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Case Report

Gastric outlet obstruction associated with cytomegalovirus infection: A complex case report

Jun Zhu[1#]; Lucheng Zheng[1]; Xiaoping Chen[1]; Liang Wu[1]; Yusheng Huang[1]; Chunzhi Yuan[1]; Hai Hu[2]; Yichong Wang[1]*

[1]Department of General Surgery, The Southern Theater Air Force Hospital, Guangzhou, China.

[2]Department of Pathology, The Southern Theater Air Force Hospital, Guangzhou, China.

[#]These authors have been equally contributed to this article.

***Corresponding Author: Yichong Wang**
Department of General Surgery, The Southern Theater Air Force Hospital, Guangzhou, China.
Email: 191646062@qq.com

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Abstract...

Cicatricial gastric outlet obstruction is an absolute indication for surgical resection in general surgery, and accurate identification of the underlying etiology is crucial for appropriate management. We report a case of a patient presenting with recurrent vomiting, cough, and dyspnea accompanied by severe malnutrition. Histopathological examination of endoscopic biopsy specimens, gross surgical specimens, and immunohistochemical staining ultimately confirmed the diagnosis of Cytomegalovirus (CMV)-associated gastritis. The patient underwent distal gastrectomy with gastrojejunostomy, followed by antiviral therapy and measures aimed at improving immune function, which led to significant clinical improvement. By analyzing the diagnostic and therapeutic approach as well as the clinical characteristics of gastric outlet obstruction caused by this relatively rare etiology, this report provides reference and insights for clinical practice.

Keywords: Cytomegalovirus; Gastric outlet obstruction; Gastritis; Distal gastrectomy; Gastrojejunostomy.

Introduction

Cicatricial gastric outlet obstruction is an absolute indication for surgical intervention. However, severe obstruction secondary to Cytomegalovirus (Cytomegalovirus, CMV) infection—manifesting as gastritis and peptic ulcer disease—is uncommon. Conventional medical therapy, including proton pump inhibitor (proton pump inhibitor, PPI)–based acid suppression and mucosal protection, is often suboptimal in CMV-associated gastritis. Timely initiation of antiviral therapy and, when indicated, prompt surgical management are therefore critical. Here, we report a case of CMV infection presenting with gastritis and gastric ulcer complicated by gastric outlet obstruction. The patient ultimately benefited from a combination of surgery and antiviral therapy, with the aim of improving clinical awareness and recognition of this entity.

Case presentation

A 79-year-old married man (Mr. Zhang, anonymized) was admitted to the Department of Respiratory Medicine of the Air Force Hospital of the Southern Theater Command on March 30, 2024, for "recurrent cough with sputum, dyspnea, and vomiting for 20 days, with worsening for 1 day," and was transferred to the Department of General Surgery on April 11, 2024.

The patient developed cough after exposure to cold on March 10, 2024. The cough was paroxysmal and occurred in bouts, producing a small amount of white mucoid sputum, accompanied by chills, fever, shortness of breath, respiratory difficulty, dizziness, myalgia, generalized arthralgia, and sore throat. He received symptomatic treatment at an outside facility, including inhaled bronchodilators, anti-infective therapy, blood transfusion, hepatoprotective agents, and gastric protection, with transient improvement.

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On the afternoon of March 29, 2024, his cough, sputum production, and dyspnea worsened again. He developed frequent vomiting of gastric contents, upper abdominal pain, dizziness, and peripheral edema. Emergency laboratory testing showed a white blood cell count of 25×10^9/L and hemoglobin of 52 g/L. Chest computed tomography (Computed Tomography, CT) demonstrated bilateral pneumonia. He was treated with intravenous cefoperazone–sulbactam and transfusion of packed red blood cells; however, he continued to experience recurrent dyspnea, epigastric pain, and vomiting. He was therefore admitted to the respiratory ward on March 30, 2024.

On admission, laboratory investigations revealed leukocytosis with neutrophilia and hemoglobin of 54 g/L. Stool testing was positive for occult blood (+). No abnormalities were detected in gastrointestinal tumor markers. During hospitalization, he had recurrent fever and dyspnea and was managed with oxygen therapy. His condition was deemed severe, and he was kept nil per os. Comprehensive supportive treatment was initiated, including anti-infective therapy, expectorants, bronchodilators, antipyretics, anti-inflammatory therapy, hepatoprotection, acid suppression with gastric mucosal protection and hemostasis, correction of anemia, intravenous human albumin, maintenance of fluid and electrolyte balance, and nutritional support. Blood typing and preoperative infection screening (eight-item panel) were completed, and a total of 6 units of type-matched packed red blood cells were transfused. After these measures, gastrointestinal bleeding ceased, oral intake was gradually resumed, and his condition stabilized sufficiently to undergo endoscopic evaluation.

