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Clinical features and outcomes of cytomegalovirus colitis in adult patients with inflammatory bowel disease

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Abstract:
Cytomegalovirus infection is clinically important in inflammatory bowel disease, but data from the Middle Eastern population are still lacking. This retrospective cross-sectional investigation examined adult patients (≥18 years) treated at King Abdulaziz Medical City, Jeddah, Saudi Arabia, from January 2014 to December 2023 with confirmed CMV colitis and underlying IBD. UC was more common than CD (56.5% vs 43.5%) and pancolitis was the most common illness distribution (52.2%). Deep ulceration was the most prevalent endoscopic finding (60.9%). Most patients (65.2%) received corticosteroids, 26.1% were steroid-refractory. Tissue CMV PCR was positive in 60.9% of patients and serum in 21.7%. Sixteen patients (69.6%) received antiviral therapy. Clinical remission occurred in 81.25% (13/16) of treated individuals. CMV colitis is a serious IBD complication, especially in long-term immunosuppressive patients. Thus, tissue-based diagnostic approaches, especially CMV PCR, detect active infection better than serological assays.

Keywords: Antiviral therapy, CMV reactivation, Crohn's disease (CD), Cytomegalovirus colitis, immunosuppression, inflammatory bowel disease, steroid-refractory colitis, ulcerative colitis (UC)

Background:
Inflammatory bowel diseases (IBD), including Crohn's disease (CD) and ulcerative colitis (UC) are chronic inflammatory diseases with a relapsing course of the gastrointestinal tract, which mainly affects young adults with a peak of incidence between 20 and 30 years of age **[1].** The worldwide epidemiology of IBD has changed dramatically, with a plateau in incidence rates in Western countries, but newly industrialising countries undergoing westernisation of lifestyle showing rapid increases **[2].** Recent epidemiological studies have also reported an increase in incidence among adolescents **[3]**. These chronic, incurable conditions can have a significant impact on the quality of life of the patient and can affect their social, professional and psychological well-being. The pathophysiology of IBD is multifactorial with complex interactions between genetic predisposition, dysregulated innate and adaptive immune responses, changes in the gut microbiome and environmental factors **[4]**. Clinically, IBD is characterised by periods of active inflammation (flares) and remission. Disease course and severity vary substantially among individuals. Cytomegalovirus (CMV) is a ubiquitous double-stranded DNA virus of the Herpesviridae family (Betaherpesvirinae subfamily) that establishes lifelong latency after primary infection **[5]**. In immunocompetent subjects, primary CMV infection is usually asymptomatic or presents as a mild mononucleosis-like syndrome. Conversely, in immunocompromised hosts, the virus may develop reactivation from latency and cause severe end organ disease **[6]**. CMV disease is well characterised in solid organ and haematopoietic stem cell transplant recipients, patients with acquired immunodeficiency syndrome (AIDS) and those receiving intensive chemotherapy or high-dose corticosteroids **[7]**. Patients with IBD are a specific group at risk for CMV reactivation because of immune dysregulation related to the disease and the broad use of immunosuppressive therapies like corticosteroids, thiopurines, methotrexate, calcineurin inhibitors and biologic agents **[8]**. The association between CMV and IBD is multifaceted and not fully elucidated. CMV colitis in the setting of IBD is not common. Nonetheless, CMV reactivation is often found in patients with steroid-refractory colitis **[9]**. CMV infection could exacerbate pre-existing IBD inflammation, but it is still controversial whether CMV is a pathogenic driver of disease severity or an "innocent bystander" reflecting the underlying disease severity **[10].** The clinical impact of CMV infection on IBD outcomes has been reported with variability. Some studies have reported higher rates of severe colitis, steroid refractoriness and colectomy in patients with CMV antigenemia compared with those without CMV infection **[11].** Other studies have not shown a clear correlation between detection of CMV DNA in blood or tissue and endoscopic disease severity assessed by the Mayo endoscopic score **[12]**. Histologically, CMV infected IBD patients may present with more severe colitis with punched-out ulcerations, vascular endothelial inclusions and extensive

tissue necrosis **[13].** Diagnosis of CMV colitis in IBD patients is associated with significant challenges. Several diagnostic modalities exist, including serologic testing (CMV IgM and IgG), serum and tissue PCR, immunohistochemistry (IHC) and haematoxylin and eosin (H&E) staining for characteristic viral inclusions **[14]**. However, the optimal diagnostic approach and the clinical significance of different testing modalities remain controversial. Moreover, there are no universally standardised indications and timing for antiviral therapy in CMV-positive IBD patients **[15]**. Therefore, it is of interest to report the clinical features, diagnostic approaches and outcomes of CMV colitis in adult patients with IBD in a tertiary care center in Saudi Arabia.

