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Self-Limited Gastrointestinal Illness With Positive Cytomegalovirus Serology in a Young Immunocompetent Adult: A Case Report

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Abstract

Cytomegalovirus (CMV) colitis is uncommon in young immunocompetent individuals, and distinguishing systemic CMV infection from tissue-invasive colonic disease can be challenging. A 28-year-old immunocompetent male patient presented with fatigue, nausea, postprandial left lower quadrant abdominal pain, watery nonbloody diarrhea, decreased oral intake, and weight loss. Laboratory testing showed lymphocytosis and elevated transaminases, while an abdominal computed tomography report described mild splenomegaly and nonspecific colonic changes potentially associated with inflammation. CMV IgM and IgG were positive, and Epstein-Barr virus (EBV) serologies were also positive, although EBV blood polymerase chain reaction (PCR) was negative. CMV quantitative PCR, stool pathogen studies, and colonoscopic biopsy were not obtained during the acute illness. Symptoms resolved without antiviral therapy. Delayed colonic biopsies showed benign mucosa without evidence of CMV infection. Thus, the patient's gastrointestinal illness occurred in association with positive CMV serology, but the absence of acute-phase virologic and histopathologic evidence prevents establishing that CMV caused the colonic abnormalities. This case highlights the diagnostic limitations of attributing a self-limited gastrointestinal illness to CMV without tissue confirmation and adequate evaluation for alternative enteric infections.

Categories: Internal Medicine, Infectious Disease, Gastroenterology
Keywords: case report, cmv colitis, colitis, cytomegalovirus, cytomegalovirus infection, immunocompetent

Introduction

Cytomegalovirus (CMV), a member of the Herpesviridae family, causes significant morbidity among elderly and immunocompromised populations, whereas infection in immunocompetent individuals is often asymptomatic or subclinical [1]. CMV infection occurs in approximately 60% of adults in developed countries and more than 90% of adults in developing countries. Still, end-organ disease is uncommon in immunocompetent hosts because of an effective immune response [2]. In contrast, immunocompromised and HIV-positive patients may develop disseminated disease involving the retina, gastrointestinal tract, lungs, or central nervous system [3,4].

CMV colitis in immunocompetent patients remains uncommon and predominantly affects older adults, particularly those with underlying comorbidities [5]. Reported median ages range from 65 to 81 years, and many affected patients have renal disease, diabetes, or other conditions requiring long-term care [1,5]. Additional reported risk factors include steroid exposure, lymphopenia, and recent red blood cell transfusion [6]. Clinical presentation is nonspecific and may include abdominal pain, diarrhea, hematochezia, melena, and fever [5]. During active disease, endoscopy may reveal well-defined punched-out ulcerations, and tissue biopsy can demonstrate characteristic CMV inclusions [7]. Positive CMV serology alone does not establish tissue-invasive CMV colitis; diagnosis generally requires colonic tissue evidence, typically by histopathology with or without immunohistochemistry. Some cases of confirmed CMV gastrointestinal disease resolve without antiviral therapy, although ganciclovir is commonly used for clinically significant disease, with foscarnet generally reserved for resistant infection [8,9]. We report a young immunocompetent adult with a self-limited gastrointestinal illness and positive CMV serology in whom no tissue evidence of CMV colitis was established.

Case Presentation

A 28-year-old male patient presented to the family medicine clinic with 1.5 weeks of fatigue, nausea, and weight loss. Approximately 10 days before presentation, he developed postprandial left lower quadrant abdominal pain accompanied by watery, nonbloody diarrhea. By the time of his initial clinic evaluation, he was no longer experiencing active abdominal pain but reported avoiding food because eating reliably triggered his symptoms. His oral intake had decreased to approximately one meal per day, resulting in a 10-pound weight loss. The pain typically began about 20 minutes after eating, reached a severity of 9/10, lasted approximately two hours, and gradually improved before resolving. By approximately 14 days after

symptom onset, the postprandial abdominal pain had nearly resolved. His medical history was notable for obesity, while his surgical and family histories were unremarkable. Relevant exposures included close contact with his child, who attended daycare, and a history of vaping.

On physical examination, he was afebrile with a temperature of 99.6°F (37.6°C), blood pressure of 115/76 mm Hg, pulse of 80 beats per minute, weight of 260 pounds (117.9 kg), and body mass index of 37.31 kg/m². Review of systems was positive for decreased appetite, abdominal pain, and nausea; all other systems were negative. Abdominal examination revealed normal bowel sounds and a soft, nontender abdomen. The remainder of the physical examination was unremarkable. Initial laboratory findings are presented in Table *1*.