Gastroscopy performed on April 7, 2024, revealed retained undigested food at the gastric outlet; distal obstruction could not be excluded, and no neoplastic lesion was identified. Contrast-enhanced upper abdominal CT on April 8, 2024, showed: (1) irregular thickening of the antrum and pylorus as well as the lesser curvature with obstruction, suggestive of a space-occupying lesion; pathological confirmation was recommended; and (2) enlarged retroperitoneal, intra-abdominal, perigastric (lesser curvature), and mediastinal lymph nodes. For further management, he was transferred to the general surgery service.

His medical history was notable for bronchial asthma, for which he used inhaled salbutamol irregularly. During an outside hospitalization in March 2024, he received 2 units of type A packed red blood cells for severe anemia and subsequently required repeat transfusion therapy. He denied any drug or food allergies. His mother died of lung cancer. He had no smoking history and reported drinking approximately 500 mL of beer daily.

Clinical examinations and investigations

Laboratory tests showed a White Blood Cell count (White Blood Cell, WBC) of 15.19×10^9/L (elevated), a Neutrophil percentage (Neu) of 75.6% (elevated), Hemoglobin (HGB) of 54 g/L (decreased), hypersensitive C-reactive protein (Hypersensitive C-Reactive Protein, hsCRP) of 56.03 mg/L, and Albumin (ALB) of 20.5 g/L (decreased). Liver and renal function tests, myocardial enzyme panel, and tumor markers were within normal limits. Electrocardiography demonstrated sinus rhythm with no abnormalities.

Abdominal CT revealed: (1) irregular thickening of the antrum and pylorus as well as the lesser curvature with gastric outlet obstruction, suggesting a space-occupying lesion; pathological correlation was recommended; (2) enlarged retroperitoneal, intra-abdominal, perigastric (along the lesser curvature), and mediastinal lymph nodes; (3) right renal atrophy; (4) bilateral bronchitis with pulmonary infection; (5) small right pleural effusion and small pericardial effusion; (6) cardiomegaly with atherosclerosis of the aorta and coronary arteries; and (7) degenerative changes of the thoracic and lumbar spine (Figure 1).

Upper gastrointestinal contrast study showed delayed passage of contrast through the distal stomach with evidence of gastric outlet obstruction (Figure 2).

Gastroscopy with biopsy demonstrated chronic inflammation of the antral mucosa with erosion and ulcer formation (Figure 3). Histologic grading was as follows: (1) chronic inflammation (++), (2) activity (++), (3) atrophy (–), (4) intestinal metaplasia (–), and (5) dysplasia (–). Immunohistochemistry supported CMV-associated gastritis, with the following results: CMV (+), CD34 (positive in vascular endothelium), CK (+), SMA (–), and Ki-67 (approximately 20% positive) (Figure 4).

Clinical diagnosis

1) Gastric ulcer with cicatricial gastric outlet obstruction
2) Cytomegalovirus (CMV)–associated gastritis
3) Pulmonary infection
4) Hypoproteinemia
5) Anemia
6) Malnutrition

Clinical treatment

The patient was treated with combination anti-infective therapy consisting of cefoperazone–sulbactam sodium and levofloxacin, antiviral therapy with ganciclovir, acid suppression and gastric mucosal protection, blood transfusion, albumin supplementation, and parenteral nutritional support.

A repeat gastroscopy performed on April 22, 2024, revealed pyloric stenosis. The endoscope could not pass through the pyloric canal into the duodenal bulb, suggesting gastric outlet obstruction. An upper gastrointestinal contrast study also demonstrated pyloric narrowing with poor passage of contrast, further supporting the diagnosis of gastric outlet obstruction.

Because conservative medical management was ineffective and there were clear surgical indications without obvious contraindications, the patient underwent distal gastrectomy with gastrojejunostomy under general anesthesia on April 25, 2024.

Intraoperative exploration revealed no ascites. The liver, gallbladder, small intestine, colon, abdominal wall, and pelvic cavity showed no obvious abnormalities. Mild adhesions were observed between the hepatic flexure of the colon, the gallbladder, and the inferior margin of the liver. Adhesions between the anterior gastric wall and the greater omentum

were noted and were carefully dissected. On palpation, mucosal thickening with slightly firm consistency was detected near the antrum along the lesser curvature. The surrounding tissues showed scar-like fibrous proliferation, but no obvious mass was identified. The local serosa showed no contraction or rigidity. The pyloric canal and the duodenal bulb were firm in consistency with marked edema, but no definite tumor was palpated.

