T4/T8 ratio of 0.36 consistent with AIDS or other viral infection. The diagnosis of AIDS was made.

The patient was transferred to the University of Michigan Medical Center in March 1984. He continued to complain of right upper quadrant pain and had lost a total of 40 lb over the previous 4 months. He also noted low grade fevers. Repeat endoscopic retrograde cholangiopancreatography showed dilatation and irregularities of the bile ducts raising the question of CMV–induced cholangitis. His abdominal pain was controlled successfully with narcotic analgesics. Evaluation of his diarrhea included a normal colonoscopy with multiple biopsies showing normal histology. EGD demonstrated candida esophagitis, a normal stomach, and duodenitis with *Giardia lamblia* infestation. None of the gastrointestinal mucosal biopsies obtained at this time showed CMV inclusions. During this hospitalization, he contin-

ued to spike fevers and his chest x-ray showed a right lower lobe infiltrate. Bronchoalveolar washings showed *Pneumocystis carinii*. He was treated initially with a combination of trimethaprin/sulfamethoxazole and low dose amphotericin and later discharged on ketoconazole and trimethaprin/sulfamethoxazole.

In May 1984, he was readmitted for fever and profuse watery diarrhea. Flexible sigmoidoscopy to 40 cm showed diffuse erythema and biopsies showed mild nonspecific cryptitis. The diagnosis of *Clostridium difficile*–induced diarrhea was made after obtaining positive culture and stool toxin. He was discharged on metronidazole.

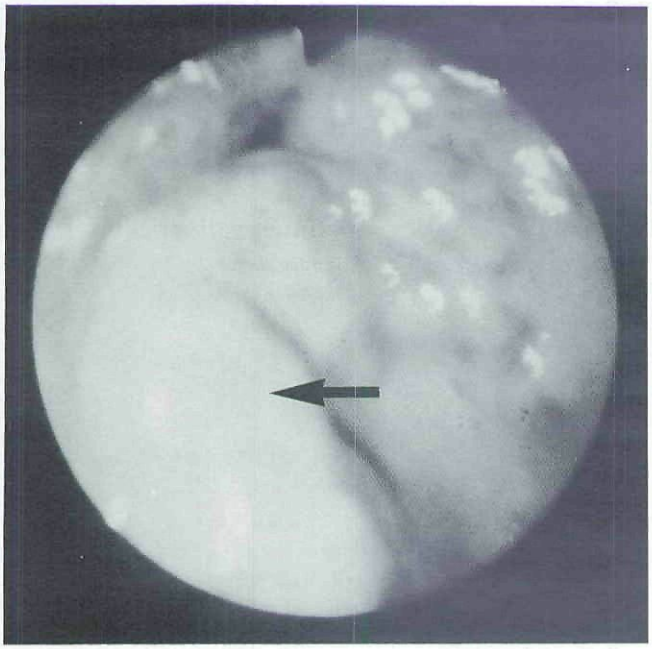

FIG. 1. Endoscopic view of submucosal mass in gastric antrum.

In June 1984, he was readmitted for persistent fever, diarrhea, and lower abdominal cramping pain. Repeat *C. difficile* culture and toxin assay was positive. He was discharged on oral vancomycin.

One month later, he was readmitted for persistent diarrhea. Repeat evaluation showed a negative *C. difficile* culture and toxin assay. At colonoscopy, the transverse colon was diffusely erythematous and granular. Colonic mucosal biopsies showed colitis with CMV inclusions. Stool ova and parasite exam showed Cryptosporidium and the investigational drug spiramycin was begun. EGD showed persistent candida esophagitis and a 4-cm antral mass along the lesser curvature of the stomach (Fig. 1). Biopsies of the gastric mucosa overlying the mass were normal. Abdominal CT scan confirmed the presence of the antral mass (Fig. 2). An attempt at CT–guided aspiration cytology was nondiagnostic. Laparotomy was performed for diagnosis. Biopsies of the peripyloric area showed only marked inflammation with viral inclusions consistent with CMV (Fig. 3).

The patient was readmitted in September 1984 with fever, cyanosis, and bilateral chest infiltrates. Bronchoscopy with washings demonstrated aspergillus, CMV, and candida. Within the first 24 h of admission, the patient developed increasing respiratory distress and required ventilatory support. Despite therapy, he developed progressive respiratory insufficiency and hypotension and died on 10/3/84.

Autopsy showed diffuse and severe infection with CMV involving lung, pancreas, biliary epithelium, colon, and the gastric submucosal mass.

