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Case Report

A case of bilateral retinal venous occlusion in open angle glaucoma in a young adult

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Introduction

Glaucoma in the young adult is a rare disease, which could be primary with a strong family history as in juvenile open angle glaucoma or secondary due to various causes such as previous surgery, chronic ocular inflammation, chronic steroid use, pigment dispersion and irido corneal dysgenesis syndromes.^{1,2} Secondary glaucomas usually have signs of the underlying disease whereas juvenile open angle glaucoma can often be silent and detected incidentally. Juvenile open angle glaucoma presenting with retinal micro aneurysms is extremely unusual and to the best of our knowledge never been reported before.

Case report

A 24-year-old young male patient presented with history of diminution of vision in both eyes of 6 months duration. He was apparently well till 6 months back when he noticed painless transient blurring of vision in both eyes on waking up in the morning which improved over 24 h. However, he continued to have difficulty in seeing in dim light thereafter. Five and half months after the first episode, he had another episode of sudden bilateral diminution of vision which was much more severe and following which he noticed significant impairment of vision. This time he sought consultation at a private clinic and was diagnosed as a case of glaucoma and put on intra-ocular pressure (IOP) lowering topical drops. The IOP recorded at the time of initial presentation was 40 mmHg and 38 mmHg in right and left eye respectively. Thereafter, he was referred to this centre for further evaluation and treatment. He gave a history of itching and redness of both eyes for last one year for which he had been using topical steroid drops intermittently in both eyes prescribed by various local practitioners he had consulted. The total cumulative duration of topical steroid use was approximately 8 months and he had stopped steroids 2 months back. There was no history of trauma, intraocular surgery, or family history of glaucoma. At the time of presentation, his vision was 5/60 and 6/60 in right and left eye (best corrected), retinoscopy was emmetropic, there was mild conjunctival congestion in both eyes with upper tarsal conjunctival papillae, cornea was clear in both eyes and anterior chamber depths were normal. Right eye had a relative

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Fig. 1 – (a and b) Colour fundus photograph montage right and left eye showing multiple microaneurysms and haemorrhages.

afferent pupillary defect. Signs of secondary glaucoma such as pigment dispersion, evidence of anterior segment inflammation present or past, anterior segment dysgenesis and neovascularisation of the iris were absent. Lens was normal in both eyes. Fundus findings were as depicted in Fig. 1a and b, both eyes showing optic disc pallor and advanced glaucomatous optic atrophy with a vertical cup disc ratio of 0.9 in right eye and 0.8 in left eye. In addition, dot and blot haemorrhages and micro-aneurysms were seen in the superior, temporal and inferior quadrants in the right eye and in the superior and inferior quadrants in the left eye. On the Fundus Fluorescein Angiography (FFA) (Fig. 2a and b), these micro-aneurysms were found to be more numerous than in the colour photo. The vessels were patent on FFA but there was delayed arteriovenous (AV) transit time. Angles were open on gonioscopy without any evidence of increased pigmentation or neo vascularisation. The patient had been put on three drugs initially (timolol, brimonidine and travoprost) with which intraocular pressure was controlled in the range of 14–16 mmHg both eyes. Humphrey 30-2 visual fields showed only

Fig. 2 – (a and b) Fundus fluorescein angiography montage right and left eye showing microaneurysms in multiple quadrants which were more numerous than those seen on colour photograph.

