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Case Report

An interesting case of rubeosis iridis with neovascular glaucoma in a young patient

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Introduction

Rubeosis iridis is defined as neovascularization of the iris characterized by numerous coarse and irregular vessels on the surface and stroma of the iris. These new blood vessels may cover the trabecular meshwork, cause peripheral anterior synechia and give rise to secondary glaucoma. Its most frequent causes are diabetes mellitus and central retinal vein occlusion (CRVO).

CRVO is one of the most common retinal vascular disorders that may lead to blindness. It usually affects older patients, with over 90% cases being over the age of 65 years.¹ Hypertension, diabetes, atherosclerosis and hypercoagulable states are all considered as its predisposing factors, in

addition to the various local ophthalmic conditions such as open angle glaucoma, ocular trauma and orbital infections.¹

Here we report an interesting case of unilateral rubeosis iridis leading to severe neovascular glaucoma following CRVO in a young patient with no major predisposing factors whatsoever.

Case report

A 34-year-old patient reported with complains of blurring of vision in his left eye for the past 2 weeks. There was no history of any pain, discharge or injury or any systemic diseases like diabetes, hypertension or hypercholesterolemia. The patient also claimed to have suffered an episode of redness in his left eye about 4 months back which he passed off as normal conjunctivitis from which he recovered without treatment.

Examination revealed the right eye to have a normal vision with normal anterior and posterior segments. However, the left eye vision was reduced to just perception of light. Slit lamp examination revealed rubeosis iridis in all the four quadrants with a very steamy cornea (Fig. 1). The anterior chamber appeared shallow with a fixed mid-dilated pupil. The fundus details could not be made out. The intraocular pressure (IOP) measured with a Non-Contact Tonometer (NCT) was normal in the right eye (12 mm of Hg NCT), but very high, 72 mm of Hg NCT in the left.

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A diagnosis of rubeosis iridis was made with the possible causes being (1) Eales' disease (2) CRVO.

The patient was started on systemic as well as topical anti-glaucoma drugs to lower the IOP and prevent any further optic nerve damage (IV Mannitol 350 ml stat followed by Tablet Acetazolamide 250 mg 2 TDS and topical Timolol 0.5% + dorzolamide TDS + brimonidine 0.2% BD). Over the next 3 days the patient's IOP reduced to 32 mm of Hg NCT. The iris neovascularization too surprisingly regressed. The cornea on becoming clearer allowed better funduscopy, revealing total glaucomatous cupping with optic atrophy. Fundus fluorescein angiography (FFA) and spectral OCT (optical coherence tomography) confirmed the diagnosis of an old long standing CRVO (left eye) with total optic atrophy. The right eye was normal.

The patient's blood pressure, blood sugar, cholesterol and triglycerides were all within normal limits. However the serum homocysteine levels were raised upto 21.08 $\mu\text{mol/L}$ (normal levels being 5.46–16.20).

Once the IOP was stabilized below 25 mm of Hg NCT the patient was transferred to an higher centre, where he underwent laser pan retinal photocoagulation (PRP) and intravitreal anti-vascular endothelial growth factor (VEGF) (Fig. 2). However his vision did not improve due to the already severely damaged optic nerve (Fig. 3).

Discussion

Retinal vein occlusion (RVO), after diabetic retinopathy, is the second most common cause of retinal vascular abnormality in adults. On the basis of the occlusion site it is divided into 3 types (a) branch retinal vein occlusion (BRVO), (b) CRVO and (c) hemiretinal vein occlusion (HRVO) based on.² CRVO is further subdivided into ischemic and non-ischemic types. The latter being the milder type with only partial venous obstruction and good visual prognosis, while ischemic CRVO is associated with severe visual loss, due to nearly total retinal vein obstruction.¹ Upto 16.4 million adults worldwide are affected by RVO, with over 90% of them being above the age of 65 years.^{1,3–5}

Fig. 2 – 2 Weeks post op.

More than half of these cases have an associated systemic disorder (e.g. hypertension, hyperlipidemia, diabetes mellitus or systemic vasculitis).^{5,6} In addition to these well-known classical risk factors, hyperhomocysteinemia appears to play a significant role in the pathogenesis of this disease,⁶ and is considered the most common “emerging” risk factor related to RVO.⁷

Rubeosis iridis is a well-known complication of ischemic CRVO, which if not treated in time may lead to neovascular glaucoma (NVG).⁸ NVG is a severe form of glaucoma characterized by iris neovascularization, a closed anterior chamber angle, and extremely high intraocular pressures (IOP) along with severe ocular pain and poor vision.

This case is unique for various reasons. To begin with, the age of the patient was just 34 years, an age when the occurrence of CRVO is rare.^{3–5,7} CRVO is usually associated with systemic diseases or local causes like primary open angle glaucoma, ocular trauma and orbital infections.^{1,3–5,7} However, in this particular case the patient was a young healthy individual with no such ailments.

Iris neovascularization seen in the infero-temporal quadrant

Fig. 1 – Iris neovascularization seen in all 4 quadrants.

Fig. 3 – 2 Months post op.

The only test positive was hyperhomocysteinemia, which is also considered as a risk factor for cardiovascular diseases especially in young individuals.^{6,9}

CRVO per se is a painless condition, however when associated with neovascular glaucoma, it is very painful due to the severely raised IOP. Surprisingly this patient had no pain and had reported with simply blurring of vision.

Though NVG is the most dreaded and blinding complication of ischemic CRVO its management is still highly challenging, unpredictable, difficult and controversial. The initial management usually involves laser pan retinal photocoagulation (PRP) as well as intravitreal anti-vascular endothelial growth factor (VEGF) and corticosteroids to reduce the ocular ischemic thus in turn causing the regression of NVI and finally reducing the IOP.¹⁰

However in our case we initially concentrated on controlling his IOP by all possible means because once NVG develops and the IOP is high, the major aspect of management is control of high IOP, which is almost invariably the main factor in irreversible and massive visual loss, rather than the original disease, which induced NVG.¹⁰ Also since on presentation there was already severe glaucomatous optic nerve damage we tried to salvage any possible vision and prevent further damage. But the interesting fact that we noticed was that as the IOP was gradually controlled it was accompanied with a simultaneous regression in the NVI, so much so that after a week of treatment with just anti-glaucoma medication the NVI had totally disappeared.

To the best of our knowledge this has never been reported anywhere in literature before.

Conclusion

Cases like these should remind us that CRVO, though a disease of the elderly can occur even in young healthy individuals. Once diagnosed it is essential to have a high index of suspicion for the possible development of neovascular glaucoma because its early diagnosis and treatment can prevent irreversible visual loss.

Homocysteine levels should always be evaluated in a suspected case of CRVO, as hyperhomocysteinemia appears to play a significant role in its pathogenesis, specially if the patient is young because elevated plasma

homocysteine levels are also associated with cardiovascular diseases which too require a complete evaluation as well as follow up.

Conflicts of interest

All authors have none to declare.

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