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Case Report

Extensive Cytomegalovirus Gastritis with High Tissue Viral Load in an Immunocompetent Host: A Case Report

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Abstract

Background: Cytomegalovirus (CMV) gastritis in immunocompetent adults is uncommon and may mimic peptic ulcer disease, gastric malignancy or severe inflammatory disease. Validated thresholds for tissue CMV DNA quantification in such adults are undefined, and correlations with clinical severity are inconsistent.

Case Presentation: A 51-year-old immunocompetent woman presented with epigastric pain and anemia. Upper gastrointestinal endoscopy revealed extensive gastric ulceration involving approximately 50% of the mucosal surface with friable mucosa and contact bleeding. Histopathology demonstrated characteristic cytomegalic inclusion bodies, confirmed by immunohistochemistry. Quantitative tissue PCR showed a high viral load of 30,321,269 copies/mg tissue (7.48 $\log_{10}$ copies/mg). The patient received intravenous pantoprazole and ganciclovir for 2 weeks, followed by oral valganciclovir for 4 weeks. Symptoms improved rapidly and repeat endoscopy with biopsy at six weeks demonstrated complete mucosal healing with no detectable CMV.

Discussion: This case highlights extensive gastric ulceration by CMV with markedly elevated tissue viral load in an immunocompetent host. Possible mechanisms include endothelial infection, microvascular ischemia and inflammatory amplification.

Conclusion: CMV gastritis should be considered in immunocompetent patients presenting with extensive gastric ulceration. Tissue PCR may facilitate diagnosis, and antiviral therapy can lead to rapid clinical and endoscopic resolution.

Keywords: Cytomegalovirus Gastritis; Immunocompetent Host; Gastric Ulceration; Tissue Viral Load; Cytomegalovirus PCR

Introduction

Cytomegalovirus gastritis in immunocompetent adults is rare and may mimic peptic ulcer disease, gastric malignancy or severe inflammatory disease [1,2]. Isolated gastric involvement without systemic immunosuppression has been reported only sporadically [3]. The pathogenesis in such individuals is incompletely understood, with proposed mechanisms including primary symptomatic infection or local mucosal reactivation [4,5]. Endoscopic findings range from superficial erosions to deep ulcers and diagnosis is clinched by histopathologic identification of cytomegalic inclusion bodies, immunohistochemistry staining, or molecular detection via polymerase chain reaction [6,7]. Management strategies vary from conservative management in milder cases to antiviral therapy in severe disease [3,8,9].

We present a case of severe CMV gastritis in a 51-year-old immunocompetent woman who achieved complete clinical and endoscopic response following antiviral therapy.

Case Presentation

A 51-year-old woman presented with a two-month history of intermittent epigastric pain, dyspepsia, nausea, decreased appetite and low-grade fever. She denied vomiting, hematemesis, melena, weight loss, or jaundice. There was no history of non-steroidal anti-inflammatory drug use, alcohol consumption, or smoking.

Her past medical history was notable for genitourinary tuberculosis treated two years earlier with a six-month course of standard anti-tubercular therapy with documented cure. She had no history of diabetes mellitus, hypertension, chronic kidney disease, or other systemic illnesses. She was not receiving corticosteroids, immunosuppressive agents, or chronic medications, and there was no history of organ transplantation or high-risk HIV exposure.

On examination she was hemodynamically stable with mild conjunctival pallor and mild epigastric tenderness without guarding or organomegaly. Laboratory evaluation showed mild normocytic anemia (hemoglobin 9.3 g/dL); Total leukocyte count and platelet counts were normal. Renal and liver function tests were within normal limits. Glycemic parameters were normal. HIV serology was negative. CD4+ T-lymphocyte count and serum immunoglobulin levels (IgG, IgA, IgM) were within normal ranges, indicating no evidence of systemic immunosuppression. Abdominal ultrasonography and contrast-enhanced computed tomography revealed mild hepatic steatosis without focal lesions or lymphadenopathy.

Laboratory and imaging findings are summarized in Table 1.

