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UNMASKING CYTOMEGALOVIRUS COLITIS: A CASE OF HAEMORRHAGIC COLITIS IN A PATIENT WITH CHRONIC MYELOID LEUKAEMIA ON DASATINIB

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ABSTRACT

Background: Tyrosine kinase inhibitors (TKIs) are the first-line treatment for chronic myeloid leukaemia (CML) with BCR-ABL1 translocation. Dasatinib, a second-generation TKI, is associated with a wide range of adverse effects such as myelosuppression, pleural effusion, diarrhoea, and haemorrhagic colitis in up to 40% of patients. Additionally, its off-target immunomodulatory effects on natural killer cells and T lymphocytes may lead to the reactivation of latent infections, including cytomegalovirus (CMV), a known cause of colitis.

Case report: We present a case of CMV-associated haemorrhagic colitis in a 69-year-old woman with CML in complete remission, who was being treated with dasatinib. She was admitted to our hospital with a 1-month history of generalized weakness, light-headedness, and intermittent haematochezia. On presentation, she was hypotensive and tachycardic due to haemorrhagic shock. Urgent colonoscopy revealed multiple small ulcers and erosions in the rectum, sigmoid, and transverse colon with signs of active bleeding, which were managed with haemostatic clips. Rectal biopsy initially showed mild, non-specific chronic inflammation. Dasatinib was discontinued due to suspected drug-related colitis. However, post-discharge immunohistochemistry revealed localized CMV inclusions, prompting readmission and initiation of valganciclovir therapy.

Conclusion: This case highlights the importance of considering opportunistic infections such as CMV in CML patients receiving TKIs. Given the overlapping clinical and endoscopic features of CMV and dasatinib-induced colitis, histopathological evaluation is essential for accurate diagnosis and timely management.

KEYWORDS

Chronic myelogenous leukaemia, haemorrhagic colitis, dasatinib, cytomegalovirus infection

LEARNING POINTS

- In immunosuppressed patients being treated with dasatinib, haemorrhagic colitis can mask an underlying infection.
- Tissue biopsy constitutes a key tool for promptly diagnosing cytomegalovirus colitis, enabling clinicians to start treatment early and thus prevent adverse outcomes.

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INTRODUCTION

The advent of tyrosine kinase inhibitors (TKIs) has significantly improved the prognosis of patients with chronic myelogenous leukaemia (CML) harbouring the BCR-ABL1 translocation, leading to increased overall survival and enhanced quality of life[1]. Second and third-generation TKIs, such as dasatinib and bosutinib, offer faster and deeper molecular responses than their first-generation counterpart (imatinib), and are now standard frontline therapies for CML[1].

Dasatinib is an adenosine triphosphate-competitive inhibitor targeting BCR-ABL, c-KIT, platelet-derived growth factor receptor (PDGFR), and SRC family kinases (SFKs)[1,2]. While effective, long-term use is associated with a range of adverse effects, including myelosuppression, pleural effusion, diarrhoea, liver enzyme abnormalities, and gastrointestinal (GI) bleeding in 17-40% of patients[1,3,4]. Among these haemorrhagic events, approximately 10% of cases require urgent intervention[1].

In vitro studies show that dasatinib exerts immunomodulatory effects by inhibiting T-cell proliferation and activation in a dose-dependent manner, impairing cell-mediated immune responses[1]. In turn, immunosuppression predisposes to the reactivation of latent viruses such as varicella-zoster virus, human herpesvirus 6, parvovirus B19, and cytomegalovirus (CMV)[2,4,5]. CMV-related haemorrhagic colitis can present with fever, diarrhoea, haematochezia, abdominal pain, and leucocytosis[6]. The diagnosis is often challenging, as the symptoms and endoscopic findings of CMV infection overlap with those of dasatinib-induced colitis[4,6,7]. In such cases, gastrointestinal biopsy with immunohistochemical (IHC) staining is crucial, particularly when serum polymerase chain reaction (PCR) testing is negative or equivocal.

We present a case of CMV-associated haemorrhagic colitis in a patient receiving long-term dasatinib therapy. This case illustrates the diagnostic challenge of differentiating CMV colitis from dasatinib-induced colitis and underscores the need for treatment guidelines.

