

## Choroidal hypoperfusion: an indicator of low tension neovascular glaucoma

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### Abstract

**Purpose** To report a case of low tension neovascular glaucoma in ocular ischemic syndrome.

**Methods** An elderly man presenting with vision loss after an episode of hemiparesis was investigated to look for the cause of vision loss and treated.

**Results** Fluorescein angiography demonstrated poor uveal perfusion and treatment with panretinal photocoagulation led to rapid closure of the angle.

**Conclusion** This case highlights the typical ophthalmic features of ocular ischemic syndrome and emphasizes the necessity of patient education and prognostication.

**Keywords** Ocular ischemic syndrome · Low tension neovascular glaucoma · Carotid stenosis

**Precis** Low tension neovascular glaucoma in ocular ischemic syndrome is presumed to be secondary to poor ciliary body perfusion [1]. We report the demonstration of the same on fundus fluorescein angiography as patchy choroidal filling and delayed

uveal perfusion. Rapid angle closure with panretinal photocoagulation was the other unique feature.

**Case report** A 63-year-old diabetic, hypertensive of 8-year duration, with minor branch retinal vein occlusion (BRVO) OU, cystoid macular edema (CME) OS and 20/60 corrected vision OU, during regular follow-up presented with reduction in vision OD 4 months after a left-sided hemiparesis.

Examination revealed corrected vision of 20/80 OD, 20/60 OS, rubeosis iridis (Fig. 1), zipping of angle due to 6 clock hours of peripheral anterior synechiae, normal intraocular pressure (IOP) and dark engorged venules in right eye (Fig. 2a). Fluorescein angiography of the right eye (Fig. 2c) revealed slow and patchy choroidal filling (110 s delay) and prolonged arteriovenous transit time (20 s). Retinal capillary non-perfusion and telangiectatic collateral vessels in the distribution of the tributaries of the occluded venule were seen at macula (Fig. 2d). Fundus view in left eye was hazy due to cataract (Fig. 2b), and there was no delay in filling of fluorescein dye; capillary non-perfusion corresponding to the branch vein occlusion was seen at macula.

Color Doppler imaging showed thrombosis of right internal and common carotid artery (CCA). Magnetic resonance angiogram corroborated near total occlusion of right CCA and decreased perfusion in right hemisphere. Right carotid endarterectomy was advised, and prophylactic panretinal photocoagulation (PRP) of right eye was

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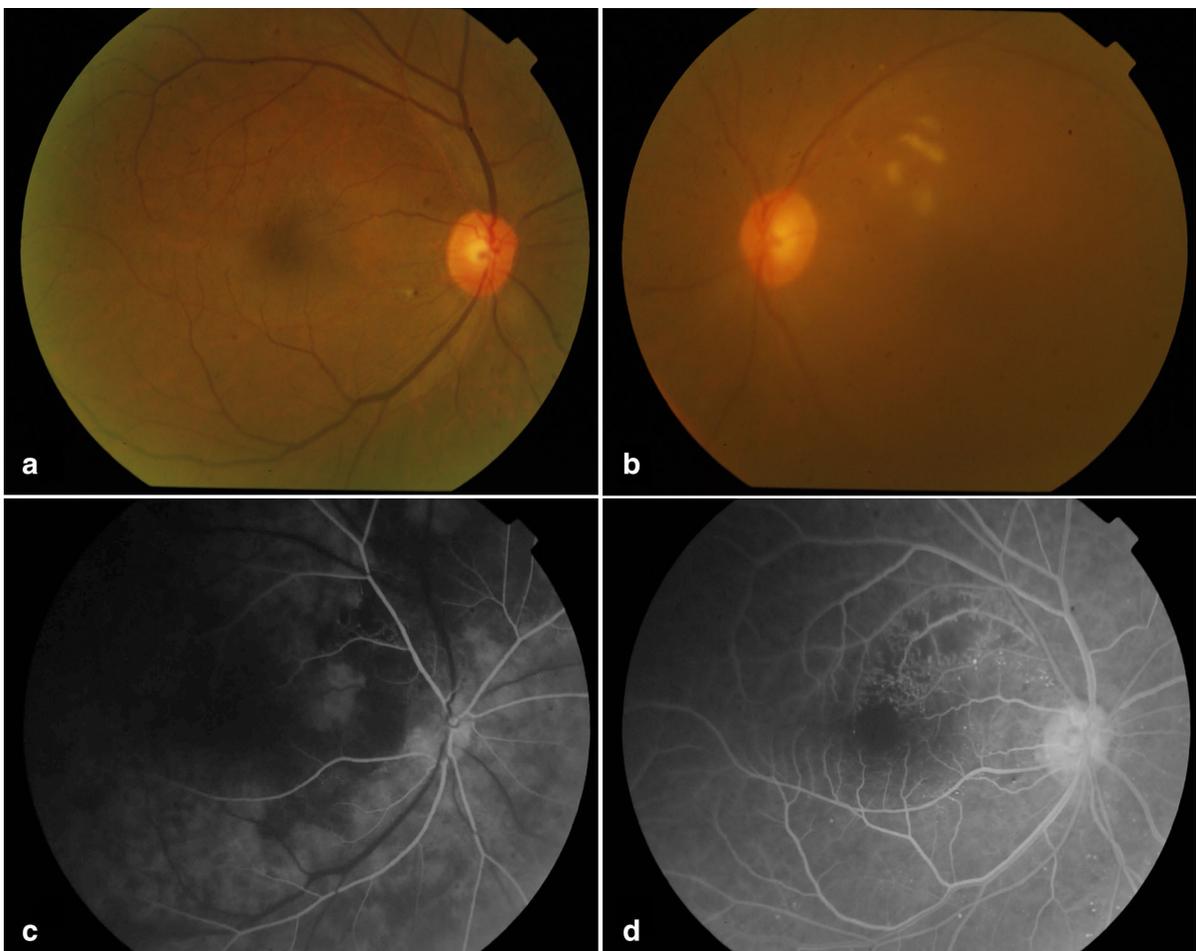


