Myelin oligodendrocyte glycoprotein antibody-associated disease – a rare cause of bilateral acute visual loss: a case report

Chiam Lee Yan[1,2], Mohd Khairul Abdul Majid[1], Kamalden Tengku Ain[2], Zaidah Mohd Kasim[1]

[1]Department of Ophthalmology, Hospital Sultan Idris Shah, Serdang, Selangor, Malaysia, [2]UM Eye Research Centre, Department of Ophthalmology, Faculty of Medicine, Universiti Malaya, Kuala Lumpur, Malaysia

ABSTRACT

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is an inflammatory disorder of the nervous system characterised by immune-mediated demyelination predominantly targeting the optic nerves, brain and spinal cord. Patients typically present with sudden severe visual loss, altered mental status and focal neurological features. We report an uncommon case of MOGAD presenting as acute bilateral visual loss. A 30-year-old Burmese male presented with a day history of acute painless bilateral blurring vision. There were no other associated ocular or neurological symptoms. At presentation, there was no light perception in the right eye with relative afferent pupillary defect. The left eye was seeing 6/24 at presentation however deteriorated to no light perception within the next 2 days. Both optic discs were hyperaemic. Serum and cerebrospinal fluid (CSF) infective markers were negative. Magnetic resonance imaging of the orbit revealed bilateral optic nerve peri-neuritis with right retrobulbar optic neuritis. The patient was started on oral followed by intravenous steroid treatment for a total of 8 days with slow response. Vision improved to hand movement over right eye and counting finger over left eye. The patient was tested positive for serum anti-MOGAD antibodies and subsequently underwent plasma exchange. Young patients with acute rapidly progressive bilateral visual loss should be investigated for uncommon inflammatory causes such as MOGAD. Anti-MOG antibody detection in serum or CSF is diagnostic. Early treatment with high dose corticosteroids followed by maintenance is crucial to prevent relapse and residual neurological disability.