Laboratory test	Patient value	Reference range
WBC count (K/µL)	9.4	4.4-10.1
Neutrophils (%)	37.8	43.7-84.9
Lymphocytes (%)	50.7	8.4-40.7
Absolute lymphocyte count (K/µL)	4.8	1.4-2.9
Absolute monocyte count (K/µL)	0.85	0.2-0.8
Aspartate aminotransferase (IU/L)	121	<34
Alanine aminotransferase (IU/L)	122	10-49

TABLE 1: Patient laboratory values

IU/L: international units per liter; K/µL: thousands per microliter; WBC: white blood cell

The official CT report of the abdomen and pelvis with contrast documented mild splenomegaly and submucosal fat deposition in the colon, most pronounced at the splenic flexure. The report noted that the colonic finding may be seen in the setting of inflammatory or infectious colitis. The original CT images were not available for inclusion in this report. The patient was advised to follow up in the family medicine clinic for additional laboratory testing. Follow-up laboratory results obtained the next day are presented in Table *2*.

Laboratory test	Result
SARS-CoV-2 PCR	Negative
HIV-1 Ag/Ab reactivity	Nonreactive
HIV-2 Ag/Ab reactivity	Nonreactive
Anti-CMV IgM	Positive
Anti-CMV IgG	Positive
Anti-EBV viral capsid antigen IgM	Positive
Anti-EBV viral capsid antigen IgG	Positive
Anti-EBV nuclear antigen IgG	Positive
Anti-EBV early antigen-diffuse IgG	Negative
EBV quantitative PCR	Negative

TABLE 2: Laboratory testing results

Ag/Ab: antigen/antibody; CMV: cytomegalovirus; EBV: Epstein-Barr virus; HIV: human immunodeficiency virus; IgG: immunoglobulin G; IgM: immunoglobulin M; PCR: polymerase chain reaction; SARS-CoV-2: severe acute respiratory syndrome coronavirus 2

Results were reported qualitatively in the available clinical records; numerical CMV antibody indices, assay cutoffs, and test-specific reference intervals were not available for inclusion

CMV IgM and IgG were positive, while HIV-1 and HIV-2 antigen/antibody testing was nonreactive. EBV viral capsid antigen IgM and IgG and EBV nuclear antigen IgG were positive, whereas EBV early antigen-diffuse IgG was negative. EBV quantitative PCR was also negative. EBV PCR was obtained to evaluate possible active EBV infection in the setting of positive EBV serologies. The positive EBV nuclear antigen IgG and negative EBV blood PCR were compatible with past EBV exposure, although they did not definitively exclude recent EBV infection. Concurrent CMV and EBV IgM positivity can be difficult to interpret because nonspecific reactivity or serologic cross-reactivity may occur. CMV PCR testing was not performed during the acute illness, and CMV IgG avidity or paired serologic testing was not reported. The available findings prompted consideration of recent CMV infection but did not establish active CMV infection or CMV involvement of the colon.

The official follow-up abdominal ultrasound report documented that the liver and spleen were at the upper limits of normal in size without enlargement. The original ultrasound images were not available for inclusion in this report. The acute diarrheal illness and CT-reported colonic abnormalities initially suggested an infectious or inflammatory colonic process. Concurrent lymphocytosis, elevated transaminases, and mild splenomegaly raised concern for a systemic viral illness. Following the positive CMV IgM and IgG serologies, CMV infection was considered a possible explanation for the gastrointestinal symptoms and reported colonic changes. However, the CT findings were nonspecific, and neither CMV quantitative PCR nor colonic tissue sampling was obtained during the acute illness. Thus, CMV colitis was considered presumptive rather than confirmed. The patient was referred to colorectal surgery.

At the colorectal surgery evaluation 14 days after his initial presentation, he weighed 249 pounds, representing an 11-pound decrease from his initial clinic weight. He reported near-complete resolution of abdominal pain but continued to experience intermittent nausea and diarrhea with ongoing weight loss. His fatigue and appetite were gradually improving. Ondansetron was prescribed for nausea. No antiviral therapy or other CMV-directed treatment was initiated because his symptoms were already improving spontaneously.

Two months later, following clinical resolution of his gastrointestinal symptoms, a repeat CT of the abdomen and pelvis with contrast was obtained before colonoscopy to reassess the previously reported splenomegaly and colonic abnormalities. The primary rationale was to evaluate for persistent colonic inflammation before proceeding with endoscopic evaluation, given concern that performing colonoscopy in the setting of significant active inflammation could increase the risk of bowel perforation. The official repeat CT report documented that the spleen was at the upper limit of normal size. The original images from this examination were unavailable for inclusion in this report.

