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Cytomegalovirus Meningoencephalitis in an Immunocompromised Patient: Case Report and Review of Literature

Khairoun Saad[1]*, Elkabbaj Abdelmajid[1], Benhlima Abdelkader[1], Elhamzaoui Hamza[1], Alilou Mustapha[1]

[1]Emergency Department, IBN SINA Hospital, Faculty of Medicine and Pharmacy, University Mohammed V, Rabat, Morocco

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***Corresponding author:** Khairoun Saad
Emergency Department, IBN SINA Hospital, Faculty of Medicine and Pharmacy, University Mohammed V, Rabat, Morocco

Abstract **Case Report**

Cytomegalovirus (CMV), is a common virus that stays dormant after the initial infection but can reactivate in immunocompromised individuals, such as those with AIDS, leading to severe conditions. CMV affects 30% to 100% of people worldwide. CMV encephalitis is seen in at least 6% of untreated advanced HIV cases. This article presents a 42-year-old HIV-positive woman who developed CMV meningoencephalitis, resulting in septic shock. CMV can cause cytomegalia and various diseases including pneumonia, gastrointestinal disease, hepatitis, and, rarely, retinitis and meningitis/meningoencephalitis. Diagnosis typically involves detecting CMV DNA in the cerebrospinal fluid (CSF) through PCR.CMV causes diverse central nervous system lesions, but MRI often fails to detect these infections, as seen in this case. Treatment involves Ganciclovir or Foscarnet, starting with induction doses for at least two weeks, followed by maintenance doses for three to four weeks until symptoms resolve and viral load is negative. In immunosuppressed patients, ongoing maintenance or close monitoring is necessary.

Keywords: Cytomegalovirus (CMV), Meningoencephalitis, HIV/AIDS, Immunocompromised, Ganciclovir.

INTRODUCTION

Cytomegalovirus (CMV), also known as human herpesvirus 5, is a ubiquitous virus that remains latent after primary infection [1]. CMV commonly causes lifelong latent infection but may reactivate in immunocompromised individuals [2]. Reactivation of CMV can lead to potentially life-threatening disease in patients with acquired immunodeficiency syndrome (AIDS) [1]. This virus is found worldwide, and its seroprevalence varies geographically, ranging from 30% to 100%. CMV encephalitis occurs in at least 6% of untreated patients with advanced HIV disease [3]. We present the case of a 42-year-old HIV-positive female who developed CMV meningoencephalitis, leading to septic shock.

CASE REPORT

A 42-year-old female presented to our hospital's emergency department with reduced consciousness and a three-day history of fever, along with speech and balance difficulties. The patient had a history of pulmonary tuberculosis treated at the age of 27, and an HIV infection diagnosed in two month ago, for which she is receiving antiretroviral treatment.

She had a Glasgow Coma Score of 9, primarily due to impaired verbal and motor responses, with equal and reactive pupils, no sensory-motor deficits or focal signs, and no signs of convulsions, but with nuchal rigidity and positive Brudzinski and Kernig signs. On admission, her blood pressure was 124/68 mm Hg, pulse rate 96beats/min, body temperature 39.1°C, and capillary blood glucose level 1.24 g/L. An initial pulmonary examination revealed coarse crackles on auscultation, a respiratory rate of 28/min, and an oxygen saturation of 84%.

After stabilization, the patient was intubated and sedated. Computed tomography (CT) scans were performed; the brain scan showed no anomalies, while the thoracic scan revealed diffuse infiltrative pneumonia. Laboratory results were as follows: white blood cell count was 5600/mm3 with a lymphocyte count of 190/mm3, C-reactive protein 140 g/l, procalcitonine 8µg/l, coagulation parameters, and liver and kidney function tests were within normal ranges. Blood CMV polymerase chain reaction (PCR) was positive. The HIV viral load was very high over 8,000,000 copies. A lumbar puncture was performed, and the cerebrospinal fluid (CSF) was clear, with a high protein concentration (over

13.35 g/L), glycorrhachia was normal (1.07 g/L), and 12/mm3 white blood cells (WBC) with 100% lymphocytes. Tests for other viruses such as varicella-zoster and herpes simplex, as well as for Cryptococcus neoformans, were negative in the CSF analysis. Other investigations, including tests for Hepatitis B, Hepatitis C, GeneXpert for BK, Pneumocystis jirovecii and other fungal infection were negative.

The patient was administered ganciclovir 250 mg twice a day at a dose of 5 mg/kg via slow infusion over one hour in 100 cc of saline solution, along with ceftriaxone 2 g/day and ciprofloxacin for her pneumonia, while awaiting the results of the Protected Specimen Brush (PSB) test. Antiretroviral treatment was continued. Despite this treatment, the patient's condition gradually worsened, and she developed refractory septic shock, passing away two days after her admission.

DISCUSSION

Human cytomegalovirus, a herpesvirus first isolated in 1956, is known for causing cytomegalia and forming intranuclear and cytoplasmic inclusion bodies [4]. CMV can lead to various diseases such as pneumonia, gastrointestinal disease, hepatitis, and more rarely, retinitis and meningitis/meningoencephalitis [1]. These conditions are potentially fatal and cause significant morbidity and mortality, particularly in individuals with compromised cellular immunity [1]. CMV meningitis/meningoencephalitis has been predominantly reported in AIDS patients [5].

CSF may sometimes show neutrophilic pleocytosis, hypoglycorrhachia, and increased protein levels. In our case, we found elevated protein levels and normoglycorrhachia. Diagnosis is usually confirmed by viral PCR, which has become a useful tool for recognizing CMV encephalitis [6], or by detecting CMV proteins (pp65) or nucleic acid through polymerase chain reaction from blood or other clinical samples [7]. In this case, CMV meningoencephalitis was diagnosed based on the presence of abundant viral CMV DNA in the CSF. Detectable CMV viremia is associated with a approximately 60% greater risk of death in persons with HIV-associated meningitis, Skipper et al demonstrated that in a recent study.

In radiology, CMV causes various CNS lesions, with periventricular lesions, ependymitis, and ventriculitis traditionally described [4]. The sensitivity of MRI in detecting CMV CNS infection remains low, and infections may be missed with imaging alone [8], as was the case in this report where the brain scan showed no anomalies.

Treatment guidelines for CMV encephalitis in immunocompromised individuals recommend combination therapy with Ganciclovir or Foscarnet [9]. Ganciclovir is an acyclic analogue of the nucleoside guanosine capable of inhibiting all herpes viruses, especially CMV [3]. Foscarnet inhibits the activity of viral DNA polymerase by binding to the pyrophosphate-binding site and blocking the cleavage of pyrophosphate from the terminal nucleoside triphosphate added to the growing DNA chain [9]. Consequently, Foscarnet can be used against pUL kinase variants that confer resistance to Ganciclovir [8]. Induction doses for at least 2 weeks are generally recommended, followed by maintenance dosing for another 3-4 weeks [6]. Treatment should be continued until symptoms resolve and viral load becomes negative. In patients with ongoing immunosuppression, continued maintenance or close virologic surveillance is recommended, and additional treatment courses may be necessary [6]. Ventilator-associated pneumonia and pleural effusion can complicate and prolong the recovery process[3], as was the case for our patient.

CONCLUSION

In conclusion, CMV meningoencephalitis should be promptly considered in HIV immunocompromised patients presenting with neurological symptoms. PCR is the most important diagnostic test. Ganciclovir is the first-line treatment for CMV meningoencephalitis. Unfortunately, the mortality remains very high. Therefore, we recommend early recognition and aggressive treatment approaches.

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