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Cytomegalovirus Colitis in a Patient Living with HIV: Review of Pathogenic Mechanisms, Diagnosis, and Therapeutic Approach

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Abstract

Cytomegalovirus (CMV) colitis is a serious and potentially fatal complication in patients living with human immunodeficiency virus (HIV), particularly those with severe immunosuppression. CMV, a virus belonging to the Herpesviridae family, takes advantage of immunosuppression to reactivate, leading to various clinical manifestations, including colitis. We report the clinical case of an HIV-positive patient with CMV colitis confirmed by colonic biopsy. This article explores the epidemiological, pathophysiological, clinical, diagnostic and therapeutic aspects of CMV colitis in HIV-positive patients, based on bibliographical references.

Keywords: *co-infections, cytomegalovirus, ganciclovir, people living with HIV.*

Introduction

Cytomegalovirus (CMV) is a virus of the Herpes viridae family that infects a large proportion of the world's population [1]; it is a common opportunistic pathogen in immunocompromised patients, particularly those infected with HIV. Before the advent of highly active antiretroviral therapy (HAART), CMV colitis was a major cause of morbidity and mortality in AIDS patients [2]. Although the incidence has decreased with the widespread use of HAART, this infection remains a significant clinical concern in untreated patients or those with advanced immunodeficiency [3]. Prevention strategies, such as isolation and regular virological monitoring, are crucial to limiting the impact of this infection [4].

Clinical case

Mr A E, aged 34, Moroccan nationality, married and father of a 6-month-old infant, known asthma sufferer for 10 years, poorly managed, smoker, cannabis and cocaine user, and occasional alcohol consumer.

The history of the illness dates back to one and a half months before his admission, with the onset of asthenia, unexplained weight loss, night sweats, complicated by dyspnoea on exertion and then at rest, retrosternal chest pain, abdominal pain and watery diarrhoea with 4 bowel movements per day, accompanied by unexplained fever.

The patient was admitted to the infectious diseases ward for specialised care, the patient was conscious with a Glasgow Coma Scale score of 15/15, feverish at 39.6°C, polypnoea at 24, oxygen saturation in free air 94%, tachycardia at 98, weight: 55 kg, BMI: 18, generalised cutaneous and mucosal jaundice, and presence of oral thrush.

The Chest CT scan showed multiple retractile foci of condensation in the bilateral basal regions, ground-glass opacities, severe diffuse bilateral interstitial involvement, and multiple septal and non-septal thickenings. The T-A-P scan showed bilateral apical nodular ground-glass opacities, a right basal condensation focus, discrete diffuse rectoanal thickening, and regular left parietal thickening of the wall of the last ileal loop. The brain CT scan showed right maxillary sinusitis and a posterior sub-tentorial extra-axial formation opposite the right cerebellar hemisphere with fluid density consistent with an arachnoid cyst.

Positive PCR for Pneumocystis jirovecii, 3 negative sputum BK tests, positive HIV serology with HIV viral load at 6,074,428 copies/ml, negative serology for hepatitis and syphilis. Stool culture: negative, 3 negative EPS tests, calprotectin: 100, urine culture: sterile, complete blood count: HB: 12.7 GB: 13,560 PNN: 11,470 LT: 1,340 PLT: 60,000 Reticulocytes: 47,200 TP low at 38% Factor V at 150%, sodium: 129 potassium: 3.9 creatinine: 8.5 Urea: 0.33 Hepatic cytolysis with cholestasis ASAT: 451 ALAT: 188 PAL: 961 GGT: 431 Bt: 88.3 Bc: 46.4 Bl: 41.9, lipasaemia: 3 CRP: 199. Cryptococcosis antigen in blood negative, toxoplasmosis serology negative. Colonoscopy was performed showing ulcerative ileitis with colic haemorrhagic suffusions, biopsy pathology: CMV inclusions, CMV PCR in colonic biopsy 765 copies/ml and CMV PCR in blood: 22,378 copies/ml, normal fundus, herpes virus I and II serology IgG positive and IgM negative, negative autoimmune liver profile, EBV IgG positive, IgM negative, blood cultures on standard and sterile fungal media.

The diagnoses of confirmed CMV colitis and confirmed pulmonary pneumocystosis were retained, and the patient was placed on Ganciclovir 5mg/kg twice daily, and subsequently placed on Rovalcyte 900 mg twice daily for 21 days after discontinuation of Ganciclovir. He was also given cotrimoxazole and oral corticosteroid therapy for 21 days.

The patient's condition improved, with the disappearance of digestive, neurological and respiratory symptoms and regression of cutaneous and mucosal jaundice. Follow-up tests showed regression of cytolysis and hepatic cholestasis. He was discharged on antiretroviral treatment with Tenofovir/Lamivudine/Dolutegravir.