CASE DESCRIPTION

A 69-year-old woman with CML in complete molecular remission, hypertension, and atrial fibrillation presented to the hospital with a 1-month history of intermittent haematochezia, light-headedness, and generalized weakness. She denied nausea, vomiting, hematemesis, chest pain, or syncope. Her medications included apixaban for atrial fibrillation (CHA_2DS_2-VASc score 3) and dasatinib, which she had taken for over 5 years. One week before admission, an outpatient colonoscopy revealed non-bleeding diverticula in the ascending colon, small internal haemorrhoids, and three small sessile polyps that were resected without complications.

On presentation, she was tachycardic (120 bpm) and hypotensive (69/50 mmHg), consistent with haemorrhagic shock. Physical examination revealed an irregular heart rhythm and a positive digital rectal exam for blood. She received intravenous fluids and two units of packed red blood cells (PRBCs), with subsequent hemodynamic stabilization. Both apixaban and dasatinib were discontinued.

Laboratory tests showed severe microcytic anaemia, elevated ferritin, and mildly prolonged coagulation parameters. White blood cell count was normal with mild monocytosis and immature blasts consistent with CML *(Table 1)*. Serologic tests for hepatitis B/C and human immunodeficiency virus were negative.

Computed tomography angiography demonstrated colonic diverticulosis without diverticulitis or abnormal mucosal enhancement. Emergency colonoscopy revealed multiple small ulcers and erosions (up to 1 mm) with active bleeding in the rectum, sigmoid, and transverse colon *(Fig. 1)*. Haemostasis was achieved with three clips, after which the bleeding subsided. The working diagnosis was haemorrhagic colitis secondary to dasatinib versus infectious colitis. Preliminary histopathology studies showed mild, nonspecific chronic inflammation of the intestinal mucosa with no viral inclusions *(Fig. 2)*. Due to clinical improvement, the patient was discharged after resuming dasatinib and apixaban.

One week later, the patient was readmitted following confirmation of CMV colitis on IHC staining of the rectal biopsy. Further testing showed elevated CMV-specific IgG and weakly positive CMV PCR (<200 copies/µl), while ophthalmologic evaluation ruled out CMV retinitis. The patient began intravenous valganciclovir for CMV-related colitis. Due to recurrent GI bleeding, she required transfusion of three more units of PRBCs, prompting discontinuation of dasatinib. She completed antiviral therapy without further bleeding and was transitioned to another TKI, remaining asymptomatic at 1-year follow-up.

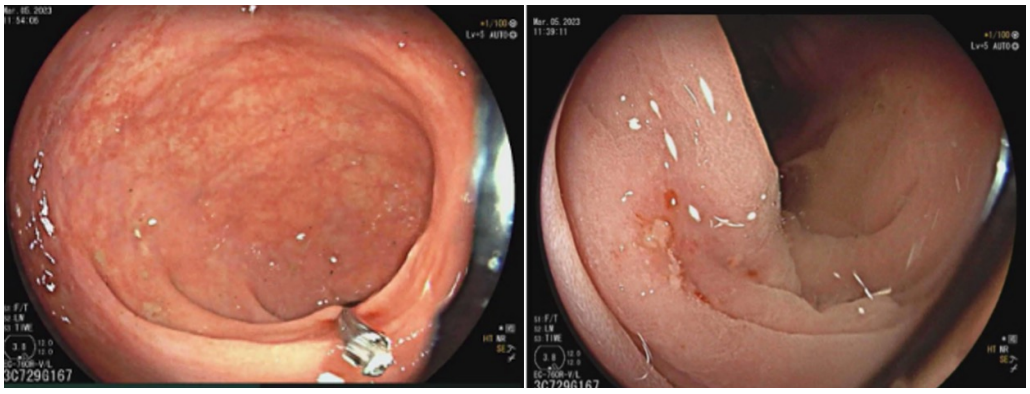


Figure 1. *Colonoscopy. Small ulcers/erosions (greatest diameter 1 mm) with stigmata of active bleeding in the rectum, sigmoid, and transverse colon.*

