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Unusual presentation of more common disease/injury

Bilateral, acute angle-closure glaucoma associated with Guillain-Barré syndrome variant

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Summary

A 55-year-old man presented with bilateral reduced visual acuity, limitation of extraocular movements, areflexia and ataxia. He was diagnosed with Miller Fisher Syndrome, precipitating bilateral simultaneous acute angle closure glaucoma due to autonomic dysfunction. He was subsequently treated for both conditions and made an excellent recovery.

BACKGROUND

Acute angle-closure glaucoma is a sight-threatening ocular emergency. In anatomically susceptible eyes, pupillary dilatation can precipitate complete obstruction of aqueous outflow, leading to a rapid rise in intraocular pressure (IOP). Bilateral simultaneous acute angle-closure glaucoma (BSAACG) is rare and is usually due to external precipitants such as drugs[1 2] or general anaesthesia.[3] Miller Fisher Syndrome (MFS) is a clinical variant of Guillain-Barré syndrome and is characterised by ophthalmoplegia, ataxia and areflexia. We describe a case of BSAACG secondary to MFS, not previously reported.

CASE PRESENTATION

A previously fit and well 55-year-old man on no medication presented to Eye Casualty with a 2-day history of bilateral blurred vision and unsteadiness. His best-corrected visual acuities (BCVA) were hand movements and 6/18 in the right and left eyes, respectively. Examination revealed bilateral limitation of extraocular movements in all gaze positions, sluggish pupillary responses, bilateral corneal oedema, shallow anterior chambers and IOPs of 56 and 48 mm Hg (normal range 10–21 mm Hg). (figure 1A). Gonioscopic assessment of the iridocorneal angle was precluded by corneal oedema. There was no proptosis, ptosis or periorbital swelling.

The raised IOP was immediately treated with intravenous acetazolamide 500 mg, and topical pilocarpine 2%, timolol 0.25%, iopidine 1% and prednisolone 0.5% bilaterally. A neurological examination revealed normal tone and power in all limbs with no truncal ataxia. Supinator, biceps and triceps reflexes were absent, and ankle and knee jerk reflexes were grossly reduced. He also had a broad-based gait with inability to tandem walk. He was systemically well otherwise with normal vital observations.

INVESTIGATIONS

Routine bloods were normal and campylobacter, syphilis and borrelia serology were negative. Magnetic resonance angiography of the brain and orbits, and lumbar puncture results were also normal and an intravenous infusion of immunoglobulins was commenced. Subsequent blood tests showed positive antiglycolipid GQ1B IgG antibodies, confirming the diagnosis of MFS.

TREATMENT

Over the next 2 days his right and left IOPs improved to 23 mm Hg and 12 mm Hg, respectively (figure 1B). Gonioscopic examination of the drainage angles revealed narrow but not occludable angles. He subsequently underwent successful YAG laser peripheral iridotomies bilaterally, thereby ensuring an alternative route for aqueous flow and permanently preventing any further episodes of angle closure. His topical antihypertensive drops were gradually withdrawn.

OUTCOME AND FOLLOW-UP

Visual acuities at 6 months follow up were 6/6 bilaterally. A recent refraction showed him to be moderately hypermetropic (longsighted) with spherical equivalents of +2.25 OD and +4.50 OS. Biometric measurements revealed short axial lengths (21.33 mm right, 20.94 mm left), in keeping with hypermetropia and a higher risk of AACG.

DISCUSSION

MFS causes autonomic dysfunction[4] and pupil dilatation,[5] thereby precipitating AACG in susceptible eyes. There is one previous report of acute glaucoma in a patient with known MFS[6,] but we are the first to report MFS presenting with BSAACG. The association between autonomic dysfunction and acute glaucoma has been described previously: in a series of 112 patients with acute glaucoma, 58% were found to have abnormalities on systemic autonomic testing.[7] This may also explain the higher rate of AACG in diabetic patients. Our case serves as a reminder that autonomic dysfunction should be considered in any patient with BSACCG.

