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Case Report

Vascular insufficiency leading to recurrent disc haemorrhage in normotensive glaucoma

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ABSTRACT

Disc haemorrhage is a sign of progression in normotensive glaucoma. We report a case who was presented with right eye (RE) optic disc haemorrhage and retinal nerve fibre layer defect at first visit, he was diagnosed as normotensive glaucoma (NTG). On subsequent follow up visits he had recurrent disc haemorrage and progression in visual fields and optical coherence tomography (OCT). On further evaluation he had h/o of dehydration episodes as he lives in hotter climates and on systemic evaluation he had carotid stenosis and nocturnal dip in 24 hr blood pressure monitoring. This case tells about importance of systemic evaluation in normotensive glaucoma.

1. Introduction

Glaucoma is characterised by chronic progressive optic neuropathy with corresponding visual field changes with or without raised Intraocular pressure (IOP). There is increased risk of structural and functional damage to the optic disc in glaucomatous eyes with disc haemorrhage (DH) being the definitive factor of glaucoma progression.[1,2] Progression is assessed by clinical examination, visual field comparison and retinal nerve fibre layer (RNFL) thinning by OCT.

2. Case Report

A 57 years old known hypertensive male presented to us 6 years back with complaints of diminution of near vision both eyes since 2-3 yrs. On examination his visual acuity in right eye (RE) 6/9, left eye (LE) 6/6 near vision with +1.75DS N6 both eyes(BE). BE anterior segment was within normal limits (WNL), IOP 14 mmHg with CCT 490microns and corrected IOP was 17mmhg. Both eye gonioscopy showed open angles. Fundus showed RE cup disc ratio (CDR) 0.65 with infero-temporal DH with corresponding RNFL defect. LE showed CDR0. 4 healthy neuro-retinal rim(NRR).(Figure 1) Background retina and macula WNL. OCT showed RE inferior quadrant thinning, LEWNL and patient was diagnosed as RE Normal tension glaucoma (NTG) and started on brimonidine 0.2% eye drop twice in RE as it increases optic nerve head blood supply and patient was advised to undergo carotid doppler to look for any vascular insufficiency.

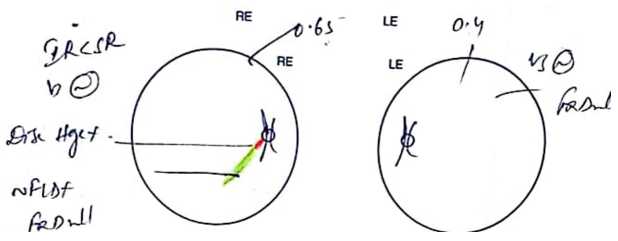

Figure 1: Fundus picture depicting RE inferior disc Hg and retinal nerve fiber layer defect

