Acute hypertensive uveitis as the first presentation of multiple sclerosis

Melissa Kate Shields[1,2,3], Sumu Simon[1,2,3], WengOnn Chan[1,2,3], Jagjit S Gilhotra[1,2,3]

Ophthalmic manifestations of multiple sclerosis are frequent including acute optic neuritis, ocular motor disturbances and intermediate uveitis. We report an unusual case of multiple sclerosis presenting as acute hypertensive uveitis. A 56-year-old man was referred by his family doctor with a 2-week history of right eye pain and decreased vision. Best-corrected visual acuity was Count Fingers on the right and 6/9-2 on the left. Intraocular pressure was 55mmHg and 14 mmHg on the right and left respectively. He had a right relative pupillary defect and a left internuclear ophthalmoplegia. Vitreous cells were present in the right eye and there was peripheral sclerosis and periphlebitis superior temporally. MRI Brain and Spine revealed multiple T2 hyperintense lesions consistent with multiple sclerosis. Multiple sclerosis may present initially with an acute elevation of intraocular pressure and intermediate uveitis.

Key words: Hypertensive uveitis, multiple sclerosis, uveitis

While ophthalmic manifestations of multiple sclerosis (MS) are frequent, and include acute optic neuritis, ocular motor disturbances, and intermediate uveitis, the authors are unaware of acute hypertensive uveitis (AHN) as an ophthalmic presentation heralding MS.

Case Report

A 56-year-old Caucasian man was referred by his family doctor with a 2-week history of right eye pain and decreased vision. Medical history included diet-controlled type 2 diabetes mellitus and treatment refractory paranoid schizophrenia managed with sodium valproate, risperidone, and olanzapine. He was a current smoker with no significant ophthalmic history.

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[1]Department of Ophthalmology, The Queen Elizabeth Hospital, [2]Department of Ophthalmology, The Royal Adelaide Hospital, [3]The South Australian Institute of Ophthalmology, Adelaide, South Australia, Australia

Correspondence to: Dr. Melissa Kate Shields, Department of Ophthalmology, The Queen Elizabeth Hospital, 28 Woodville Road, Woodville South, South Australia, Australia 5011, Australia. E-mail: melissa.kate.ellis@gmail.com

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Best-corrected visual acuity was Count Fingers and 6/9-2 in the right and left eyes, respectively. Intraocular pressure (IOP) was 55 and 14 mmHg on the right and left, respectively. He had a right relative pupillary defect and a left internuclear ophthalmoplegia.

Anterior segment showed mild hyperemia on the right, with diffuse Keratic Precipitates (KPs) but with no cells. Angles were open bilaterally (Grade 4). There was no posterior synechia or iris bombe. Vitreous cells were present. Cup-to-disk ratio was 0.3 bilaterally. There was no disk swelling or hemorrhage. Peripheral sclerosis and periphlebitis were present superior temporally in the right eye. There was no retinitis.

Vitreous tap was negative for CMV, HSV-1, HSV-2, Varicella-zoster, and EBV. CSF analysis showed oligoclonal bands (CSF IgG: 0.27 g/L [0.01–0.03], CSF albumin: 0.38 g/L [0.10–0.25], CSF IgG/albumin ratio: 71% [<12%]). MRI brain and spine revealed multiple T2 hyperintense lesions consistent with MS.

The patient was initiated on oral prednisolone (1 mg/kg) and topical prednisolone acetate. Cycloplegia was achieved with topical atropine. His ocular hypertension (OHT) was managed initially with oral acetazolamide and topical brinzolamide. Oral prednisolone was slowly weaned over several months. He was referred to neurology for formal diagnosis and ongoing management of MS.

Discussion

MS is a chronic autoimmune demyelinating and degenerative disorder of the central nervous system that frequently affects both ocular motor and visual sensory systems.

Acute demyelinating optic neuritis is the most common ophthalmic manifestation with up to 50% of adult patients experiencing at least one episode over the course of their disease.[1] Ocular motor manifestations include internuclear ophthalmoplegia, saccadic hypermetria, and gaze-evoked and pendular nystagmus.[2,3] Reduction in contrast sensitivity and color perception is often present in patients despite normal visual acuity. Local anatomical changes including thinning of the retinal nerve fiber layer and macular volume loss may also be present in asymptomatic patients.[4]

Ocular inflammatory diseases, particularly pars planitis and retinal periphlebitis, are also associated with MS. The prevalence of uveitis in patients with MS varies widely in the literature from 0.65% to 36.7%.[4] Examining three large respective studies with cohorts greater than 2000 MS

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participants, the prevalence of uveitis ranged from 0.65% to 1.06% (average mean prevalence 0.75%).[5-7] Intermediate uveitis and panuveitis comprised 38.1% and 25.8%, respectively.

Elevated IOP is a common complication of uveitis. The prevalence of uveitis-related OHT [IOP >21 mmHg] ranges from 7.6% to 23%.[8] Chronicity, male sex, increasing age, and the presence of anterior uveitis are risk factors for developing OHT and glaucomas.[9,10] Panek *et al.* observed OHT in 12% of eyes acute uveitis and 26% with chronic disease.[11]

Mechanisms through which iridocyclitis results in aqueous outflow obstruction include accumulation of inflammatory debris within intertrabecular spaces, trabeculitis resulting in trabecular lamellae edema, or angle closure secondary to ciliary body swelling.[12] Angle closure was not a feature in our patient and the former two mechanisms are favored. Prostaglandin-mediated increase in vascular permeability increasing aqueous humor production has been proposed in syphilitic inflammatory OHT syndrome and could potentially be applied to MS models.[13]

Most causes of inflammatory OHT syndrome are infectious in nature and include anterior herpetic uveitis, toxoplasmic retinochoroiditis, and syphilis. Posner–Schlossman and sarcoidosis have also been implicated. Our patient was investigated extensively and each of these pathologies was excluded. Furthermore, he had no history of damage to the trabecular meshwork, no recent topical steroid use, and no posterior or peripheral anterior synechia.

Conclusion

To our knowledge, MS as a cause of an acute elevation of IOP has not been described. This finding suggests that MS should be considered as a cause of acute OHT, particularly in the presence of intermediate uveitis.

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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