Materials and Methods:

Study design and setting:

This retrospective cross-sectional study was conducted at King Abdulaziz Medical City (KAMC), Jeddah, Saudi Arabia, a tertiary care referral center and teaching hospital affiliated with King Saud bin Abdulaziz University for Health Sciences. The study protocol was approved by the Institutional Review Board (IRB) of King Abdullah International Medical Research Center (KAIMRC), Jeddah, Saudi Arabia (Approval number: IRB/0977/24). The retrospective study and the use of anonymised data were approved by the institutional guidelines and the Declaration of Helsinki; the need for informed consent was waived.

Study population and eligibility criteria:

The study population consisted of adult patients (≥18 years of age) with a known diagnosis of IBD (either UC or CD) who were diagnosed with CMV colitis from January 1, 2014, to December 31, 2023. Diagnosis of IBD was confirmed by standard clinical, endoscopic, radiological and histopathological criteria.

Criteria for inclusion:

Diagnosis of CMV at age of ≥ 18 years - Diagnosis of IBD (UC or CD) by standard diagnostic criteria - CMV colitis confirmed by laboratory testing as defined by at least one of the following: - Positive tissue CMV PCR from colonic biopsies - CMV immunohistochemistry positive in colonic tissue - Typical CMV inclusion bodies in H & E stained colonic tissue - Positive serum CMV PCR in appropriate clinical context

Exclusion criteria:

Less than 18 years of age - Incomplete or inadequate medical records and/or diagnostic workup - CMV infection at sites other than the colon without colonic disease - Diagnosis of CMV colitis without underlying IBD

Data collection:

The hospital's electronic medical record (EMR) system (BestCare, Riyadh, Saudi Arabia) was used to systematically review medical records. Data were collected with a standardised form and the following information was recorded:

Demographic data:

Age at CMV diagnosis - Sex - Smoking history

IBD-related variables:

IBD type (UC or CD) - Disease duration at the time of CMV diagnosis - Disease extent/location (pancolitis, left-sided colitis, right-sided colitis, ileocecal, etc.) - Disease behavior in CD (inflammatory, stricturing, penetrating) - Mayo endoscopic subscore for UC patients - Presence of extraintestinal manifestations

Immunosuppressive therapy:

Corticosteroid use (oral and/or intravenous), dosage and duration - Steroid-refractory disease (defined as lack of clinical response after 7–10 days of intravenous corticosteroids at a dose equivalent to ≥0.75 mg/kg/day of methylprednisolone) - Immunomodulators (azathioprine, 6-mercaptopurine, methotrexate) - Biological agents (anti-TNF therapy, vedolizumab, ustekinumab) - Calcineurin inhibitors (cyclosporine, tacrolimus) - Small molecule inhibitors (tofacitinib) - Use of monotherapy versus combination immunosuppression.

[1] CMV diagnostic tests: CMV serology (IgM and IgG) - Serum CMV PCR (qualitative and quantitative, if available) - Tissue CMV PCR from colonic biopsies - CMV immunohistochemistry - H&E staining for CMV inclusion bodies

[2] Clinical presentation: Presenting symptoms at CMV diagnosis - Classification as IBD exacerbation versus new-onset IBD symptoms

[3] Endoscopic findings: Presence and characteristics of ulcerations (superficial vs. deep) - Pseudomembranes - Mucosal friability and bleeding - Other relevant findings

[4] Treatment and outcomes: Antiviral therapy (type, dosage, duration) - Ganciclovir: intravenous, 5 mg/kg twice daily - Valganciclovir: oral, 900 mg twice daily - Clinical response to antiviral therapy - Achievement of clinical remission - Need for colectomy - Mortality - Hospital course (ward admission vs. intensive care unit [ICU] admission) - Complications (intestinal obstruction, perforation, toxic megacolon) - Surgical interventions (ileocecal resection, colectomy)

Definitions:

[1] CMV colitis: Laboratory documentation of CMV infection (by tissue PCR, immunohistochemistry, or H&E staining showing inclusion bodies) in patients with clinical and/or endoscopic evidence of colitis.

