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CASE REPORT

Eosinophilic gastroenteritis with cytomegalovirus infection in an immunocompetent child

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Abstract

A 3-year-old boy developed transient protein-losing gastroenteropathy associated with cytomegalovirus (CMV) infection. Both IgG and IgM antibodies to CMV were positive in a serologic blood test. Upper gastrointestinal endoscopy showed multiple erosions throughout the body of the stomach, without enlarged gastric folds. Histological examination of the biopsy specimens indicated eosinophilic gastroenteritis and CMV infection. The patient had complete resolution without specific therapy for CMV in four weeks. An allergic reaction as well as CMV infection played important roles in the pathogenesis of this case.

Key words: Eosinophilic gastroenteritis; Cytomegalovirus; Protein-losing gastroenteropathy; Allergy; Menetrier's disease

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INTRODUCTION

Eosinophilic gastroenteritis (EG) is a chronic inflammatory disorder of the gastrointestinal (GI) tract characterized by the infiltration of eosinophils. Klein *et al*[1] proposed classification of EG based on the depth of eosinophilic infiltration; mucosal, muscular and serosal. The mucosal disease tends to present with protein-losing gastroenteropathy (PLG). As for pathogenesis, an allergic mechanism has been implicated in many cases[2,3]. A viral etiology has also been suggested in several cases[4]. In this report, we present a case of PLG caused by allergy- and cytomegalovirus (CMV)-related EG in a 3-year-old boy.

CASE REPORT

A 3-year-old boy was referred to our hospital for evaluation of hypoproteinemia and edema. He had experienced allergic reactions to milk, soybeans, shrimp and pork. His initial symptoms of diarrhea and edematous swelling of the eyelids developed during one week before admission. Physical examination revealed periorbital and pretibial edema. His abdomen was slight distended. The liver and spleen were not palpable. Abdominal ultrasound detected a small amount of ascites and mild swelling of the mesenteric lymph nodes. On laboratory examination, the white blood cell count was 11 300/mm^3, with 24% eosinophils. Total serum protein concentration was 3.2 g/dL, with an albumin concentration of 1.8 g/dL. Serum IgE level was within normal limits (24 IU/mL). RAST for specific IgE antibodies and skin prick test failed to reveal reactions to common food antigens. The results of a serological test for CMV were positive for IgM and IgG. Stool samples showed normal flora on culture and the absence of blood, *H pylori* antigen, ova and parasites. Enteric protein loss was confirmed by a fecal α_1-antitrypsin clearance of 68.8 mg/d (normal, < 20 mg/d). We excluded urinary protein loss as a cause of the hypoalbuminemia. Upper GI endoscopy showed multiple erosions with abundant whitish mucus throughout the gastric body. Enlarged gastric folds were not observed. The esophagus exhibited no remarkable change and the duodenum appeared mildly reddened. A sigmoidscopy showed mucosa with a normal vascular pattern and a mild lympho-nodular appearance. Histological examination of the gastric mucosa revealed neutrophilic and eosinophilic infiltrates with focal clustering of eosinophils in the lamina propria and regenerative epithelial changes. We also observed a small number of cells with intranuclear and intracytoplasmic inclusions that were immunohistochemically positive for CMV. *H pylori* was not detected. Duodenal and colonal biopsies showed increased numbers of eosinophils in the lamina propria. Histological diagnosis was compatible with EG.

The patient was treated with transfusions of albumin, oral famotidine, oxatomide and cromoglicic acid, but not corticosteroid or a specific therapy for CMV. There was a gradual recovery from edema with a concomitant increase in the serum protein level to 6.8 g/dL and a normalization of the eosinophil count over the next four

weeks. The endoscopic abnormalities also subsided after four weeks of hospitalization. Histological assessment of the mucosal biopsy specimens demonstrated clearing of the eosinophilic infiltration. He had no recurrences in the subsequent 36 mo.

DISCUSSION

CMV infection in the GI tract is unusual in an immunocompetent person. Primary infection with CMV is generally asymptomatic and usually remains latent for life. If host immune defenses are impaired, latent CMV may be activated and produce symptoms of overt disease. GI tract infection with CMV usually occurs in immunocompromised patients by activation of the latently infectious virus. On the other hand, there are several reports of CMV infection in normal healthy persons and the important role of CMV in the acute gastrointestinal disease has been emphasized[5]. In our case, evidence of gastric CMV involvement was shown by histological findings of characteristic inclusion bodies and by the immunohistochemical detection of viral antigens. It was likely that mucosal disruption due to allergic mechanism facilitated the CMV infection, which then led to further injury. As well, CMV infections in the GI tract might be locally cytopathogenic, possibly allowing mucosal penetration of allergens that then stimulated the allergic reaction.

Allergy has been suggested as the cause of transient PLG in children[6]. However, the target allergens in our patient were unclear; serum RAST and skin prick tests against common food allergens were all negative. Lin *et al*[7] suggested that a localized IgE-mediated response could cause the gastrointestinal symptoms seen in skin-prick test-negative and serum IgE antibody-negative patients with suspected food allergy.

EG may present with a wide variety of clinical and endoscopic findings. However, the multiple erosions throughout the body of the stomach seen in our patient are unusual for EG; commonly endoscopic findings of EG are edema, erythema and erosions in the antrum of the stomach. Only one case of EG with similar endoscopic features has been reported[8]. Additionally, the clinical course of our case was unusually benign and short for EG; patients with EG usually have a prolonged course of relapse and remission and for control may require therapy with steroids.

Menetrier's disease is another common cause of PLG. Allergic phenomena and CMV infection have been implicated in pediatric cases[6,9], and the course of the disease in children, unlike that in adults, is usually benign and short[9]. EG and Menetrier's disease exhibit several common features in childhood, leading to the assumption that they may represent a continuum of gastroenteropathy associated with allergic mechanism.

Although our patient remains well, he requires long-term follow-up since the natural history of this condition remains unclear. Kristopaitis *et al*[10] reported a case of EG in a 24-year-old female, who had experienced infantile EG, that recurred after a long period of dormancy.

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