DISCUSSION

The gastrointestinal tract is a frequent site of CMV

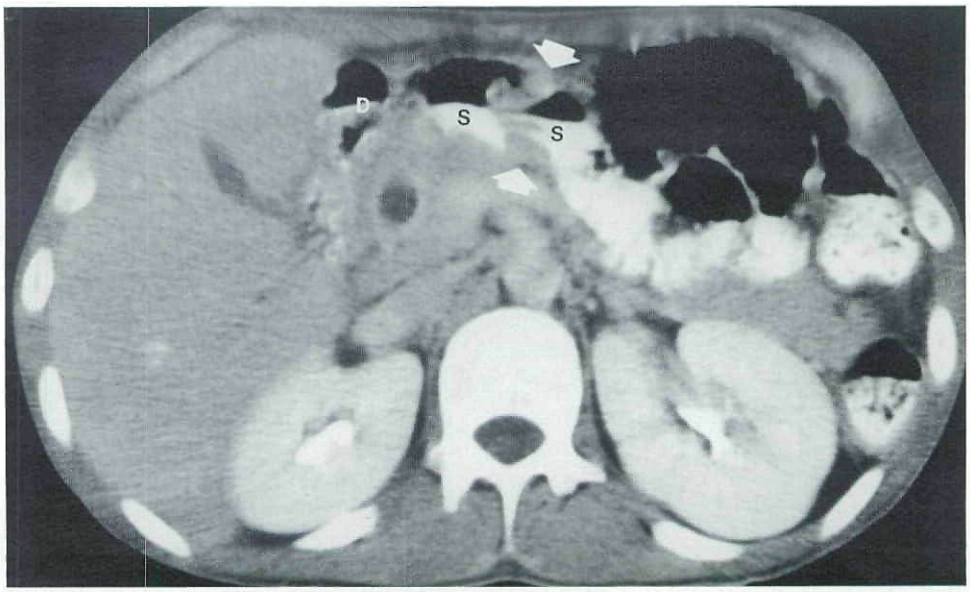

FIG. 2. Representative cut from CT scan showing antral mass (*arrows*) *S*, stomach; *D*, duodenum.