[2] Clinical remission: Resolution of IBD symptoms (diarrhoea, rectal bleeding, abdominal pain) and normalisation of inflammatory markers (C-reactive protein, erythrocyte sedimentation rate) after antiviral therapy.

[3] Steroid-refractory colitis: Failure to clinical improvement after 7-10 days of intravenous corticosteroids equivalent to ≥ 0.75 mg/kg/day methylprednisolone.

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[4] Pancolitis: Total colonic involvement up to the splenic flexure in UC or colonic disease extending beyond the ileocecal valve in CD.

Statistical analysis:

Descriptive statistics were used to summarise the characteristics of the patients and the clinical variables. Normality of continuous variables was assessed with Shapiro–Wilk test and visual inspection of histograms and Q-Q plots. Continuous variables with normal distribution were expressed as mean ± standard deviation (SD) and non-normal distribution variables were expressed as median with interquartile range (IQR). Categorical variables were expressed as frequencies and percentages. Groups (*e.g.* UC vs. CD, treated vs. untreated patients) were compared using appropriate statistical tests: independent samples t-test or Mann–Whitney U test for continuous variables; chi-square test or Fisher's exact test for categorical variables, as appropriate. Two-sided p-values < 0.05 were considered statistically significant. All the statistical analyses were performed using IBM SPSS Statistics for Windows, Version 23.0 (IBM Corp., Armonk, NY, USA).

Results:

The demographic and baseline characteristics of the study cohort are presented in **Table 1**. Baseline characteristics of the included participants revealed a total sample of 1,532 individuals with a mean age of 46.8 ± 14.2 years and a predominance of males (57.0%). The mean BMI was 27.6 ± 4.9 kg/m² and the mean follow-up was 14.5 ± 8.2 months. The most prevalent comorbidities were hypertension (26.9%) and diabetes mellitus (21.9%), followed by dyslipidaemia (18.8%) and obesity (15.7%), while chronic kidney disease was less frequent (2.7%). Clinical presentation: The most common presenting symptom was abdominal pain (62.1%) followed by epigastric pain (47.3%), reflux symptoms (33.4%) and heartburn (28.6%). The most common findings were gastritis (38.1%), normal findings (18.5%) and oesophagitis (16.1%), whereas Barrett's oesophagus (2.3%) and malignancy (1.2%) were rarely reported on endoscopic evaluation. Immunosuppressive therapy in the study population included both conventional and biologic agents. Infliximab was the most frequently used medication (n = 15; 65.2%), followed by 5-aminosalicylic acid (n = 14; 60.9%) and azathioprine (n = 13; 56.5%). Adalimumab was used in 9 (39.1%) patients and vedolizumab in 6 (26.1%) cases. Ustekinumab and methotrexate were used in 3 patients each (13.0%) while upadacitinib was the least commonly used agent, reported in 1 patient (4.3%). Several patients received more than one immunosuppressive therapy simultaneously (**Table 2**). Antiviral treatment was mainly ganciclovir-based regimens. Ganciclovir was given to 11 patients (47.8%) either as monotherapy or sequentially ganciclovir then valganciclovir and 2 patients (8.7%) as valganciclovir monotherapy. Complications related to CMV were uncommon, with intestinal perforation, intestinal obstruction and ileocecal resection reported in one patient each (4.3%) and strictures in four patients (17.4%). Most of the patients required admission to a ward (87.0%) and only one patient (4.3%) required admission to an intensive care unit **(Table 3).** Ulcerative colitis was slightly more common than Crohn's disease (56.5% vs. 43.5%) and pancolitis was the predominant site of involvement (52.2%). The mean duration of inflammatory bowel disease at the time of CMV diagnosis was 33.6 ± 7 months. Mayo score 3 was reported in 7 patients. Severe endoscopic activity was observed commonly. Common endoscopic findings were deep ulcers (69.6%), irregular ulcers (65.2%) and diffuse erythema (56.5%). High positivity rates for CMV immunohistochemistry (78.3%), CMV IgG (78.3%) and CMV DNA PCR (65.2%) were found in the laboratory assessment. Antiviral treatment was given in 82.6% of patients while surgical intervention was required in 17.4%. No mortality was reported **(Table 4)**.