Parameter	On admission	Normal range
White blood cells (x10³/ul)	10.8	4-10
Neutrophils (%)	31.4	34-71.1
Eosinophils (%)	0.4	0.7-5.8
Monocytes (%)	10.1	4.7-12.5
Lymphocytes (%)	56.9	19.3-51.7
Basophils (%)	0.6	0.1-1.2
Platelets (x10³/ul)	214	150-450
Haemoglobin (g/dl)	6.4	11.2-15.7
Mean corpuscular volume (fl)	97.2	79.4-94.8
Red cell distribution width (%)	17.4	11.7-14.4
Iron (µg/dl)	26	50-170
TIBC (µg/dl)	103.6	250-400
Ferritin (ng/dl)	115	12-150
PT (sec)	11.5	9.1-11.7
PTTa (sec)	25.9	23.2-31.6
INR (ratio)	1.1	0.9-1.1
Total bilirubin (mg/dl)	0.1	0.1-1.2
Direct bilirubin (mg/dl)	0.1	0-0.3
Serum albumin (g/dl)	2.3	3.8-5
Total protein (g/dl)	4.5	6-8
Alkaline phosphatase (U/l)	75	38-126
Alanine transferase (U/l)	14	4-36
Aspartate transferase (U/l)	37	8-33
Creatinine (mg/dl)	2.4	0.6-1.2
HIV	negative	non-reactive
CMV quantitative DNA PCR (IU/ml)	200	non-reactive
HbsAg	negative	non-reactive
HbsAB (IU/ml)	0.96	0-12

Abbreviations: TIBC, total iron binding capacity; PT, prothrombin time; PTTa, activated thromboplastin time; INR, international normalized ratio; HIV, human immunodeficiency virus; CMV, cytomegalovirus; DNA, deoxyribonucleic acid; PCR, polymerase chain reaction; HbsAg, hepatitis B surface antigen, HbsAB, hepatitis B surface antibody.

Table 1. Laboratory investigations of the patient.

DISCUSSION

Dasatinib, a potent second-generation TKI, has markedly improved outcomes for patients with CML resistant or intolerant to imatinib[1]. However, adverse events frequently necessitate dose adjustments or discontinuation[3]. Gastrointestinal side effects, including nausea, diarrhoea, abdominal pain, and bleeding, occur in 30–50% of patients[3]. Bleeding events with dasatinib primarily affect the GI tract and are more frequently observed in patients with advanced-phase CML, blast-phase disease, or thrombocytopenia[1,8]. Experimental data suggest that dasatinib interferes with platelet aggregation and activation by inhibiting tyrosine kinase-dependent pathways and impairs intramedullary thrombopoiesis[1]. It also exerts a reversible, dose-dependent anticoagulant effect, increasing haemorrhagic risk[1]. In our case report, the patient lacked conventional risk factors such as thrombocytopenia or disease progression. In the end, bleeding was attributed to CMV colitis, likely exacerbated by concurrent apixaban use. Platelet dysfunction from dasatinib could not be excluded, as platelet function tests (PFA-100 system or platelet aggregometry) were not performed.

Dasatinib is also known to exert immunomodulatory effects by inhibiting SRC (SFK) family kinases[1]. Inhibition of the SFK pathway leads to reduced regulatory T-cell populations, impairing tolerance to commensal GI flora, which contributes to the onset of colitis[1]. Moreover, dasatinib affects the activity of natural killer (NK) cells and impairs T-cell activation, proliferation, and memory function in a dose-dependent manner[1,9]. A study demonstrated that patients with prior CMV infection who are treated with dasatinib exhibit an expansion of large granular lymphocytes, comprising NK cells and $CD8^+$ T lymphocytes[4]. In this context, Dasatinib seems to promote CMV reactivation and NK cell activity against the virus, enhancing anti-tumoral effects but also treatment-related adverse reactions[4]. Although the exact mechanism remains unclear, dasatinib has been implicated in herpesvirus reactivation, especially in patients receiving high doses for more than 3 months[2,10].