He subsequently underwent colonoscopy with biopsies of the cecum, splenic flexure, and rectum. According to the official pathology report, the cecal biopsy contained fragments of benign polypoid colonic mucosa

with lymphoid aggregates, the splenic flexure biopsy contained fragments of benign colonic mucosa with lymphoid aggregates, and the rectal biopsy contained fragments of benign polypoid mucosa. The pathology report did not identify evidence of CMV infection. Original histopathology slides or photomicrographs were not available for inclusion in this report.

Because tissue sampling occurred after clinical resolution, the negative pathology may have reflected resolution of an earlier inflammatory process or sampling of previously uninvolved mucosa. Equally, CMV may never have caused the gastrointestinal illness, which could instead have resulted from an unrelated self-limited viral or bacterial enteric infection. Systemic CMV infection without colonic tissue invasion also remains possible, but was not confirmed. The clinical presentation and positive CMV serology prompted consideration of CMV colitis; however, the absence of CMV PCR or histopathologic confirmation during active illness, together with negative delayed biopsies and incomplete stool pathogen testing, precludes attributing the colonic abnormalities to CMV. The patient did not return for further follow-up.

Discussion

This case describes a self-limited gastrointestinal illness with positive CMV serology in an immunocompetent 28-year-old male patient. Confirmed CMV colitis would be unusual at this age because it most commonly occurs in older or immunocompromised patients. Reported risk factors include chronic steroid use, inflammatory bowel disease, HIV/AIDS, lymphopenia, recent transfusion, and significant medical comorbidity [5,6,10]. In contrast, this patient was young and immunocompetent and had no major comorbidities other than class II obesity. A multicohort study of approximately 540,000 participants found that obesity was associated with a 1.3-fold increased hazard of herpesvirus infection and a 2.3-fold increased hazard of acute viral infection. However, the study did not report CMV-specific risk [11]. Obesity may also impair antiviral immune responses by affecting CD8+ T cells, natural killer cells, and interferon signaling [12]. These mechanisms offer a potential association between obesity and viral susceptibility but do not establish that CMV caused this patient's gastrointestinal illness.

A potential source of CMV exposure, if the patient had acquired a recent infection, was his close contact with a young child attending daycare. Young children may acquire CMV, remain asymptomatic, and shed virus for prolonged periods [13]. A systematic review and meta-analysis reported a higher prevalence of CMV infection among children attending daycare centers than among children without daycare exposure [14]. Although this exposure is epidemiologically plausible, this patient's route of transmission, as well as the timing or presence of an active CMV infection, cannot be established.

CMV infection can produce laboratory abnormalities across multiple organ systems [15,16]. Lymphocytosis, including atypical lymphocytes, is frequently described in immunocompetent patients, and hepatic involvement may produce elevations in aspartate aminotransferase and alanine aminotransferase [15]. In this patient, the absolute lymphocyte count was elevated, and AST and ALT were 121 IU/L and 122 IU/L, respectively. These findings were consistent with a systemic viral illness but were nonspecific and could not independently establish CMV infection. In particular, given the patient's BMI of 37.31 kg/m^2, metabolic dysfunction-associated steatotic liver disease was another potential explanation for elevated transaminases, although this was not diagnosed. The cause of the liver enzyme abnormalities therefore remained uncertain.

Alternative explanations for the acute gastrointestinal illness include self-limited viral gastroenteritis, bacterial enterocolitis (including *Salmonella*, *Shigella*, or *Campylobacter* infection), *Clostridioides difficile* (*C. difficile*) infection, and parasitic infection such as giardiasis. Noninfectious considerations include new-onset inflammatory bowel disease and colorectal malignancy [17]. Stool bacterial cultures, multiplex gastrointestinal pathogen testing, *C. difficile* testing, and stool ova-and-parasite examinations were not performed during the acute illness. Therefore, an alternative enteric infection could not be excluded. Importantly, CMV serology alone cannot establish a diagnosis of CMV colitis because it indicates systemic exposure or possible recent infection rather than tissue-invasive gastrointestinal disease. Additionally, while quantitative CMV PCR provides evidence of active CMV infection, it alone does not establish a diagnosis of CMV colitis. Histopathologic confirmation from colonoscopic biopsy remains the diagnostic standard and may demonstrate CMV staining and characteristic intranuclear inclusions. During active disease, colonoscopy may also show large ulcers, mucosal hemorrhage, and friable mucosa [9]. CT findings are nonspecific but can include colonic wall thickening, mucosal edema, and thickened haustral folds, while splenomegaly has been reported in systemic CMV infection [15,17].