Parameter	Result	Reference Range
Hematological parameters		
Hemoglobin	9.3 g/dL	12.0–15.0 g/dL
Total leukocyte count	7,800 cells/µL	4,000–11,000 cells/µL
Platelet count	245,000/µL	150,000–450,000/µL
Biochemical parameters		
Serum creatinine/blood urea	0.8 mg/dL/ 26mg/dL	0.6-1.1 mg/dL / 15-40mg/dL
AST/ALT/Total Bilirubin/Albumin	24/21/0.7/4.1	5-40 U/L / 5-40 U/L / 0.2-1.2 mg/dL / 3.5 -5.2 g/dL
Glycemic parameters		
Fasting plasma glucose	92 mg/dL	70–99 mg/dL
Post-prandial plasma glucose (2 hr)	118 mg/dL	<140 mg/dL
HbA1c	5.4 %	4.0–5.6 %
Immunological parameters		
HIV serology	Negative	Negative
CD4+ T-lymphocyte count	612 cells/µL	500–1,500 cells/µL

Total Serum IgG	1120 mg/dL	700–1600 mg/dL
Total Serum IgA	186 mg/dL	70–400 mg/dL
Total Serum IgM	124 mg/dL	40–230 mg/dL
Imaging		
Abdominal ultrasonography	Mild hepatic steatosis	
Contrast-enhanced CT abdomen	Mild hepatic steatosis, no focal lesion or lymphadenopathy	
Pathology and virology		
Gastric histopathology	CMV inclusion bodies present	Absent
CMV immunohistochemistry	Positive (7–8 CMV-positive cells/HPF)	Negative
Tissue CMV PCR	30,321,269 copies/mg tissue (7.48 log10 copies/mg)	Not detected

Table 1: Laboratory and Imaging Findings in the Present Case.

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; CMV, cytomegalovirus; CT, computed tomography; HbA1c, glycated hemoglobin; HPF, high-power field; IgA, immunoglobulin A; IgG, immunoglobulin G; IgM, immunoglobulin M; PCR, polymerase chain reaction.

Upper gastrointestinal endoscopy revealed multiple superficial and deep gastric ulcers measuring 0.5–3.5 cm with irregular margins involving the fundus, body and antrum, affecting approximately 50% of the gastric mucosal surface with friable surrounding mucosa bleeding easily on contact (Figure 1). The esophagus and duodenum appeared normal. Differential diagnoses included severe peptic ulcer disease, gastric malignancy, granulomatous infection, and severe inflammatory disease (upper GI Crohn's disease).

Multiple biopsies were obtained. Histopathology demonstrated chronic inflammatory infiltrates with enlarged cells showing smudged nuclear chromatin and prominent eosinophilic intranuclear inclusions-classic "owl's-eye" cytomegalovirus inclusion bodies (Figure 2). Immunohistochemistry confirmed CMV antigen positivity, with 7–8 infected cells per high-power field (Figure 3). No Helicobacter pylori, acid-fast bacilli, fungi, dysplasia or malignancy were identified.

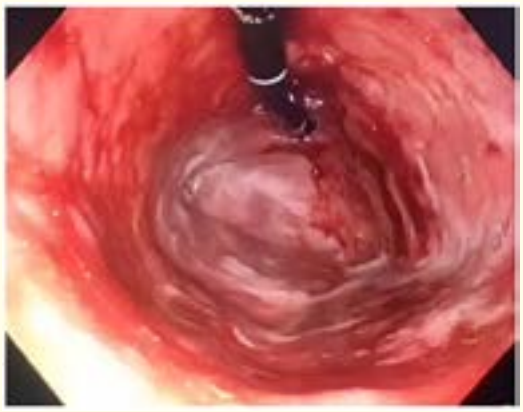

Figure 1: Initial upper gastrointestinal endoscopy showing extensive gastric ulceration with irregular margins, friable mucosa, and contact bleeding.

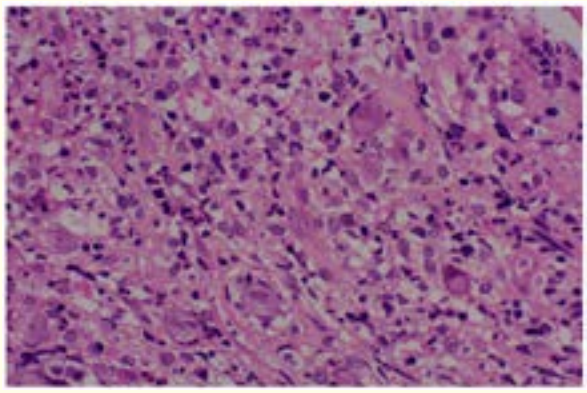

Figure 2: Histopathology showing enlarged cells with characteristic intranuclear "owl's-eye" inclusions (hematoxylin and eosin stain).