Discussion

Cytomegalovirus (CMV) colitis is a serious gastrointestinal complication in immunocompromised patients, particularly those living with human immunodeficiency virus (HIV). This opportunistic infection occurs mainly in patients with CD4 counts below 50 cells/µL [5]. Low CD4 counts and high viral loads (VL) are risk factors for CMV infection in HIV-positive individuals.

CMV is a ubiquitous virus. In HIV-positive individuals, its prevalence increases with disease progression, reaching up to 90% in those with CD4 counts below 50 cells/µL [6]. CMV colitis is one of the most common manifestations of CMV disease in these patients, accounting for approximately 10-15% of cases of invasive CMV disease [3][7].

CMV is transmitted through direct contact with infected bodily fluids. After primary infection, the virus establishes latency in endothelial cells and monocytes. In immunocompromised patients, CMV reactivation leads to active viral replication, causing direct tissue damage and an exacerbated inflammatory response [2]. In the gastrointestinal tract, CMV infects endothelial and epithelial cells, leading to necrosis, ulceration and inflammation of the colonic mucosa, intestinal inflammation, mucosal necrosis and ulcerations that can lead to gastrointestinal haemorrhage and perforation [8]. The pathogenesis is based on direct damage to intestinal epithelial cells and a deficient immune response, thereby promoting disease progression [9].

The symptoms of CMV colitis are often non-specific and include abdominal pain, diarrhoea (sometimes bloody), weight loss, fever and fatigue. In severe cases, complications such as intestinal perforations, massive haemorrhages or toxic megacolon may occur [3].

The diagnosis of CMV colitis is based on a combination of clinical, endoscopic, histopathological and virological criteria. Endoscopy with biopsy is the gold standard method. Endoscopy usually reveals deep mucosal ulcerations, erosions and areas of necrosis, mainly in the right colon [2]. Biopsies should be analysed for the presence of giant cells with typical intranuclear inclusions (‘owl's eyes’) and/or viral antigens by immunohistochemistry [10]. Quantitative PCR on tissue or blood can also be used to confirm active infection [6][11]. It should also be noted that viraemia in the blood is not always associated with visceral involvement and vice versa [12].

Treatment of CMV colitis in HIV-positive individuals is based on the use of specific antiviral drugs. intravenous ganciclovir (5 mg/kg every 12 h) is the standard treatment, followed by oral valganciclovir for a period of 3 to 6 weeks [13]. In cases of resistance or intolerance to (val)ganciclovir, foscarnet or cidofovir may be used. The duration of treatment is generally 3 to 6 weeks, followed by maintenance treatment to prevent relapse in patients whose CD4 count remains low [3].

Ganciclovir and foscarnet, viral DNA polymerase inhibitors, have shown indisputable, albeit incomplete, efficacy in the treatment of retinitis, gastrointestinal infections and CMV pneumonia. Both drugs have a short half-life and low oral bioavailability, requiring exclusive parenteral use. Their systemic toxicity, particularly haematological toxicity in the case of ganciclovir and renal toxicity in the case of foscarnet, limits the dosage administered, resulting in residual serum concentrations slightly above the 50% inhibitory doses in vitro for CMV [14].

Recent progress has been made in the development of new antiviral molecules (in particular letermovir and

brincidofovir). These could be particularly interesting due to their tolerance profile and their preserved activity against certain CMVs that are resistant to current molecules. Antiviral adoptive cell immunotherapy has proven effective in treating CMV infections after hematopoietic stem cell transplantation (HSCT) [15]. Initiating or optimising antiretroviral therapy (ART) is essential for restoring immunity and preventing recurrence [2].

Primary prevention of CMV infection involves a series of diagnostic tests. The results of these tests determine the nature of follow-up and the implementation of pre-emptive treatment with valganciclovir. For example, serological testing for anti-CMV antibodies is routine for HIV-positive patients if their CD4 count is below 100/mm^3. If this antibody test is positive, CMV gene amplification ('CMV PCR') is performed. A positive CMV PCR result requires a fundus examination. In the absence of a positive PCR result, prevention relies on the rapid initiation of antiretroviral treatment [16][17].

The prognosis for CMV colitis depends on how quickly it is diagnosed and treatment is initiated. With appropriate antiviral treatment and immune restoration through ART, the majority of patients show significant clinical improvement. However, without treatment, mortality can reach 30–40% [6]. In France, the 3-year survival rate after CMV infection was estimated at 64% in the period 2001-2003 [7].

Conclusion

CMV colitis in HIV-positive individuals is a serious condition that requires rapid diagnosis and early management to improve prognosis. Antiviral treatment, combined with immune restoration through ART, is the optimal therapeutic strategy. Close monitoring is essential to avoid complications and recurrences. Continued efforts are needed to raise awareness among clinicians about this condition and optimise the management of at-risk patients.

Compliance with ethical standards

Disclosure of conflict of interest No conflict of interest to be disclosed.

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