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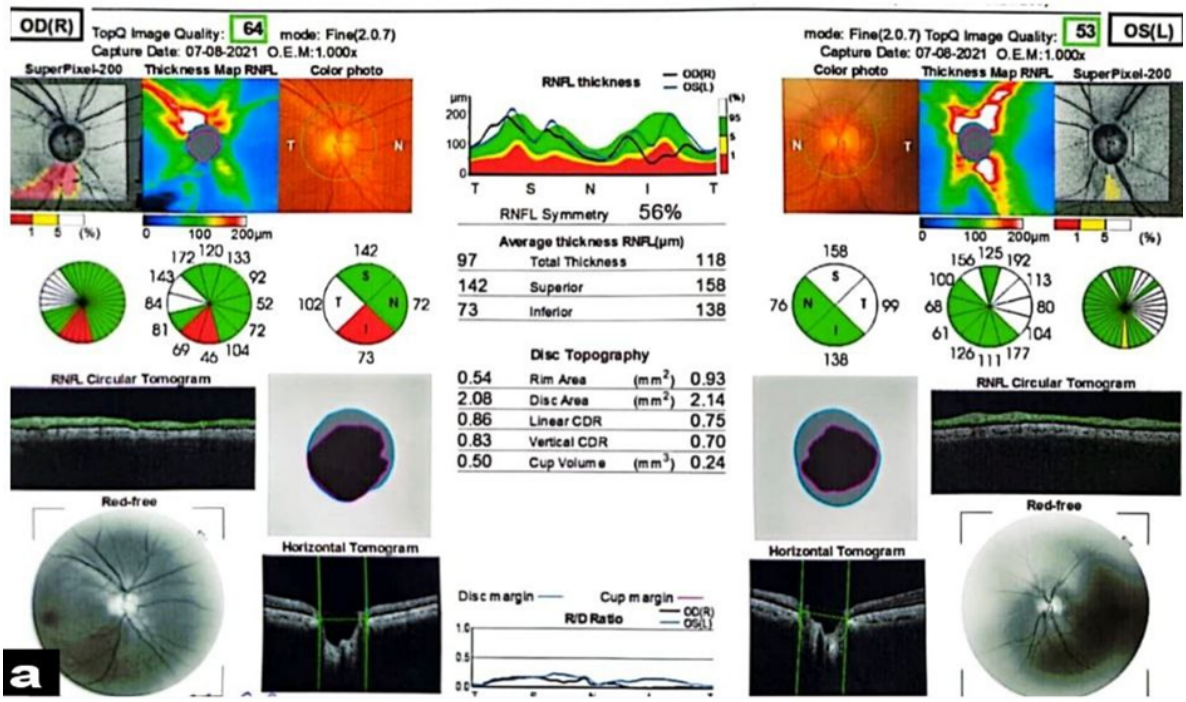