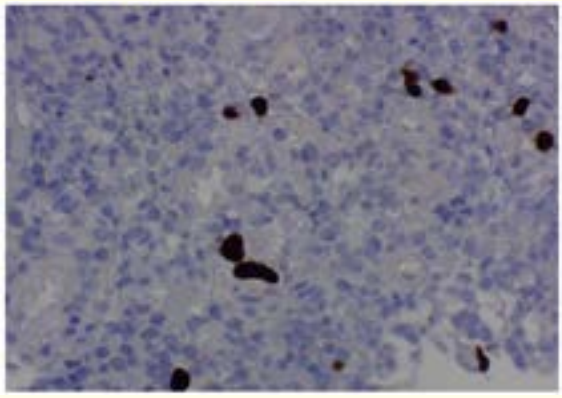

Figure 3: Immunohistochemistry staining demonstrating CMV antigen positivity in infected gastric epithelial cells.

Quantitative PCR for CMV DNA revealed 30,321,269 copies per mg of tissue (7.48 log10 copies/mg), Serum CMV serology and plasma CMV DNA PCR were not obtained. However, the diagnosis of tissue-invasive CMV gastritis was established based on histopathology, positive immunohistochemistry, and elevated tissue viral load.

She was treated with intravenous pantoprazole 40 mg twice daily and intravenous ganciclovir 5 mg/kg 12 hourly. Clinical response was rapid as symptoms improved within one week. Repeat endoscopy at two weeks showed marked reduction in ulcer size and mucosal inflammation (Figure 4). She was discharged on oral pantoprazole 40 mg once a day and valganciclovir 900 mg twice daily for 4 weeks.

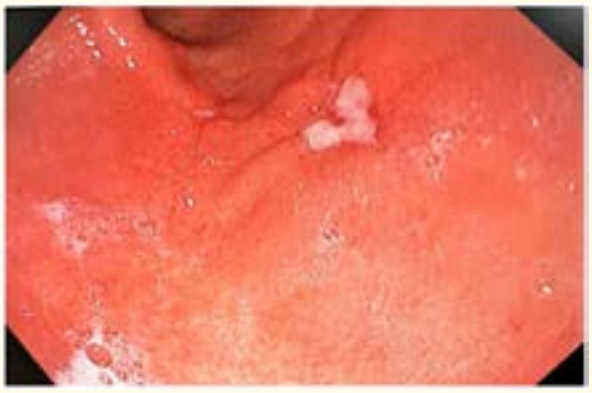

Figure 4: Follow-up endoscopy after two weeks of antiviral therapy showed a significant reduction in ulcer size and improvement in surrounding mucosal inflammation.

At six weeks she remained asymptomatic with haemoglobin increasing to 12.1 g/dL. Follow up endoscopy showed complete resolution of gastric ulcers (Figure 5A and 5B). Repeat biopsies were negative for CMV inclusions, immunohistochemistry and tissue PCR, confirming viral clearance. At six-month follow-up, she remained asymptomatic without recurrence.

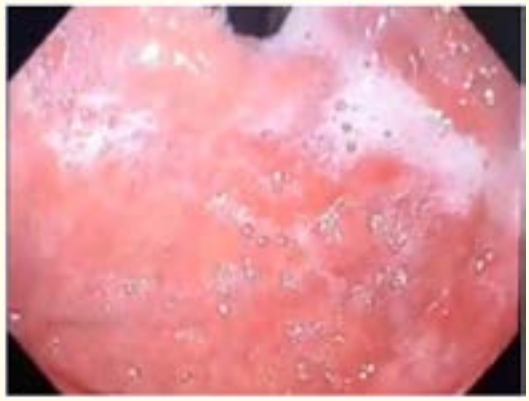

Figure 5A: Endoscopic view of the gastric fundus at six weeks showing complete resolution of gastric ulcers.

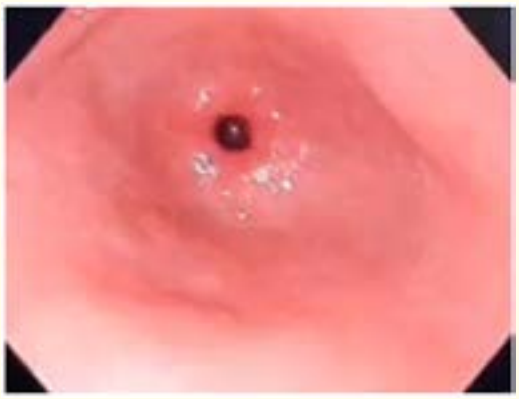

Figure 5B: Endoscopic view of the antrum at six weeks showing complete resolution of ulcers.