Based on the intraoperative findings, gastric ulcer with cicatricial gastric outlet obstruction was considered, and distal gastrectomy with gastrojejunostomy was performed.

Gross examination of the resected specimen revealed an ulcer located on the lesser curvature of the gastric antrum, measuring approximately 2.0×2.0 cm. The pyloric canal was markedly narrowed with significant edema and scar-like fibrous proliferation. The lesion was approximately 4.0 cm from the distal resection margin, while the proximal margin exceeded 10 cm. Postoperative pathological examination showed gastric ulcer in the distal stomach with severe chronic inflammation and atrophic changes in the surrounding gastric mucosa. Edema of the gastric wall stroma and infiltration of acute and chronic inflammatory cells were also observed (Figure 4).

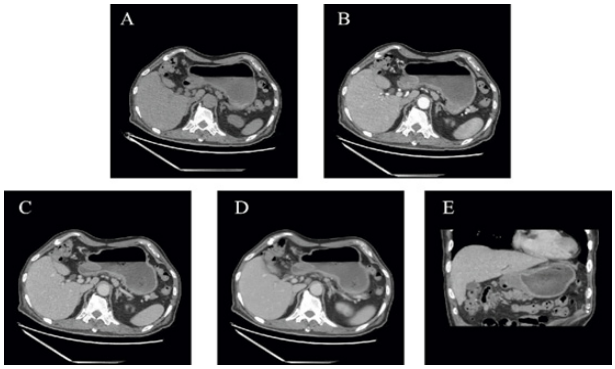

Figure 1: Abdominal plain and contrast-enhanced CT scans. **(A)** Non-contrast abdominal CT; **(B)** Arterial phase; **(C)** Venous phase; **(D)** Equilibrium phase; **(E)** Coronal CT view.

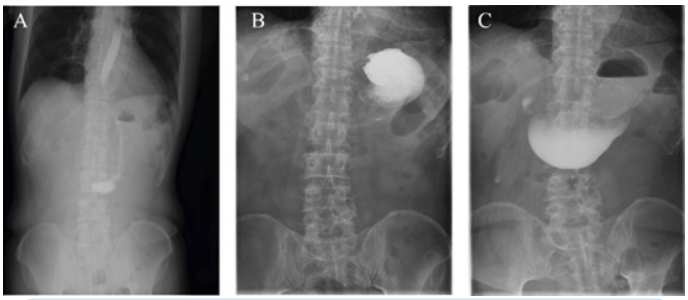

Figure 2: Upper gastrointestinal contrast study. **(A)** 1 Minute after contrast administration; **(B)** 5 Minutes after contrast administration; **(C)** 10 Minutes after contrast administration.

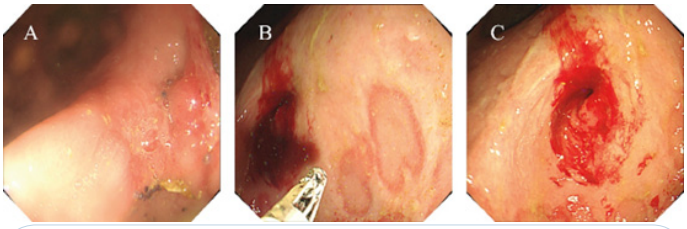

Figure 3: Endoscopic findings. **(A)** Localized polypoid hyperplasia; **(B)** Multiple scattered ulcers; **(C)** Lesion located below the pylorus.

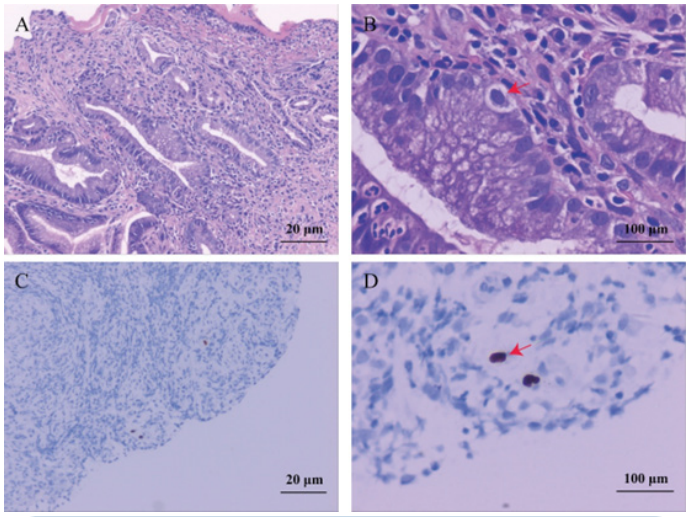

Figure 4: Hematoxylin–eosin (H\&E) staining and immunohistochemistry of the resected specimen. **(A,B)** H&E staining of the postoperative specimen: **(A)** ×100 magnification and **(B)** ×400 magnification; the red arrows indicate CMV viral inclusion bodies. **(C–D)** Immunohistochemical staining for CMV in the postoperative specimen: **(C)** ×100 magnification and **(D)** ×400 magnification; the red arrows indicate strongly positive cells.

