CASE REPORT

Cytomegalovirus-associated Acute Gastric Mucosal Lesion in an Immunocompetent Host

Takashi Himoto¹, Fuminori Goda¹, Hiroyuki Okuyama¹, Takeaki Kono¹, Ayumu Yamagami¹, Michio Inukai², Hisashi Masugata¹, Mitsuyoshi Kobayashi³, Hideyuki Inoue³, Fumihiko Kinekawa², Tsutomu Masaki², Reiji Haba³, Eiji Ohashi⁴, Toshihiro Mori⁴ and Shoichi Senda¹

Abstract

Involvement of the gastrointestinal tract in cytomegalovirus (CMV) infection is commonly observed in immunocompromised hosts. We encountered an immunocompetent patient with CMV associated acute gastric mucosal lesion (AGML). The emergence of inclusion bodies characteristic of CMV infection in the specimens obtained from the patient’s gastric ulcers was helpful in identifying the cause of AGML. The patient recovered without the administration of antiviral drugs. This case illustrates that CMV infection can be one of the causative agents that trigger AGML even in immunocompetent hosts, and that gastric biopsies are extremely useful for ascertaining the etiology of AGML.

Key words: cytomegalovirus, acute gastric mucosal lesion, immunocompetent host

(Intern Med 48: 1521-1524, 2009)

DOI: 10.2169/internalmedicine.48.2308

Introduction

Cytomegalovirus (CMV) infection is usually asymptomatic in immunocompetent hosts, and most susceptible individuals are infected with CMV until adolescence (1). However, the infection occasionally causes a mononucleosis-like syndrome with the involvement of multiple organs in young adults who are immunocompetent (2, 3). Gastrointestinal (GI) involvement caused by CMV infection is commonly observed among patients with acquired immunodeficiency syndrome (AIDS), those receiving immunosuppressive treatments after organ transplantsations, those receiving chemotherapy for the treatment of a malignant tumor and those receiving frequent blood transfusions.

In this case report, we describe an immunocompetent adult with acute gastric mucosal lesion (AGML) that derived from CMV infection.

Case Report

A 31-year-old Japanese man who was previously healthy consulted a physician with a one-month history of low-grade fever and epigastric pain. These symptoms suddenly occurred. His previous medical history was an appendectomy at the age of 12. He had no history of blood transfusion. He neither took medications including non-steroidal anti-inflammatory drugs (NSAIDs), nor suffered from the mental stress. He is not an alcohol abuser. He underwent blood examinations and upper gastrointestinal endoscopic study at the clinic. He was referred to the Hospital of Kagawa University School of Medicine for detailed examinations and treatments for liver dysfunction and gastric lesions.

Physical examination on admission revealed no conjunctival anemia and no jaundice. He had no abnormal cardiopulmonary findings. The abdomen was soft and flat, although tenderness was present in the epigastrium. Hepatospleno-
megaly was not obvious upon palpation. Neither ascites nor edema was found. No abnormal neurologic findings were observed. Superficial lymph nodes including cervical, axillary and inguinal lymph nodes were not enlarged.

Laboratory data on admission were as follows: peripheral blood revealed leukocytosis with atypical lymphocytes (white blood cells: 11,700/μL, atypical lymphocytes: 10.5%). Blood biochemistry analyses exhibited liver dysfunction, indicating elevated aspartate aminotransferase (AST) 117 IU/L, alanine aminotransferase (ALT) 231 IU/L and lactate dehydrogenase (LDH) 612 U/L (normal: 100 to 220 U/L), gamma-glutamyl transpeptidase (γ-GTP) 114 U/L (normal: less than 60 U/L) and alkaline phosphatase (ALP) 336 U/L (normal: 100 to 340 U/L). Other abnormal test results included elevated C-reactive protein (CRP) 1.4 mg/dL (normal: less than 0.2 g/dL). Serological tests indicated no recent infection with hepatitis B virus (HBV), hepatitis C virus (HCV), human immunodeficiency virus (HIV), or Epstein-Bar virus (EBV). He was diagnosed as acute cytomegalovirus (CMV) infection on the basis of seropositivity for the IgM class of antibody against CMV and the detection of CMV antigenemia (pp65) using C10/C11 monoclonal antibodies (4). Bone marrow aspiration showed normocellular bone marrow.

Upper GI endoscopy showed a mixture of multiple shallow ulcers and mucosal edema extending from the lower body to the prepyloric antrum of the stomach (Fig. 1). Colonoscopy showed no remarkable findings in his colon and rectum. Computed tomography (CT) scan obtained on admission revealed the diffuse wall thickening of the stomach and mild splenomegaly (Fig. 2). Multiple small-sized cysts were also scattered throughout the liver. Gastric biopsy specimens from the ulcers revealed intranuclear inclusion bodies, characteristic of CMV infection, in the stroma of the gastric mucosa (Fig. 3a). The CMV antigens corresponding to the intranuclear inclusion bodies were also detected by the immunoperoxidase method using monoclonal CMV antibody (Fig. 3b). The emergence of bacillus corresponding to Helicobacter pylori was not confirmed in the specimens with hematoxylin and eosin staining.

In the present patient, the epigastric pain disappeared
CMV infection spontaneously affects multiple organs including the heart, lung, liver, GI tracts, skin and central nervous system in immunocompetent hosts (2, 3). CMV can cause ulcerations anywhere in the GI tract ranging from the esophagus to the rectum (1-3, 9, 11-13). The most susceptible organs in the GI tract are the stomach and colon. CMV infection is occasionally associated with Ménétrier’s disease as well as ulcerations in the GI tract (14, 15).

As a possible mechanism of ulceration in the GI tract, ischemic mucosal injury that derives from CMV infection into the endothelial cells of small vessels has been speculated (13, 16). Inclusion bodies may be also observed within the endothelial cells of small vessels in ulcer tissues (16). In such cases, the term “cytomegalic vasculitis” has been proposed. In CMV-associated Ménétrier’s disease, the involvement of endothelial cells by CMV infection may cause an increase in the permeability of the blood vessels, and eventually lead to protein-losing gastropathy (17).

We supposed that the involvement of the liver as well as the stomach was caused by CMV infection in this case, because the patient also experienced liver dysfunction and mild splenomegaly, which suggest acute viral hepatitis. Unfortunately, we could not confirm the involvement of the liver pathologically, since liver biopsy was not performed.

Several reports have previously documented CMV-associated AGML in immunocompetent hosts. Most of these patients have inclusion bodies in the specimens obtained from the gastric ulcers (18-22). However, it is sometimes difficult to detect them in specimens from immunocompetent hosts. In those cases, molecular technology including in situ hybridization (23) and/or the polymerase chain reaction (PCR) procedure (24) is necessary to prove CMV infection (21, 22).

It is of interest that most of the patients with CMV-associated AGML who were immunocompetent individuals did not require an antiviral treatment such as ganciclovir (18-22), because CMV-associated ulcers in the GI tracts are often self limited (11, 25). Only supportive therapy is required in most of the patients with CMV-associated gastric ulcers in immunocompetent hosts. The present patient was treated with PPI alone and recovered soon. On the contrary, the administration of antiviral drugs is usually essential in immunocompromised hosts experiencing CMV infection with the involvement of the GI tract due to the poor prognosis. A recent report disclosed that spontaneous remission of CMV-induced colitis occurred in most young immunocompetent individuals, who were less than 55 years old (26).

In summary, CMV-associated AGML is uncommonly observed in immunocompetent hosts. Gastric biopsies are quite helpful for identifying the cause of AGML. CMV is considered to be one of the entities that cause AGML.

References