Discussion

The striking endoscopic appearance in this case can be understood from the biological behavior of CMV. It shows epithelial and endothelial tropism, causing direct cytopathic effects leading to cellular enlargement, intranuclear inclusion body formation, and cell lysis [7,10]. This contributes to the mucosal friability and superficial erosions, however, deep, irregular ulcers with necrotic

bases is due to vascular injury. It replicates in endothelial cells, inducing injury, fibrinoid necrosis, and microvascular thrombosis [11]. It also contributes to localized tissue ischemia and hypoperfusion, producing geographic areas of mucosal necrosis, an appearance that may mimic malignancy on endoscopy. CMV replication and mucosal inflammation is bidirectional interaction, with inflammatory mediators perpetuating mucosal damage [12]. The geographic ulceration in our patient reflects multifocal endothelial injury leading to regional ischemia across contiguous mucosal territories, despite focal distribution of CMV-positive cells on histology. Similar mechanisms have been described in CMV colitis, where endothelial infection and microvascular injury produce deep colonic ulcers that can perforate [13]. The parallel suggests common biological mechanisms across gastrointestinal sites.

CMV gastritis in an immunocompetent individual raises questions about viral latency and reactivation biology. After primary infection, it establishes lifelong latency in myeloid progenitors, endothelial cells, and epithelial cells [14]. Viral reactivation typically occurs in the setting of immunosuppression-organ transplantation, advanced HIV infection, or high-dose corticosteroid therapy-where impaired T-cell immunity permits unchecked viral replication and disseminated disease. But, localized CMV disease can occur in immunocompetent hosts under specific circumstances, with both primary infection and reactivation recognized as plausible mechanisms [4,5]. Several factors may contribute to compartmentalised gastric disease despite preserved immunity. Local mucosal injury- acid exposure, ischemia, or inflammation creates a permissive microenvironment for viral reactivation within latently infected epithelial or endothelial cells [7,14]. Also, gastric mucosa is an immunologically distinct compartment, and localised immune dysregulation may occur despite intact systemic immunity, as seen in reported cases of tissue-positive but plasma-negative CMV gastrointestinal disease [4,5,9]. Although our patient was not elderly, subtle host-specific variations in mucosal immune regulation may lower the threshold for localised viral reactivation in otherwise immunocompetent hosts [2]. These findings suggest that systemic T-cell–mediated immunity may be sufficient to prevent disseminated infection while localised mucosal immunity becomes transiently impaired [14].

No validated diagnostic thresholds for tissue viral load in CMV gastritis in immunocompetent hosts have been established; high viral tissue burdens have been described in invasive gastrointestinal CMV disease in immunocompromised cohorts [4], consistent with active and clinically significant replication in our patient (7.48 $\log_{10}$ copies/mg), rather than incidental detection of latent virus.

We conducted a focused review of published case reports describing CMV gastritis in immunocompetent adults. PubMed and Google Scholar were searched for articles published between 1990 and 2025 using the terms ("cytomegalovirus" OR "CMV") AND ("gastritis" OR "gastric ulcer") AND ("immunocompetent" OR "normal host"). Reports describing histologically confirmed gastric CMV infection in immunocompetent adults were included. Representative cases are summarized in Table 2.

Author/Year	Age/Sex	Endoscopic Findings	Diagnostic Modality	Quantitative CMV Load	Treatment	Outcome
Kim., *et al*. 2004 [3]	27 M	Multiple gastric ulcers and nodules	Histology and IHC	Not reported	PPI and IV ganciclovir	Resolution
Phyun., *et al*. 2004 [6]	27 F	Ulcer mimicking malignancy	Histology	Not reported	IV ganciclovir	Healing
Baig., *et al*. 2005 [15]	76 F	Erythema and erosions	Histology	Not reported	Supportive care	Improvement
Crespo., *et al*. 2015 [16]	31 M	Superficial erosions	Histology and Plasma PCR	Plasma CMV DNA 5,700 copies/mL	IV ganciclovir - 7 days	Complete resolution
Andrawus and Rainis, 2021 [1]	66 M	Giant gastric ulcer with bleeding	Histology	Not reported	PPI monotherapy	Full resolution
Gonzalez., *et al*. 2023 [17]	36 M	Multiple deep ulcers	Histology and IHC	Not reported	PPI then IV ganciclovir	Healing