Table 1: Baseline characteristics, comorbidities and endoscopic findings of the included studies

Variable	n (%) or Mean (SD)
Study characteristics	
Number of included studies	15
Total participants	1,532
Mean age (years)	46.8 (14.2)
Male sex	874 (57.0)
Female sex	658 (43.0)
BMI (kg/m²)	27.6 (4.9)
Follow-up duration (months)	14.5 (8.2)
Comorbidities	
Hypertension	412 (26.9)
Diabetes mellitus	335 (21.9)
Dyslipidemia	288 (18.8)
Obesity	241 (15.7)
Smoking history	468 (30.5)
Alcohol use	156 (10.2)
Cardiovascular disease	82 (5.4)
Chronic kidney disease	41 (2.7)
Previous gastrointestinal disease	198 (12.9)
Family history of gastrointestinal disease	113 (7.4)
Presenting symptoms	
Abdominal pain	951 (62.1)
Epigastric pain	724 (47.3)
Dysphagia	205 (13.4)
Nausea/vomiting	321 (21.0)
Gastrointestinal bleeding	95 (6.2)
Weight loss	108 (7.0)
Reflux symptoms	512 (33.4)
Heartburn	438 (28.6)
Endoscopic findings	
Normal findings	284 (18.5)
Gastritis	583 (38.1)
Erosive gastritis	192 (12.5)
Esophagitis	247 (16.1)
Gastric ulcer	101 (6.6)
Duodenal ulcer	78 (5.1)
Barrett's esophagus	35 (2.3)
Polyps	61 (4.0)
Intestinal metaplasia	49 (3.2)
Malignancy	18 (1.2)
Other findings	96 (6.3)

Table 2: Immunosuppressive medications used among the study population

Type of immunosuppressive medication	n (%)
5-Aminosalicylic acid	14 (14.3)
Azathioprine	13 (56.5)
Infliximab	15 (65.2)
Adalimumab	9 (39.1)
Ustekinumab	3 (13.0)
Vedolizumab	6 (26.1)
Methotrexate	3 (13.0)
Upadacitinib	1 (4.3)

Table 3: Antiviral Treatment, CMV-Related Complications and Hospital Course Among Participants

Variable	n (%)
Type of antiviral medication	
Valganciclovir alone	2 (8.7)
Ganciclovir alone	11 (47.8)
Ganciclovir followed by valganciclovir	11 (47.8)
IBD complications after CMV	
Intestinal perforation	1 (4.3)
Intestinal obstruction	1 (4.3)
Ileocecal resection	1 (4.3)
Stricture	4 (17.4)
Hospital course after CMV	
ICU admission	1 (4.3)
Ward admission	20 (87.0)

Table 4: Summary of the Patients' Characteristics and Their Clinical and Laboratory Findings

Characteristic	Number (%) or Mean ± SD
Total patients	23
Female	13 (56.5)
Male	10 (43.5)
Age (years)	38.7 ± 13.3
IBD type	
Ulcerative colitis (UC)	13 (56.5)
Crohn's disease (CD)	10 (43.5)
Site of involvement	
Pancolitis	13 (52.2)
Colonic with fistulizing ulcerative disease	3 (21.6)
Left-sided colon	2 (8.7)
Right-sided colon	1 (4.3)
Other sites	5 (21.7)
Duration of IBD at CMV diagnosis (months)	33.6 ± 7
Mayo endoscopic scores (severe)	
Score 3	7 patients
Score 2	4 patients
Score 1	1 patient
Missing data	12 patients
Smoking history	None
Endoscopic findings at CMV diagnosis	
Deep ulcer	16 (69.6)
Irregular ulcer	15 (65.2)
Aphthous ulcer	9 (39.1)
Cobblestone appearance	8 (34.8)
Luminal narrowing	5 (21.7)
Diffuse erythema	13 (56.5)
Mucosal friability	10 (43.5)
Mucopurulent exudate	4 (17.4)
Laboratory findings	
CMV IgG positive	18 (78.3)
CMV IgM positive	1 (4.3)
CMV DNA PCR positive	15 (65.2)
CMV immunohistochemistry positive	18 (78.3)
CMV inclusion bodies	6 (26.1)
Response to CMV diagnosis	
No antiviral treatment	4 (17.4)
Antiviral therapy administered	19 (82.6)
Surgical intervention required	4 (17.4)
Mortality	0 patients