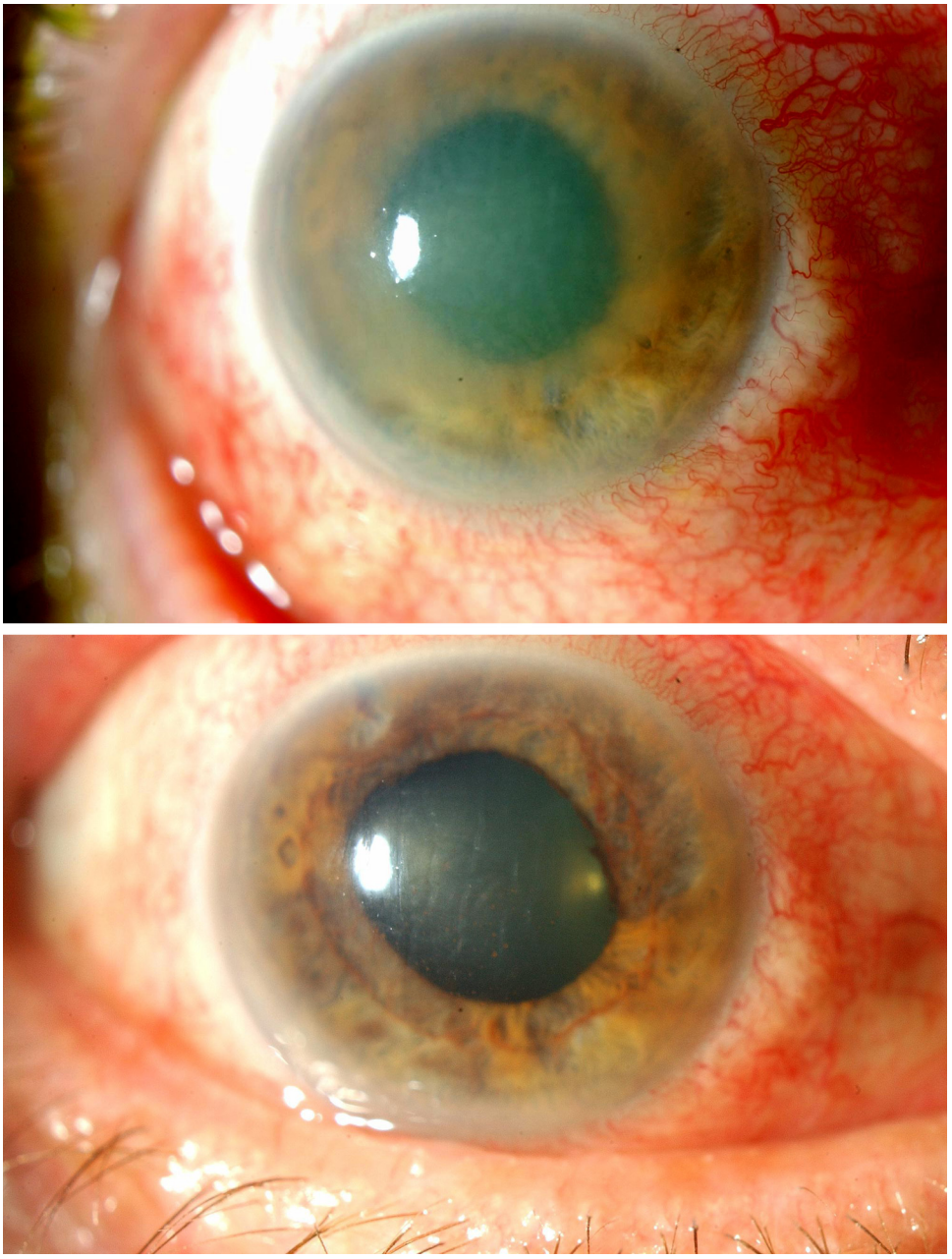

Figure 1 (A) Right eye at presentation showing corneal oedema and a mid dilated pupil. (B) Right eye after treatment, note that the pupil is persistently enlarged.

Learning points

- Hypermetropic (longsighted) patients are at higher risk of angle closure glaucoma.
- Always consider external agents such as drugs as precipitants of acute angle-closure glaucoma.
- Our case serves as a reminder of the role of autonomic dysfunction as a precipitant for BSAACG.

Competing interests None.

Patient consent Obtained.

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