Discussion

This case describes CMV-associated gastritis presenting predominantly as gastric outlet obstruction, accompanied by localized polypoid hyperplasia and multiple superficial ulcers. The patient achieved marked symptomatic improvement after surgical resection combined with antiviral therapy. It is well recognized that CMV can injure vascular endothelial cells, and the host immune response to CMV may further amplify inflammation, thereby aggravating vascular damage within the gastric wall [1]. Persistent inflammation and injury may lead to mucosal ischemia and ulcer formation, and in severe cases, may progress to gastric outlet obstruction [2].

CMV is generally regarded as an opportunistic pathogen. In immunocompetent individuals, infection is often asymptomatic. When immune function is compromised, latent CMV may become reactivated and trigger inflammation, resulting in clinical disease. Yuri Longatto Boteon et al. reported an AIDS patient with concomitant CMV infection, in whom CMV led to a gastric pseudotumor and subsequent gastric obstruction [3]. Sung Hwan Kang et al. described CMV infection in a patient with ulcerative colitis, which was also associated with gastric ulceration and gastric outlet obstruction [4]. The present patient belonged to a vulnerable population—an elderly individual with profound malnutrition—yet no evidence of HIV infection or other inflammatory gastrointestinal disorders was identified. In our case, the serum albumin level was 20.5g/L (reference range: 35–55g/L). A comparable report by Suter et al. described CMV-related hypertrophic gastropathy with severe hypoalbuminemia (albumin 17.6 g/L) [5], and Zunaira Jalil also reported a low albumin level of 30g/L [6]. Nevertheless, CMV infection has also been reported in immunocompetent hosts, in whom it may cause inflammatory lesions of the gastrointestinal tract. Zunaira Jalil et al. described a patient with disseminated CMV infection who developed distal gastric outlet obstruction, accompanied by elevated anti-CMV IgM and IgG titers [6]. In addition, Monrobel et al. reported concurrent gastric and colonic CMV infection in an immunocompetent woman, underscoring the diagnostic value of biopsy in elucidating the etiology of mucosal lesions [7]. Although CMV infection

can occur in immunocompetent individuals and manifest as gastritis and peptic ulcer disease, we noted that even in such patients, CMV infection appears to be closely associated with hypoproteinemia, and the two often coexist. Moreover, Verena Dries and Susmitha Tangirala reported CMV infection in preterm and extremely preterm infants, respectively, both of whom developed gastric outlet obstruction; after surgical management combined with anti-CMV therapy, the obstructive gastrointestinal symptoms resolved [8,9].

Whether CMV infection is associated with concomitant Helicobacter pylori (H. pylori) infection remains controversial. Claeys reported CMV and H. pylori coinfection–associated gastritis, whereas in the present case and in the report by Zunaira Jalil, CMV infection occurred in the absence of H. pylori. In addition, Crespo et al. described CMV-associated gastritis concomitant with Epstein–Barr virus infection [10].

In summary, we report a case of CMV-associated gastritis in which virus-driven, long-standing chronic inflammation likely contributed to gastric ulceration, a mass-like lesion, and gastric outlet obstruction. After distal gastrectomy with gastrojejunostomy in combination with antiviral therapy, the patient's symptoms improved substantially. However, several issues remain unresolved. First, the patient had episodes of melena and gastrointestinal bleeding, but a definitive causal relationship with CMV-associated gastritis was not established. Second, it remains unclear whether the patient's pulmonary manifestations (dyspnea, cough, and radiologic evidence of pneumonia) were related to CMV infection. Finally, the directionality of the association between CMV infection and severe malnutrition is uncertain—whether gastric outlet obstruction secondary to CMV led to reduced oral intake and subsequent malnutrition, or whether chronic malnutrition in an elderly patient resulted in immune impairment, predisposing to secondary CMV infection. We speculate that these factors may be bidirectionally related, reinforcing each other and driving a vicious cycle. This case highlights that, in patients with gastric outlet obstruction of unclear etiology—particularly those who are immunocompromised or have significant comorbidities—pathologic evaluation and appropriate tissue staining should be performed to assess for CMV infection, and timely surgical intervention and antiviral therapy should be considered when indicated.

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