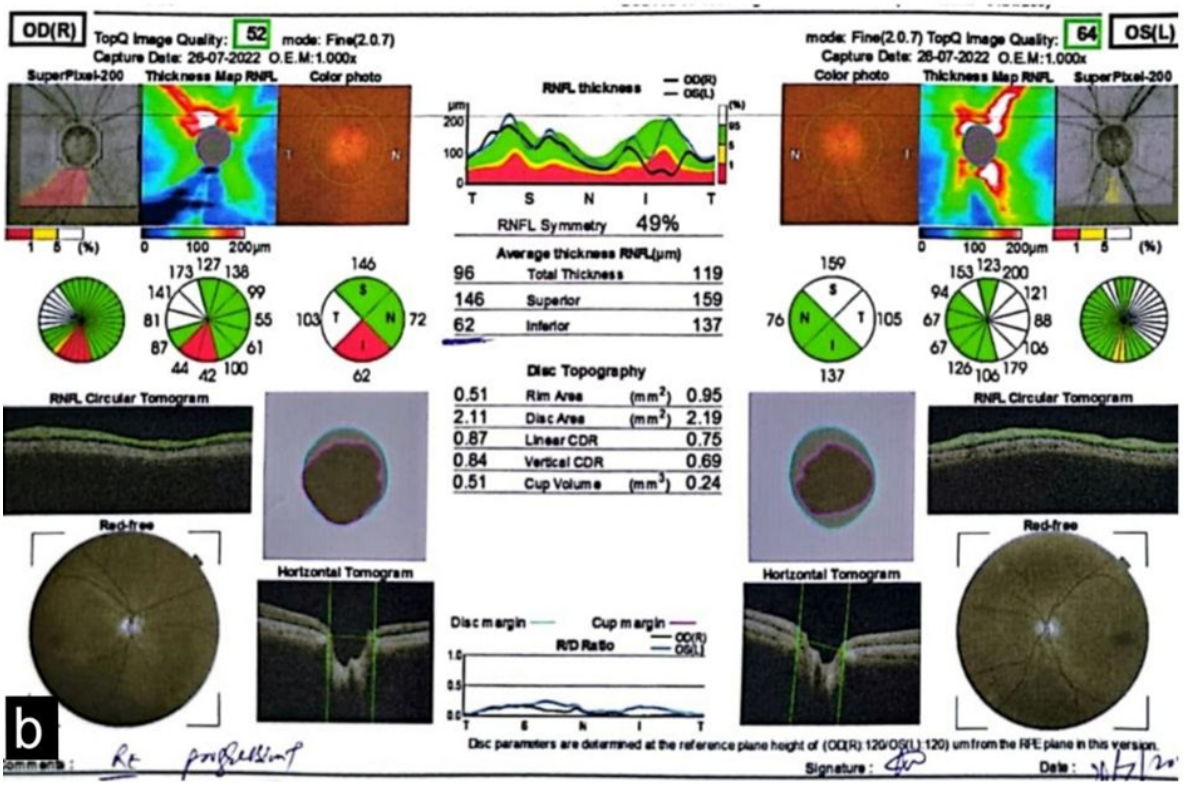

Figure 2: a, b): OCT Glaucoma image showing progressive inferior quandrant thinning in RE

