INTRODUCTION

CMV infections in immunocompromised patients (especially AIDS and transplant recipients) can cause important clinical conditions. Reactivation of latent CMV is the main pathophysiological mechanism and reinfection with a new strain has been reported in such situations (Karton 2013). On the other hand, primary CMV infection is mostly seen in immunocompetent hosts. While the asymptomatic infection and mononucleosis syndrome are more prevalent, organ specific involvement are also reported in the immunocompetent hosts. In the current review of the literature, 290 immunocompetent adults with CMV infection were analyzed and 56 central nervous system infections were identified (Rafailidis et al. 2008).

Neurological manifestations of these patients related with brain (encephalitis, meningoencephalitis) and spinal (myelitis, meningoradiculopathy) involvements. Although quite adequate in terms of the number of cases, this review is unsatisfactory as contains no the details of the clinical pictures (Wingerchuk and Weinshenker 2013). Transverse myelitis is another neurological deficit that has been described very rarely in CMV infection patients.

CASE SUMMARY

A 62-year-old male patient presented to our clinic with complaints of visual disturbances, listlessness on the lateral part of the right limb, muscle weakness and a short lasting complaint of dysarthria. He had visited an outside clinic setting with similar symptoms 2 weeks prior to presentation and was diagnosed with idiopathic transverse myelitis for which he was given methyl prednisone 250 mg pulse for 10 days. His visual symptoms started while on the corticosteroid treatment and observed deterioration in muscle weakness. Dilated fundoscopic exam showed moderate vitritis, vasculitis, and extensive perivascular retinitis with areas of retinal hemorrhage and whitening bilaterally extending from the peripheral retina to the posterior pole. A final diagnosis of Transverse myelitis was made and the patient was started on parenteral ganciclovir (5 mg/kg/day) and methylprednisolone 1000 mg/kg/day) and methylprednisolone 1000 mg pulse 1x1/3 days. Thrombocytopenia resolved on the 21st day of follow-up, and there was a regression in the retinal areas with necrosis but both eyes revealed proliferative vitreoretinopathy leading to tractional retinal detachments. Bilateral vitrectomy surgery, left eye priority, was recommended but the patient refused the operation. The treatment was continued and eight months after treatment, partial improvement in his conditions was observed. He could stand on his own.

DISCUSSION

Acute retinal necrosis (ARN) is a clinical syndrome characterized by vitritis, severe exudative vasculitis of the retina and the choroid, and a full thickness, confluent, necrotizing retinitis that affect primarily peripheral retina, caused by the Herpesviridae family (Duker and Blumenkranz 1991). In rare instances, however, cytomegalovirus has been reported to cause ARN in healthy adults (Akpek et al. 1999).

Although our patient was a healthy adult he had a history of steroid therapy. This situation may have predisposed him to an aggressive disease course.

REFERENCES