The principal limitation in this case is the absence of histopathologic confirmation during the symptomatic phase. We did not have access to the original imaging or pathology slides for independent review and therefore relied on the findings documented in the radiology and pathology reports. The initial CT imaging report described mild splenomegaly and colonic submucosal fat deposition, most pronounced at the splenic flexure, and interpreted these findings as consistent with inflammatory or infectious colitis. Colonoscopy was deferred during the acute illness, and by the time it was performed two months later, the patient's symptoms had resolved. The repeat CT was obtained before colonoscopy to evaluate for persistent inflammation that might increase the risk of perforation during endoscopic evaluation. However, imaging alone cannot definitively exclude active mucosal inflammation or eliminate procedural risk. Moreover, given the patient's young age and clinical improvement, the need for repeat CT was uncertain and must be

weighed against additional radiation exposure. The pathology report did not identify CMV in biopsies from the cecum, splenic flexure, or rectum, and CMV PCR testing was not done at the initial presentation. Consequently, this case is best characterized as a self-limited gastrointestinal illness associated with positive CMV serology, without sufficient evidence to establish tissue-invasive CMV colitis. The negative delayed biopsies do not confirm the earlier diagnosis; they may reflect resolution before tissue sampling, sampling of uninvolved tissue, or a gastrointestinal illness that was not caused by CMV. In addition, the absence of stool bacterial studies, C. difficile testing, ova-and-parasite examinations, and other enteric pathogen investigations limited the evaluation for alternative infectious etiologies. The initial illness could therefore have represented a self-limited infection unrelated to CMV.

The concurrent EBV and CMV serologic findings introduced additional uncertainty. EBV viral capsid antigen IgM and IgG and EBV nuclear antigen IgG were positive, whereas EBV quantitative blood PCR was negative. Positive EBV nuclear antigen IgG supported prior exposure, but negative blood PCR did not definitively exclude recent EBV infection. Likewise, positive CMV IgM may reflect recent infection, persistent IgM, or nonspecific or cross-reactive antibody responses, particularly in the setting of concurrent EBV IgM positivity [18]. Accordingly, neither the EBV nor the CMV serologic profile was interpreted as definitive evidence of an active infection. Without CMV quantitative PCR, IgG avidity, serial serologic studies, or histopathologic evidence during active symptoms, the timing of CMV infection and any causal link to the gastrointestinal illness remained uncertain.

The patient's improvement without antiviral therapy was consistent with a self-limited gastrointestinal illness but did not establish its etiology. In a retrospective cohort of 86 immunocompetent patients with confirmed CMV gastroenterocolitis, a subset improved without antiviral therapy, and untreated patients often showed clinical improvement before histologic confirmation [9]. By contrast, severe CMV colitis can present with significant gastrointestinal bleeding, hemodynamic instability, perforation, or toxic megacolon and may require hospitalization and antiviral treatment [19]. This patient remained hemodynamically stable, had no gastrointestinal bleeding, and experienced progressive symptom resolution with supportive care alone; these findings are not specific to CMV disease.

This case is clinically relevant because it illustrates the challenges of interpreting positive CMV serology in a young immunocompetent adult with nonspecific gastrointestinal symptoms. CMV can be considered within the differential diagnosis, but serology alone does not establish tissue-invasive colonic disease. When clinically indicated, testing for alternative enteric pathogens and appropriately timed endoscopic tissue sampling may help clarify the diagnosis. A negative biopsy obtained after symptoms have resolved does not prove either prior CMV colitis or complete spontaneous clearance.

Conclusions

This case describes a self-limited gastrointestinal illness in a young, immunocompetent adult with positive CMV IgM and IgG, lymphocytosis, elevated transaminases, and nonspecific colonic abnormalities on CT. No CMV quantitative PCR or tissue biopsy was obtained during the symptomatic period; delayed colonic biopsies showed no evidence of CMV, and stool testing for other enteric pathogens was not performed. Therefore, the available findings do not establish that CMV caused the gastrointestinal illness or that CMV colitis spontaneously resolved. The case emphasizes the distinction between positive systemic CMV serology and proven tissue-invasive colonic disease, as well as the importance of considering alternative infectious etiologies and diagnostic uncertainty.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Hassan W. Makki, Mohamad Kassem, Alexis K. Grover, Tana K. Cooper

Acquisition, analysis, or interpretation of data: Hassan W. Makki, Tana K. Cooper

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Critical review of the manuscript for important intellectual content: Hassan W. Makki, Mohamad Kassem, Alexis K. Grover, Tana K. Cooper

Supervision: Tana K. Cooper

Disclosures

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