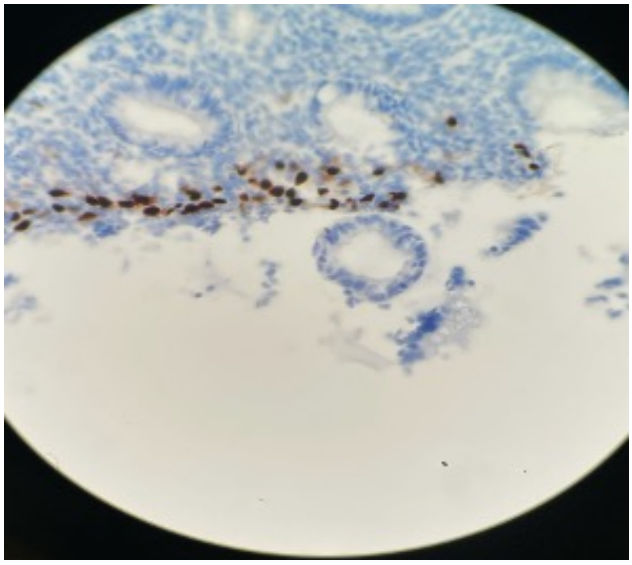


Figure 2. Rectal mucosa biopsy. Non-dysplastic colonic mucosa with mild non-specific chronic inflammation of the lamina propria with rare eosinophilic aggregates and neutrophils. Negative for granuloma, cryptitis, or crypt abscess. Negative for intracellular apoptosis or viral inclusions. Immunohistochemical analysis was positive for cytomegalovirus.

Our patient developed CMV colitis despite low-dose dasatinib (100 mg daily) but had additional risk factors, including recent corticosteroid use (10-day dexamethasone course for coronavirus disease 2019 pneumonia).

The overlapping clinical and endoscopic features of CMV and dasatinib-induced colitis complicate diagnosis[4,6,7]. Colonoscopy often reveals mucosal oedema, ulcerations, or friability, while histology may show nonspecific chronic inflammation indistinguishable between the two aetiologies[4,7]. In our case, colonoscopy revealed punched-out ulcerations with oedematous mucosa, and histopathology showed mild chronic inflammation without viral inclusions. Diagnosis required IHC confirmation and viral PCR detection. Notably, the patient had undergone a colonoscopy just 1 week before admission, which showed no signs of inflammation or bleeding, highlighting the quick progression of the disease. This raises the question of whether CMV screening should be considered in patients receiving dasatinib who present with early symptoms of colitis.

Early recognition and treatment of CMV infection in immunocompromised patients are essential, as CMV colitis, pneumonitis, and viremia can be life-threatening[5,6]. However, no guidelines exist for CMV screening or management in CML patients outside the post-transplant setting[5]. Consequently, therapeutic decisions are largely guided by clinician experience and isolated case reports, with limited data on outcomes or mortality.

Evidence from small case series remains heterogeneous. In one report (on 7 cases), 2 patients with dasatinib-associated CMV colitis had symptom recurrence without antiviral therapy, suggesting that ganciclovir reduces symptom duration and improves outcomes[7]. Conversely, other studies describe persistent or relapsing symptoms despite antivirals, necessitating dasatinib discontinuation[4,7]. Choi et al. described 5 cases of CMV colitis in patients treated with dasatinib for CML or Philadelphia chromosome-positive acute lymphoblastic leukaemia, four of whom developed relapsing haemorrhagic colitis[7]. Notably, the only patient who remained relapse-free received a prolonged 19-day course of ganciclovir and suspended dasatinib at symptom onset. Among the remaining four, dasatinib was continued during antiviral therapy in 2 patients and discontinued after symptom persistence or recurrence in the other two. All patients required antiviral treatment for clinical improvement, and 60% were able to resume dasatinib after completing therapy[7]. Four patients had a favourable long-term outcome, whilst one succumbed to refractory leukaemia 11 months later[7].

Consistent with most cases reported by Choi et al., our patient experienced recurrent GI bleeding during valganciclovir therapy and improved only after dasatinib discontinuation, ultimately achieving complete recovery. These cases emphasize the complexity of managing CMV colitis in patients receiving dasatinib and the substantial risk of relapse during or after treatment. Although spontaneous resolution without antiviral therapy has been reported in asymptomatic patients with negative CMV PCR[4,5], current evidence seems to favour temporary dasatinib discontinuation at symptom onset and reintroduction after antiviral completion.

CONCLUSION

Diagnosing CMV colitis in patients with CML undergoing dasatinib treatment is challenging due to its nonspecific clinical presentation and the overlapping endoscopic and histopathological features shared with drug-induced colitis. Definitive diagnosis relies on tissue biopsy coupled with targeted immunohistochemical staining. Given the paucity of data, there is a pressing need for evidence-based screening and treatment guidelines tailored to this population to determine which patients would benefit from antiviral treatment and continuation of dasatinib.

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