Paladiya., *et al*. 2023 [18]	66 M	Mild erythema	IHC positive	Not reported	Valganciclovir - 3 weeks	Improvement
Solito., *et al*. 2023 [19]	70 F	Delayed gastric emptying with mucosal involvement	Histology	Not reported	Antiviral therapy (2 months)	Resolution
Current case, 2025	51 F	Extensive ulceration (50% gastric surface)	Histology + IHC + Tissue PCR	Tissue CMV DNA 7.48 $\log_{10}$ copies/mg	IV PPI +IV ganciclovir (2 weeks) followed by Valganciclovir × 4 weeks	Complete resolution

Table 2: Few published cases of CMV gastritis in immunocompetent adults.

Reported patients span a wide age range and heterogenous endoscopic findings varying from superficial erosions to deep ulcers mimicking malignancy. Diagnosis is based on histopathological identification of cytomegalic inclusions supported by immunohistochemistry or molecular detection. Treatment approach vary, mild disease responds to conservative management, while severe cases require antivirals [1,3,8,9].

Compared with previously reported cases, our patient exhibited two notable features: extensive mucosal involvement affecting approximately half of gastric mucosa and a markedly elevated quantitative tissue viral load. While several reports describe large ulcers or malignancy-mimicking lesions, quantitative tissue viral burden is seldom provided, limiting direct comparison between cases.

The rapid therapeutic response observed provides insight into interaction between antiviral therapy and host immunity. By reducing viral replication and cytopathic injury, antiviral therapy allows mucosal regeneration to proceed while host immune system clears residual infected cells. Prompt clinical improvement and complete endoscopic healing observed in six weeks reflect potential effectiveness of antiviral therapy in severe disease. The decision to initiate antiviral therapy in this case was guided by disease severity—extensive ulceration, ongoing symptoms, anemia, and elevated tissue viral load. Published reports demonstrate that conservative management can be effective in localized disease with mild symptoms [1], while antiviral therapy appears helpful in severe disease [3,8,9]. The dramatic treatment response observed may indicate that viral burden alone does not predict outcome in immunocompetent hosts.

This report has certain limitations. First, plasma CMV DNA and serological markers of CMV were not done, limiting the ability to fully differentiate between localized reactivation and systemic infection. Second, quantitative thresholds for tissue CMV DNA associated with clinical severity are not well established, hence future studies across cohorts of patients may be needed to determine whether tissue viral load serves as a useful biomarker for treatment decisions or prognostication.

Conclusion

Cytomegalovirus gastritis can occur in immunocompetent individuals and may present with extensive ulceration mimicking severe peptic ulcer disease, malignancy or inflammatory disease (Crohn's disease). Diagnosis relies on histopathology, immunohistochemistry and molecular detection. In severe mucosal disease, antiviral therapy may lead to rapid clinical improvement and complete endoscopic healing. Clinicians should maintain a high index of suspicion for CMV infection in atypical or severe gastric ulceration, even in the absence of overt immunosuppression.

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Conflicts of Interest

The authors declare that they have no financial or non-financial competing interests related to this work.

Ethics Approval

This case report was conducted in accordance with the ethical standards of the institutional and national research committee and with the principles of the Declaration of Helsinki and its later amendments.

Consent to Participate

Informed consent was obtained from the patient for participation in this study.

Written Consent for Publication

Written informed consent was obtained from the patient for publication of this case report and the accompanying clinical images.

Availability of Data and Material

The data supporting the findings of this study are contained within the article. No additional datasets were generated or analyzed during the current study.

Code Availability

Not applicable.

Author Contributions

- Subhadeep Banerjee -conception of the case report, patient management, data collection, literature review, and drafting of the manuscript.
- Dawesh Prakash Yadav- supervised the study, contributed to interpretation of clinical findings, and critically revised the manuscript.
- Sunit Shukla – contributed to interpretation of clinical findings and critically revised the manuscript
- Anurag Tiwari- contributed to endoscopic evaluation and interpretation of findings.
- Vinod Kumar -contributed to endoscopic evaluation and interpretation of findings.
- Sarita Mittal- contributed to histopathological interpretation and diagnostic confirmation.
- Latika Gupta- contributed to histopathological interpretation and diagnostic confirmation.
- Vinit Kumar -contributed to literature review and critical revision of manuscript
- Nitesh Kumar Patel - contributed to literature review and critical revision of manuscript
- Ajay Singla- contributed to literature review and critical revision of manuscript
- All authors read and approved the final manuscript.

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