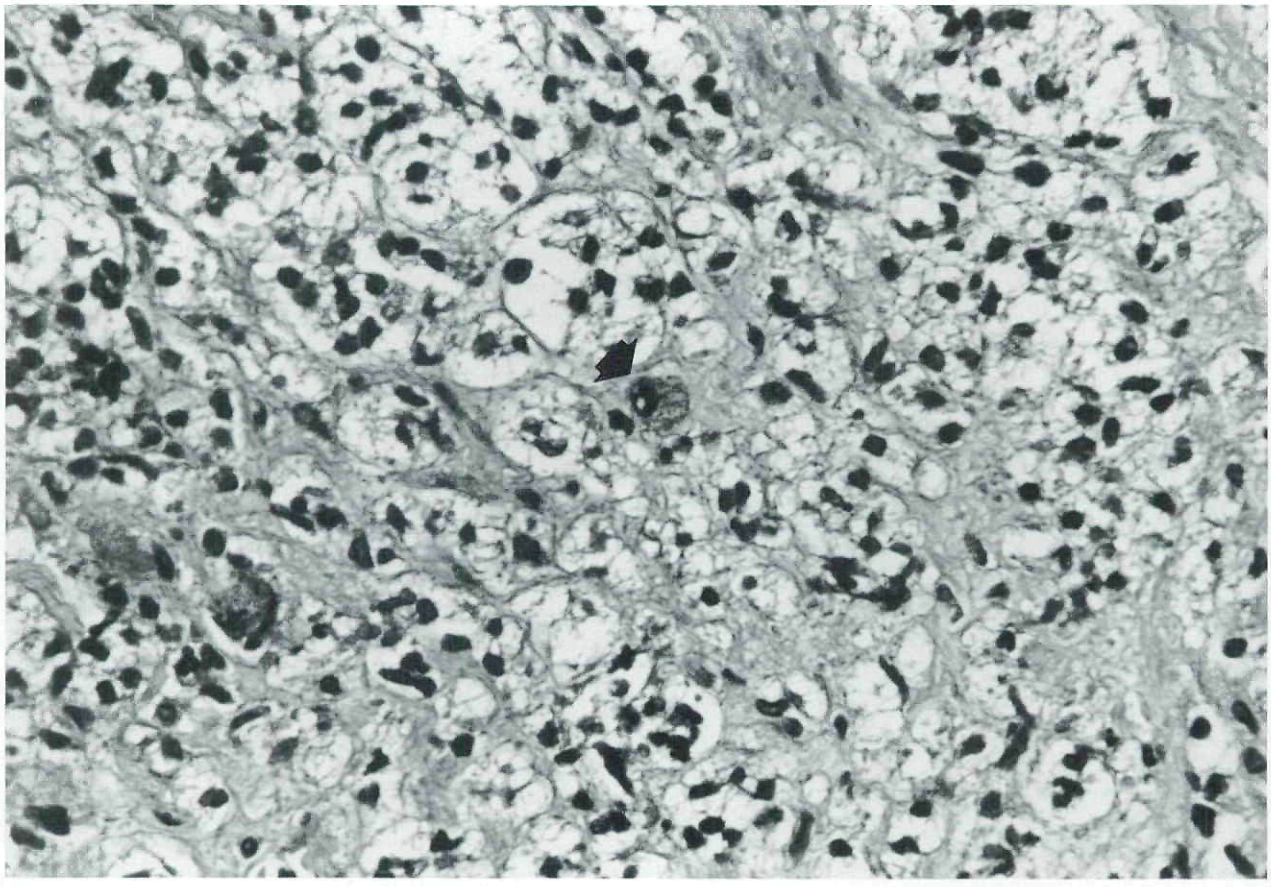

FIG. 3. Photomicrograph of tissue specimen obtained from peripyloric mass showing characteristic intranuclear inclusion bodies (*arrow*) and predominantly a mononuclear cell infiltrate.

infection. CMV infection in immunocompromised patients is known to involve esophageal (7), gastroduodenal (8, 9), colonic (10), and biliary tract mucosa (6). Gastrointestinal involvement by CMV has also been described in normal hosts, but is quite rare (11). In one autopsy study of CMV infection reported by Wong and Warner (12), evidence of CMV involvement of the gastrointestinal tract was noted in 21 of 41 patients (51%). These authors observed that the stomach was the most frequently involved portion of the gastrointestinal tract. In contrast, Franzin et al. (13) subjected immunosuppressed renal transplant recipients to fiberoptic EGD and found CMV inclusions in biopsies of duodenal bulb mucosa more frequently than in biopsies of stomach mucosa.

Gastrointestinal CMV infection may present with widely diverse symptoms and signs including profuse diarrhea, abdominal pain, weight loss, hemorrhage, hypoproteinemia resulting from protein-losing enteropathy secondary to widespread mucosal ulceration and perforation of the bowel (14–17). A localized inflammatory pseudotumor due to CMV infection in the GI tract has not been reported. Our patient exhibited many of the features of generalized CMV infection including recurrent fevers, weight loss, and protracted diarrhea. However, these symptoms may be due to other opportunistic infections in the AIDS patient. The gastric antral mass diagnosed by EGD in our patient was confirmed by CT scan of the abdomen. Endoscopic biopsies of the mucosa overlying the mass were normal. Laparotomy with biopsy of the mass lesion was performed to rule out the possibility of a treatable neoplasm. Both Kaposi's sarcoma and non–Hodgkin's lymphoma are reported in patients with AIDS (18–22). This was the rationale for aggressively pursuing a histological diagnosis of the antral mass, despite the fact that it was asymptomatic. The endoscopic appearance of Kaposi's sarcoma is often characteristic, although mucosal biopsies are only positive in one–quarter of patients (23). The frequent submucosal location of gastrointestinal lymphomas often gives neither a diagnostic endoscopic appearance or adequate tissue for histological diagnosis. A recent report of 88 homosexual men with non-Hodgkin's lymphomas described 15 cases with primary gastrointestinal sites (22).

At operation, the only abnormality noted was an area of thickening in the peripyloric region. Biopsies obtained from the site showed typical intranuclear inclusions of CMV and an inflammatory cell infiltrate consisting of lymphocytes and plasma cells. The patient

recovered uneventfully from surgery, but died 2 months later from CMV-associated pneumonitis.

Endoscopy is frequently performed for the diagnosis of opportunistic infections involving gastrointestinal mucosa in immunocompromised patients (24). CMV appears to be a common organism in these patients. It is conceivable, therefore, that more lesions of the type described in our patient may be identified in the future. Definitive diagnosis should be pursued to rule out treatable lesions. CT or ultrasound-guided fine needle aspiration cytology should be considered first, but negative results may need to be followed by exploratory laparotomy to obtain sufficient tissue for diagnosis. Currently, no effective therapy for CMV infection is available, although interferon has been tried (25). As better antiviral agents evolve, the correct diagnosis of CMV induced mass–like lesions may become increasingly important.