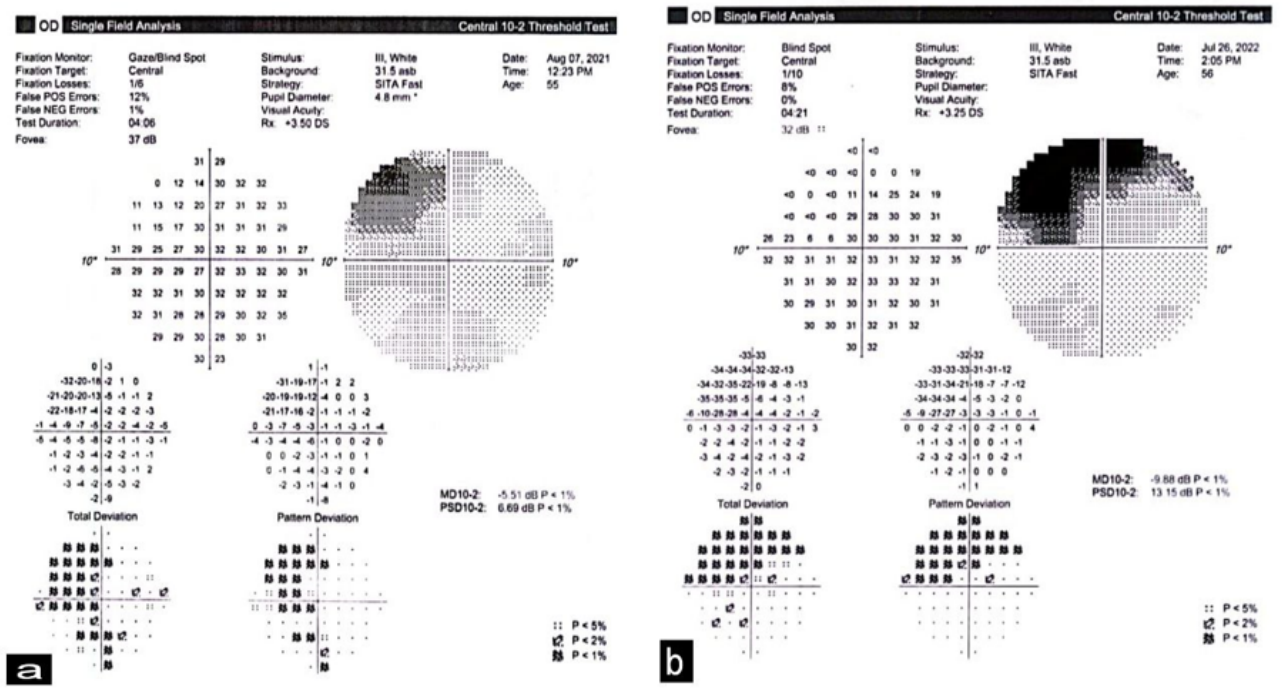

Figure 3: a, b): RE visual fields 10-2 showing progression in mean deviation

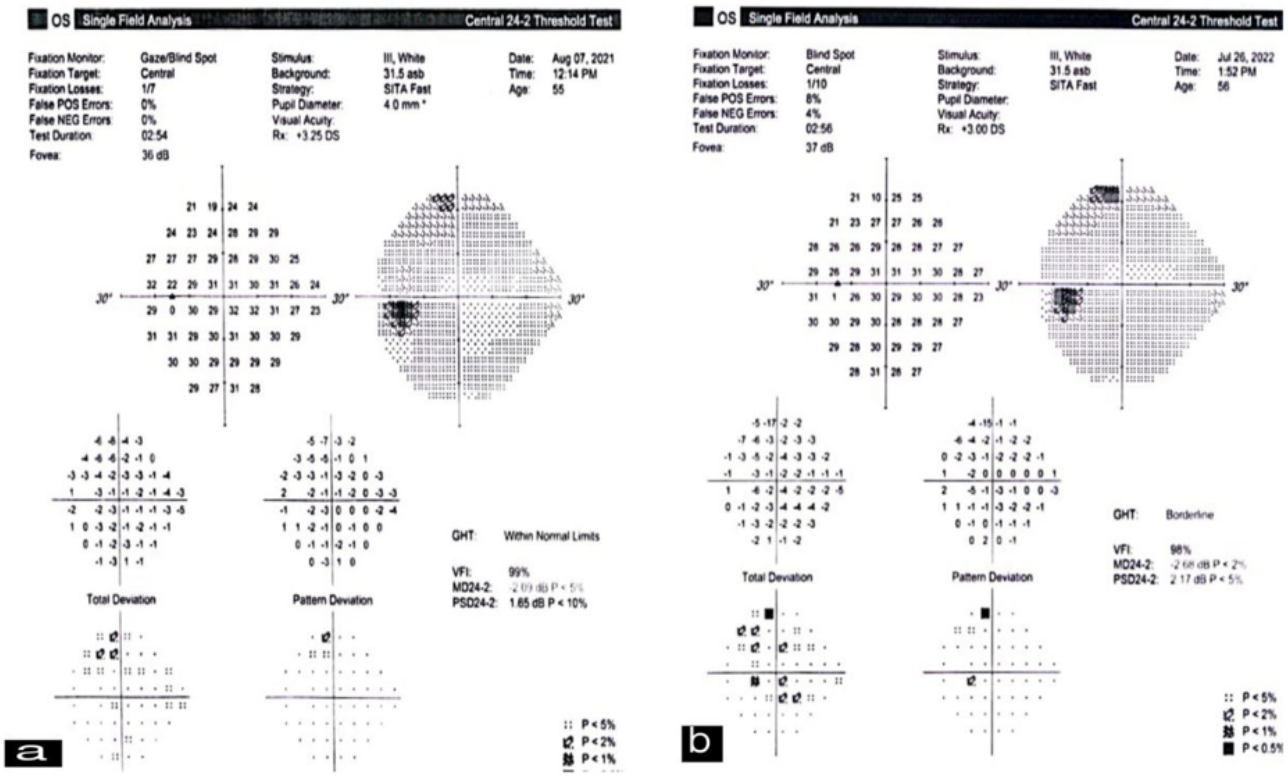

Figure 4: a,b): LE visual fields 24-2 with in normal limits

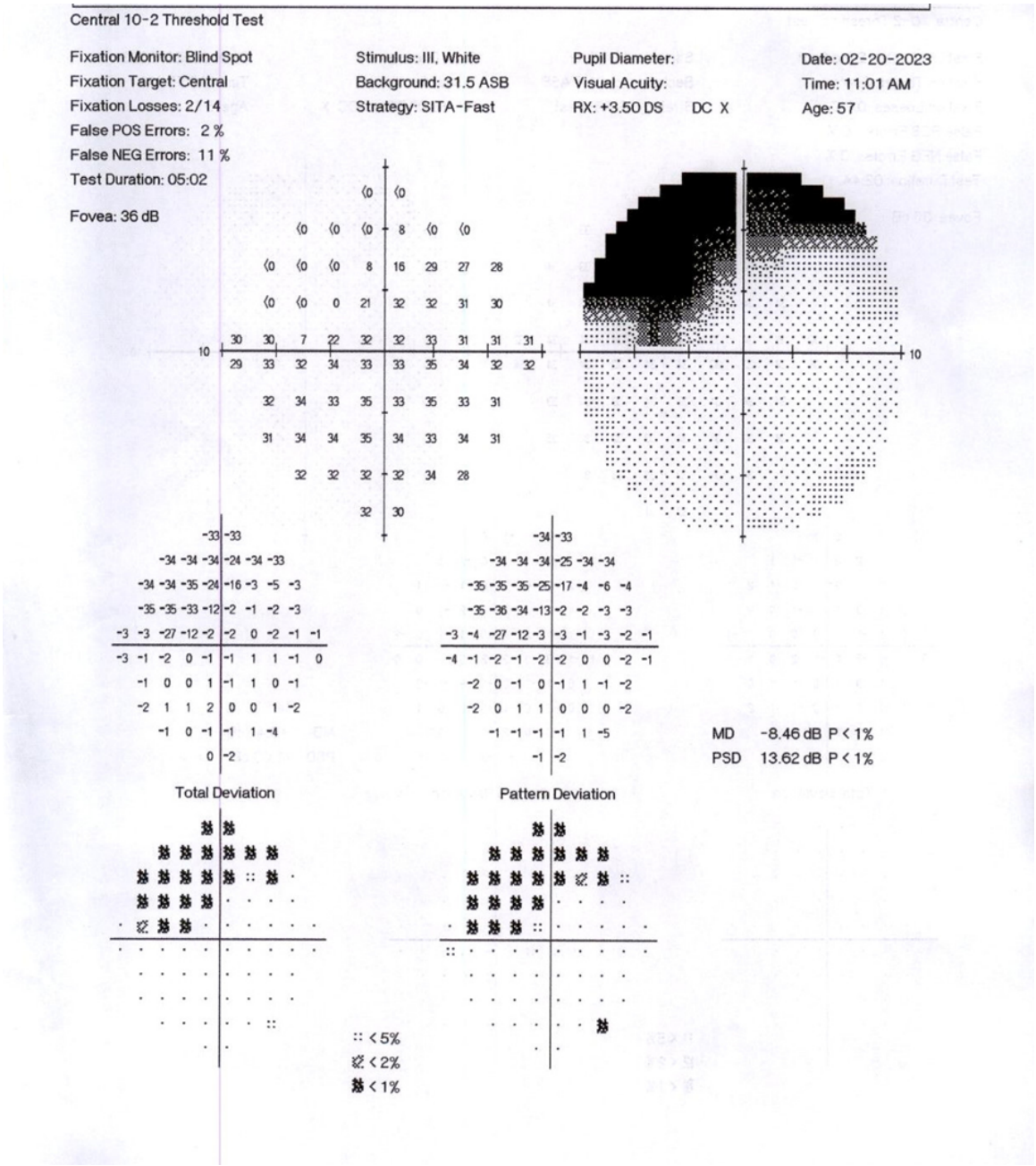

Figure 5: RE 10-2 HFA showing superior arcuate scotoma with stable MD

Patient came after 4 years with IOP 16mmHg and 12mmHg in RE and LE respectively without using antiglaucoma medications(AGM)and has not gone for carotid doppler. Right eye CDR was 0.7 with inferior rim loss with infero-temporal RNFL defect. LECDR was 0.4 with appearance of inferior DH suggestive of development of glaucoma in LE also. Visual field showed RE superior arcuate scotoma with macular spared with MD-11.82db. LE HFA was WNL. OCT showed RE inferior quadrant thinning (70microns). LE36 sector map borderline thinning (144 microns) corresponding to DH. Patient was started on ripasudil 0.4% twice in BE as he was not comfortable with brimonidine. Though fields were normal in LE and ripasudil was started because of DH. After 3 months IOP was 12mmhg BE with AGM.CDR0.7 inferior rim loss RE. DH was resolved in LE with CDR0.4 and was asked to continue AGM. Patient came after 6 months with BE ripasudil on, IOP of 12 mmHg. RE CDR 0.7 inferior rim loss. LE showed reappearance of inferior DH with CDR 0.4. OCT showed RE Inferior thinning (73 microns) and

LE borderline thinning (138 microns) corresponding to disc haemorrhage in 36 sector map, (Figure 2). Visual fields 24-2 and 10-2 showed progression in RE with superior arcuate scotoma, MD -5.51db (Figure 3). LE visual field was WNL (Figure 4). Despite good IOP control he was asked to continue ripasudil BE and added Dorzolamide 2% eye drop three times a day in BE as it increases optic nerve head blood supply, to reduce the nocturnal dips and was insisted to undergo carotid doppler.