Discussion:

This retrospective study provides valuable insight into clinical features, diagnostic approaches and outcomes of CMV colitis in adult IBD patients in a tertiary care center in Saudi Arabia. Our results show that CMV colitis occurs in about 7.7% of IBD patients, mostly in UC patients and in those with extensive colonic disease and is strongly associated with immunosuppressive therapy use. Importantly, tissue-based diagnostic modalities, particularly CMV PCR, were more sensitive than serological testing and early antiviral therapy was associated with high rates of clinical remission with no mortality or colectomy. The rate of CMV colitis in our IBD cohort (7.7%) is consistent with the published literature, which describes CMV detection rates of 4% to 36% in IBD patients, depending on disease severity, diagnostic methods used and patient selection criteria **[16]**. The predominance of females (56.5%) observed in our study differed from other reports with equal or male predominance **[17]**, which may be due to the demographic characteristics of the region or referral bias. The mean age at diagnosis of CMV (38.7 ± 14.3 years) is consistent with the age distribution of IBD patients and with previous studies demonstrating reactivation of CMV mainly in young to middle-aged adults **[18]**. The relatively short mean duration of IBD at diagnosis of CMV (11.6 ± 7.0 months) suggests that CMV reactivation can occur early in the course of disease, especially in patients who require early aggressive immunosuppression for severe or refractory disease.

The fact that UC was more frequently affected than CD (56.5% vs. 43.5%) is in line with the established literature, showing that UC patients have higher rates of reactivation of CMV, especially those with severe, extensive colitis **[19]**. The predominance of pancolitis (52.2%) further supports the association between extensive mucosal inflammation and risk of CMV reactivation. The high proportion of patients with steroid-refractory disease (26.1%) and on combination immunosuppressive therapy (60.9%) reflects the relationship between disease severity, intensive immunosuppression and CMV reactivation. This is in line with previous studies that identified steroid-refractory colitis as a major risk factor for CMV reactivation **[20]**. The most common endoscopic finding was deep ulceration (60.9%), which is typical for CMV colitis and has been associated with poor outcomes in some studies **[21]**. However, the lack of colectomy in our cohort indicates that early diagnosis and treatment may reduce the risk of progression to severe complications.Our study highlights important differences in the diagnostic yield of various CMV testing modalities. Tissue CMV PCR demonstrated the highest positivity rate (60.9%), significantly exceeding that of serum CMV PCR (21.7%), CMV IgM (4.3%) and even CMV immunohistochemistry (34.8%). These findings are consistent with emerging evidence suggesting that tissue-based molecular diagnostics are superior to serological and blood-based tests for diagnosing CMV colitis in IBD patients **[22]**. The low positivity rate of serum CMV PCR (21.7%) compared to tissue PCR (60.9%) suggests that CMV reactivation in IBD may be primarily localized to the colonic mucosa, with limited systemic viremia. This has important clinical implications, as reliance on serum-based testing alone may result in underdiagnosis of clinically significant CMV colitis. Our findings support current recommendations advocating for tissue-based diagnostics (PCR and/or immunohistochemistry) as the gold standard for diagnosing CMV colitis in IBD patients **[23]**. The high rate of CMV IgG seropositivity (69.6%) indicates previous exposure to CMV and is consistent with population seroprevalence data. However, low CMV IgM positivity (4.3%) confirms that most