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REFERENCES

1. Drew WL, Mintz L, Miner RC, et al. Prevalence of cytomegalovirus infection in homosexual men. J Infect Dis 1981;143:188–92.
2. Mintz, L, Drew WL, Miner RC, et al. Cytomegalovirus infections in homosexual men. Ann Intern Med 1983;99:326–9.
3. Betts RF. Cytomegalovirus infection epidemiology and biology in adults. Semin Perinatol 1983;7:22–30.
4. Knapp AB, Horst DA, Eliopoulos G, et al. Widespread cytomegalovirus gastroenterocolitis in a patient with acquired immunodeficiency syndrome. Gastroenterology 1983;85:1399–402.
5. Gertler SL, Pressman J, Price P, et al. Gastrointestinal cytomegalovirus infection in a homosexual man with severe acquired immunodeficiency syndrome. Gastroenterology 1983;85:1403–6.
6. Galloway PG. Widespread cytomegalovirus infection involving the gastrointestinal tract, biliary tree, and gallbladder in an immunocompromised patient. Gastroenterology 1984;87:1407.
7. Onge GS, Bezahler GS. Giant esophageal ulcer associated with cytomegalovirus. Gastroenterology 1982;83:127–30.
8. Franzin G, Muolo A, Griminelli T. Cytomegalovirus inclusions in the gastroduodenal mucosa of patients after renal transplantation. Gut 1981;22:698–701.
9. Ayulo M, Aisner SC, Margolis K, et al. Cytomegalovirus-associated gastritis in a compromised host. JAMA 1980;243:1364.
10. Guttman D, Raymond A, Gelb A, et al. Virus-associated colitis in homosexual men: two case reports. Am J Gastroenterol 1983;78:167–9.
11. Rustgi VK, Sarcher RA, O'Brien P, et al. Fatal disseminated cytomegalovirus infection in an apparently normal adult. Arch Intern Med 1983;143:372–3.
12. Wong T, Warner NE. Cytomegalic inclusion disease in adults. Arch Pathol 1962;74:403–22.
13. Franzin G, Muole A, Griminelli T. Cytomegalovirus inclusions in the gastroduodenal mucosa of patients after renal transplantation. Gut 1981;22:698–701.
14. Fauci AD. Acquired immunodeficiency syndrome: epidemiologic, clinical, immunologic, and therapeutic considerations. Ann Intern Med 1984;100:92–106.
15. Diethelm AG, Gore I, Ch'ien LT, et al. Gastrointestinal hemorrhage secondary to cytomegalovirus after renal transplantation: case report and review of the problem. Am J Surg 1976;131:371–4.
16. Underwood JCE, Corbett CL. Persistent diarrhea and hypoalbuminamia associated with cytomegalovirus enteritis. Br Med J 1978;1:1029–30.
17. Goodman ZD, Boitnett JK, Yardley JH. Perforation of the colon associated with cytomegalovirus infection. Dig Dis Sci 1979;24:376–80.
18. Jaffe HW, Choi K, Thomas PA, et al. National case-control study of Kaposi's sarcoma and pneumocystis carinii pneumonia in homosexual men. Ann Intern Med 1983;99:145–58.
19. Ziegler JL, Drew WL, Miner RC, et al. Outbreak of Burkitt's-like lymphoma in homosexual men. Lancet 1982;1:631–3.
20. Levine AM, Meyer PR, Begandy MK, et al. Development of B-cell lymphoma in homosexual men. Ann Intern Med 1984;100:7–13.
21. Diffuse, undifferentiated non-hodgkins lymphoma among homosexual males-United States. MMWR 1982;31:277–9.
22. Ziegler JL, Beckstead JA, Volberding PA, et al. Non-Hodgkin's lymphoma in 90 homosexual men. Relation to generalized lymphadenopathy and the acquired immunodeficiency syndrome. N Engl J Med 1984;311:565–70.
23. Friedman SL, Wright TL, Altman DF. Gastrointestinal Kaposis sarcoma in patients with acquired immunodeficiency syndrome. Gastroenterology 1985;89:102–8.
24. Allen JI, Silvis SE, Sumner HW, et al. Cytomegalovirus inclusion disease diagnosed endoscopically. Dig Dis Sci 1981;26:133–5.
25. Pahwa S, Kirkpatrick D, Ching C, et al. Persistent cytomegalovirus infection associated with profound immunodeficiency and treatment with interferon. Clin Immun Immunopathol 1983;28:77–89.