Sir,

Cytomegalovirus (CMV), a beta-herpes virus, is an important opportunistic pathogen in the setting of human immunodeficiency virus (HIV) infection. Mucocutaneous manifestations of CMV are rare with their presence heralding the development of systemic infection. Presence of persistent anogenital ulceration in HIV infection may be a marker of CMV in the lesion. CMV and Herpes simplex virus (HSV) may co-infect the same ulcer and it is widely believed that HSV is the main pathogenic organism while CMV plays a little role.

A 35-year-old woman, a case of HIV infection with disseminated tuberculosis on antiretroviral therapy (tenofovir, lamivudine and efavirenz) since four months and on antitubercular therapy (ATT) since last two months, presented to dermatology outpatient department with a non-healing painful genital ulcer over the vulva from past six weeks, along with a rapid increase in its size over the last two weeks. She had been prescribed to take Acyclovir tablet for past two months for a positive HSV serology. In spite of treatment, the ulcer continued to progress in size involving the perineum extending up to the gluteal region. At the same time, the patient also developed diarrhea for two weeks which did not respond to antidiarrheal therapy. Moreover, as enzymes in her liver continued to increase, antitubercular treatment was also withheld considering ATT induced hepatotoxicity. There was no history of breathlessness, cough, chest pain, or any ocular complaints.

General examination revealed an emaciated female with bilateral inguinal lymph nodes, enlarged and tender. Dermatological examination showed a well-defined ulcer with a necrotic yellow exudate over labia majora and minora involving bilateral crural fold and extending up to the perineum [Figure 1]. Investigation findings were Hb- 8.5 gm%; positive PCR (polymerase chain reaction) for CMV at 20,000 copies/mL; IgG positivity for HSV-2 (herpes simplex virus-2). CD4 count was 25 cells/µL; and an HIV viral load of 6,00,000 copies/mL was detected. Liver enzymes were elevated with SGOT/SGPT at 657/432 IU/L. The Venereal Disease Research Laboratory (VDRL) test and Treponema pallidum hemagglutination assay (TPHA) for syphilis were negative while scrapings from the perigenital ulcer were positive by PCR for HSV-2 and CMV.

Due to lack of consent from the patient, histopathological examination of the ulcer could not be carried out.

In view of persistent non-healing genital ulcer along with symptoms of colitis and features of hepatitis in the patient, a possibility of disseminated CMV infection was considered.

The patient was treated with Tab. Valganciclovir 900 mg once a day for 21 days in continuation with Tab. Acyclovir 400 mg five times a day. Within seven days of starting the treatment, the patient showed good response in the form of decrease in pain and drying up of the exudate. The ulcer completely healed over a course of three weeks after which the patient was continued on Acyclovir suppressive prophylaxis in the form of Tab. Acyclovir 400 mg twice a day [Figure 2]. After treatment with Valganciclovir, her symptoms of colitis and deranged liver enzymes also got resolved indicating disseminated CMV infection responding to treatment. ATT was restarted with the patient tolerating it well.

Differential diagnosis of genital ulcers in an immunocompromised person includes typical as well as atypical varied presentations of primary syphilis, chancroid and genital herpes. Although rare, CMV as a cause of persistent perigenital ulcer in an immunocompromised...
person is now known and numerous cases are being reported.

Primary or recurrent CMV infections in immunocompromised patients either remain completely asymptomatic or as disseminated disease. Disseminated CMV manifests with conditions such as fever, leukopenia, hepatitis, pneumonitis, esophagitis, gastritis, colitis, and retinitis.[2]

Mucocutaneous lesions due to CMV infection are very rare in contrast with high frequency of ocular and visceral involvement which occurs when the CD4+ lymphocyte count is below 50 cells/mm³. Cutaneous CMV infection occurs by reactivation of the latent virus and hematogenous dissemination, or by autoinoculation following viral shedding in body fluids.[3]

Cutaneous presentations of CMV include generalized maculopapular rashes, papules or localized painful ulcerative lesions, commonly located in the perigenital or perianal region, often coexisting with HSV.[3,4] CMV is generally found to co-infect herpetic ulcers and the presence of persistent anogenital ulcers suspects the presence of CMV in these lesionslike in the case of our patient.[4] CMV plays an important role both in the origin as well as the chronicity of these ulcers. It is speculated that in severely immunocompromised patients CMV viremia can cause cutaneous vasculitis which can lead to localized vascular damage and ulceration.[2] Gouveia et al.[5] reported a case of HSV and CMV co-infection, as an exuberant genital ulcer in a woman infected with HIV.

The pathogenesis of CMV in co-infected cutaneous lesions is controversial. Some clinicians have concluded that CMV does not play a significant pathogenic role in these lesions as it is often cultured with other infectious agents with a recognized pathogenic role and that CMV may appear in otherwise healthy skin.[1] However, others maintain that CMV does have a pathogenic role with case reports stating improvement of such lesions only with anti-CMV medications.

The early detection of CMV has high prognostic value because its presence in skin or mucosa indicates disseminated CMV infection which should be treated as early as possible.

Drugs including ganciclovir, valganciclovir, foscarnet, and cidofovir have been approved for the treatment of CMV.[6]

In conclusion, our patient with a previously non healing genital ulcer responded very well to a three week course of oral Valganciclovir in the form of complete resolution of genital ulcer and with an improvement in systemic symptoms of colitis and deranged liver enzymes thereby suggesting CMV as a causal pathogenic agent as a cause of non-healing genital ulcer and the importance of early institution of treatment for treating other manifestations of disseminated CMV infection.

Hence, in an immunocompromised patient, with perianal or perigenital lesions, it is important to consider CMV as a causative agent for lesions recalcitrant to conventional therapy.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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**Conflicts of interest**

There are no conflicts of interest.

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