He presented after 1 year with using only dorzolamide on and off BE and IOP was 20mmHg BE Ripasudil was not available in his place, so he discontinued. RECDR was 0.7 inferior rim loss, LE 0.4,? inferior slope. OCT also showed RE progressive inferior thinning(62 microns) and LE 36 sector map borderline thinning with inferior quadrant 137 microns thickness.(Figure 2) Fields showed RE progression with macula spared MD -9.88db (Figure 3). LE early superior depression MD-2.68DB.(Figure 4) Patient was advised to start bimatoprost 0.03% eye drop at bed time and Dorzolamide 2% eye drop three times.

Doppler study showed mild stenosis in right carotid bulb. Cardiologist started him on antiplatelets. On further evaluation had severe episodes of sweating and dehydration due to hotter climates and on 24 hr BP monitoring showed nocturnal hypotension suggesting decreased perfusion to optic nerve blood supply so, cardiologist changed his anti hypertensive medication. Reviewed 6months later with IOP 18mmHg, visual fields RE 10-2 showed stable MD -8.46dB (Figure 5). LE stable MD -0.47dB.

3. Discussion

H is a diagnostic feature of glaucoma, occurring at rates of 4.0 to 5.7% in glaucoma patients compared with 0.2% in normal eyes. DH is a significant negative prognostic factor for NTG. It is a sign of progressive damage of the RNFL[3] DH can be caused not only by ischemic microinfarction in the optic disc, but also by mechanical rupture of small blood vessels arising from structural changes at the level of the lamina cribrosa.[4]

Significant RNFL loss was already present in the DH only eyes with apparently normal RNFL configuration by red-free fundus photography, indicating that pre perimetric changes of the RNFL are already present. These results suggest that OCT has the potential to detect subclinical or pre perimetric RNFL loss in the eyes with DH.[5] In our case LE showing recurrent DH with retinal nerve fibre thinning is evident in clock hr map and 36Q map suggesting subclinical loss.

Progression of visual fields in NTG patients with DH is faster than in NTG patients without DH.[6] Moraes and associates[7] showed that rapid and localized visual field progression detected using automated visual fields preceded development of a DH.

CNTGS (Collaborative normotension glaucoma study) showed 12% progression in cases with DH despite 30% IO prediction.[8] OHTS (ocular hypertension study) showed 5% progression in cases with DH despite a 20% IOP drop.[9]

Associated risk factors for progression in normotension glaucoma are Corneal thickness:[10] A thin cornea can fool us into believing that a patient's IOP is better controlled than it is.

Non-IOP disease mechanisms[11] are perfusion deficit and vascular dysregulation, systemic hypertension, nocturnal hypotension, migraine, raynaud phenomenon and obstructive sleep apnoea.

These risk factor should be evaluated despite of low IOP in normotensive patients which are closely associated with severity and progression.

With 24-hour BP monitoring in this case demonstrated the duration and magnitude of nocturnal systemic hypotension that is MAP (mean arterial pressure) during his sleep below the daytime MAP and was associated with progressive visual field loss. As vasculopathy associated with systemic hypertension and diabetes can cause microinfarctions and ischemic changes in optic disc vessels, making these vessels vulnerable to mechanical rupture, it is logical that such microvascular diseases are correlated with DH in NTG patients.[12] In our case hypovolemic episodes leading to hypoperfusion of the optic nerve head is the cause for recurrent disc haemorrage and progression.

4. Conclusion

This case tells us the importance to evaluate the patient thoroughly with history and systemic evaluation to rule out the factors causing progression despite normal IOP. As the eye is the window of brain, it also gives us the clue about importance of vascular factors and its insufficiency need to be ruled out to improve treatment guidelines. Normotensive glaucoma and recurrent disc haemorrhage systemic causes should be evaluated.

5. Source of Funding

None.

6. Conflict of Interest

None.

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