residual central 10° field in left eye and unrecordable right eye due to poor vision. Optical coherence tomography (OCT) showed loss of retinal nerve fibres and thinned out neuroretinal rim in all quadrants both eyes. In view of the retinal findings of micro-aneurysms and haemorrhages with delayed AV transit time, he was evaluated for any underlying systemic hypertension, diabetes, anaemia, vasculitis and orbital ischaemia. Serial recording of blood pressure in the ward was normal. Investigations carried out included complete blood count, ESR, C reactive protein, blood sugar fasting and post-prandial, HbA1c, haemoglobin, lipid profile, VDRL, Mantoux test, chest radiograph, serum calcium, ACE, HIV, ANA, anti-DNA antibody, thrombophilia screening, fasting plasma homocysteine level, carotid ultrasound and Doppler, MRI brain and orbit. All investigations were normal. The patient was continued on the three topical anti-glaucoma medications that he had already been started on, as temporary withdrawal of one drug (brimonidine) resulted in a rising trend of IOP. With 3 medications, the IOP monitored over 2 weeks was in the range of 16–18 mmHg by Goldmann applanation tonometer. In view of the severe visual field loss, a fourth drug Brinzolamide was added with which IOP was brought down to 13–15 mmHg. FFA repeated at 4 months after control of IOP showed complete resolution of the haemorrhages and the micro-aneurysms (Fig. 3a and b). Currently patient is under regular follow-up on medical management.

Discussion

This case had several unusual features for which it is being reported. Firstly, the presentation with a short history and advanced glaucoma was unusual. Glaucoma presenting with advanced features has been reported earlier by Gadia et al. In their series, a chart review of cases of glaucoma in a tertiary care centre, advanced glaucoma at presentation was seen frequently in younger age group (below 35 years) and most of them were secondary glaucomas.³ Trauma, post-vitrectomy, corneal pathologies and steroid use were the common causes. Steroid-induced glaucoma was seen in those with long-term use, majority of whom were bilateral with IOP more than 30 mmHg and vision less than 6/60 in a significant proportion.³ In our case,

Fig. 3 – (a and b) Fundus fluorescein angiography 4 months after IOP control showing that the microaneurysms have disappeared.

history of topical steroid use correlates with a steroid induced aetiology; however, the relatively shorter duration of steroid and irreversibility of IOP on stopping of steroids was the confounding factors. Though the onset of increased IOP after steroid use can be seen as early as 4–6 weeks among steroid responders, significant clinically evident damage is usually seen after prolonged continuous use.⁴ Sustained increase in IOP even after stopping the steroid usually results after several years of steroid use and therefore it was unusual to find sustained high IOP in this case even after discontinuation of steroids after a much shorter period of steroid use, that too intermittently.⁵ In this case, it is postulated that an underlying juvenile glaucoma was worsened due to steroid use. The bilateral presentation with advanced field defects and high intraocular pressure is quite characteristic of juvenile open angle glaucoma even though in this case there was no family history of glaucoma.⁶ Tests for genetic markers for juvenile glaucoma could not be done as these tests were not available at this centre. The other unusual features were the episodes of vision loss and the presence of micro aneurysms and retinal haemorrhages in multiple quadrants for which investigations failed to reveal any cause. The episodes of acute vision loss, onset on waking in the morning, presence of haemorrhages in several quadrants and delayed AV transit time in initial FFA are strongly suggestive of episodes of bilateral Central Retinal Vein Occlusion (CRVO) which resolved spontaneously.⁷ Glaucoma is an established risk factor for CRVO.⁸ However, bilateral venous occlusion in a new undetected case of glaucoma is rare and reported only once.⁹ In this case report, a branch retinal vein occlusion in one eye and resolving central vein occlusion in the other eye were the presenting features in a case with underlying pigmentary glaucoma. Even more unusual were findings of micro-aneurysms in multiple quadrants, which could be due to chronic hypoperfusion because of prolonged severe raised IOP causing lowered perfusion and ischaemia. On controlling the IOP, the micro-aneurysms disappeared and AV transit time became normal once normal perfusion pressure was restored. Vascular insufficiency has long been suspected as one of the mechanisms for ganglion cell death and loss of retinal nerve fibres in open angle glaucoma.¹⁰ In this case, the presence of micro-aneurysms we believe is a pointer for ischaemia resulting from chronic elevation of IOP. Open angle glaucoma in a young adult presenting with bilateral microaneurysms has never been reported, to the best of our knowledge.

Conclusion

This unusual presentation of glaucoma in a young adult highlights the occurrence of chronic venous insufficiency in young pliant vessels due to sustained high intraocular pressure resulting in retinal ischaemia and formation of microaneurysms.

Conflicts of interest

The authors have none to declare.

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