cases represented viral reactivation rather than primary infection, as expected in immunocompromised IBD patients. The high rate of clinical remission after antiviral therapy (81.25% of treated patients) is encouraging and is consistent with meta-analyses showing clinical benefit of antiviral treatment in CMV-positive IBD patients **[24, 25]**. Our cohort did not experience any mortality or colectomy, which is particularly significant as CMV colitis has been linked to increased risks of colectomy and mortality in some studies **[26, 27]**. Our good results may be due to early diagnosis, early start of antiviral therapy and appropriate continuation of immunosuppressive therapy for underlying IBD. Interestingly, 30.4% of patients did not receive antiviral therapy and no adverse outcomes were found. These questions are of importance for the clinical relevance of detecting CMV and for the indications for antiviral therapy. The current evidence suggests that not all cases of CMV-positive IBD require antiviral therapy and treatment decision should be individualised according to the severity of disease, viral load and clinical context **[28]**. The "innocent bystander" hypothesis suggests that detection of CMV may be a marker of disease severity and not of pathogenic viral activity and that aggressive management of underlying IBD may be adequate in certain cases **[29]**. The best antiviral treatment regimen and duration for CMV colitis in IBD are still under active investigation. In our study, the main agents were ganciclovir and valganciclovir according to the current guidelines **[30]**. But new data indicate that shorter courses of therapy (14-21 days) may be as effective as long courses of therapy (≥3 weeks) in achieving clinical remission **[31]**. Large data collection from a well-defined IBD cohort - Inclusion of multiple diagnostic modalities for sensitivity comparison - Long study period (10 years) to capture temporal trends - Detailed documentation of immunosuppressive exposures and outcomes - Focus on a Middle Eastern population, addressing a gap in regional data. Retrospective design which has the potential for selection bias and missing data inherent in it - Single-center study may limit generalisability - Small sample size (n=23) may limit statistical power for subgroup analyses - Lack of quantitative CMV PCR data to define treatment thresholds - Mayo endoscopic subscores missing for ~50% of UC patients - Lack of standardisation of CMV testing and treatment decisions. - Few long-term follow-up data to evaluate recurrence rates and late complications - CMV cannot be definitively distinguished as a pathogenic driver versus innocent bystander. Future research should attempt to use Larger sample size, prospective, multi-center studies to confirm these results, Develop and validate quantitative CMV PCR thresholds for clinical management, conduct randomised controlled trials comparing antiviral therapy with conservative management in CMV positive IBD patients with different disease severities, study optimal antiviral regimens, dosing and duration of therapy, investigate virological and immunological host factors predicting risk of CMV reactivation and also long-term outcomes, including rates of CMV recurrence and impact on the course of IBD along with cost-effectiveness analyses of different diagnostic and therapeutic strategies

Conclusion:
CMV colitis is an important clinical consequence in IBD patients, particularly in those on immunosuppressive medication for moderate-to-severe disease. Early diagnosis and treatment with antivirals is associated with good clinical results including high rates of remission and avoidance of colectomy. However, not all patients with positive CMV need antiviral therapy and the decision to treat should be individualised according to the severity of disease, diagnostic findings and clinical situation.

Author contributions:
Conceptualization and methodology, Duaa Bakheet, Mohammed S. Alzahrani, Hadeel Altayib; data curation and formal analysis, Duaa Bakheet, Mohammed S. Alqarni, Ahmad Alsolmi, Marwan Alsehli, Mohammed Almutairi; investigation and resources, all authors; writing-original draft, Duaa Bakheet, Mohammed S. Alzahrani; writing-review and editing, all authors; supervision, project administration and funding acquisition, Mohammed S. Alzahrani, Hadeel Altayib. All authors have read and agreed to the published version of the manuscript.

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Institutional review board statement:
The study was conducted in accordance with the Declaration of Helsinki and approved by the Institutional Review Board of King Abdullah International Medical Research Center (KAIMRC), Jeddah, Saudi Arabia (Protocol Code: IRB/0977/24).

Informed consent statement:
Patient consent was waived due to the retrospective nature of the study and the use of anonymized data, in accordance with institutional guidelines and applicable regulations.

Data availability statement:
The data presented in this study are available on request from the corresponding author. The data are not publicly available due to privacy and ethical restrictions.

Conflicts of interest:
The authors declare no conflicts of interest.

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