**Fig. 1** Slit lamp capture of the right eye shows radiating iris new vessels at pupil in ocular ischemic syndrome

conducted in two episodes at 2-week interval, to prevent likelihood of ocular hypertension post-endarterectomy [2].

Pan retinal photocoagulation result was unusual in that no regression of iris new vessels occurred and visual acuity reduced further to 2/60 at 1-month follow-up post-laser. Repeat gonioscopy at this point showed worsening of angle zipping with 360 degree peripheral anterior synechiae (PAS), with IOP still in the normal range at 16 mmHg. The vision loss was presumed to be secondary to ischemic optic neuropathy [3].

Despite close follow-up of 9 months, secondary glaucoma did not supervene and IOP remained



**Fig. 2** Color fundus photograph shows dark and engorged venules in right eye (a) in contrast to the left (b). Cotton-wool spots at macula in left eye (b) seen secondary to minor branch retinal vein occlusion. Fluorescein angiogram of right eye in arterial phase shows prominent choroidal patchy hypoperfusion

(c) progressing to complete perfusion in later phase of angiography (d). Capillary non-perfusion, telangiectasia and distortion of foveal avascular zone represent a minor branch vein occlusion in the setting of ocular ischemic syndrome (d)

stable at 16 mmHg. He had not undergone carotid endarterectomy due to financial constraints. At the last follow-up, ectropion uveae and total cataract were the prominent anterior segment changes. Posterior segment examination was obscured by cataract, and B scan ultrasonography, however, showed normal posterior segment images.

*Comment* Ocular ischemic syndrome represents decreased blood flow in the retrobulbar vessels secondary to stenosis or occlusion of ipsilateral common carotid artery and internal carotid artery [4]. Among the various early presentations of this ominous condition, iris neovascularization stands as the most commonly detected sign [5]. Iris and angle neovascularization without any rise in IOP is a strong pointer to poor ocular perfusion. Delayed choroidal and retinal vascular filling on fluorescein angiography may explain the normal intraocular pressure despite complete PAS, as aqueous production gets affected due to poor ciliary body perfusion [6]. Standard treatment regimens like PRP to prevent rubeotic glaucoma may have unexpected outcomes, necessitating patient education and prognostication. The unusual PRP complication of accelerated synechial closure as seen in this patient is less easy to understand and could represent an aberrant response due to subclinical, transient inflammation induced by laser or undetected anterior chamber shallowing subsequent to anterior shift of lens iris diaphragm in these cases. A tight follow-up post-PRP in such cases and prophylactic use of atropine could help circumvent this rare complication.

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#### **Compliance with ethical standards**

**Conflicts of interest** The authors declare that they have no conflict of interest.

**Ethical approval** All procedures performed in this study involving human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

**Informed consent** For this type of study, formal consent is not required.

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