# Neovascular glaucoma

## *Rubeosis*

### DESCRIPTION

Neovascular glaucoma is always secondary to ocular ischaemia, typically conditions that result in retinal capillary non-perfusion and retinal hypoxia. The release of vasogenic factors stimulating the growth of new vessels in the fundus also promotes neovascularisation in the anterior segment of the eye. In the fully developed disease, contraction of a fibrovascular membrane in the angle causes peripheral anterior synechiae, ectropion uveae and high intraocular pressure. Once this stage is reached, the prognosis for vision is very poor.

The most common causes are central retinal vein occlusion and proliferative diabetic retinopathy. Other potential causes include occlusive disease of the carotid artery, leading to ocular ischaemic syndrome, central retinal artery occlusion, branch retinal vein occlusion, longstanding uveitis and retinal detachment, and intraocular tumours such as choroidal melanoma and retinoblastoma.

### SYMPTOMS

In the early stages of the disease process, symptoms may be conspicuous by their absence. As the condition becomes established and intraocular pressure rises, the patient may complain of poor vision, haloes around lights, a red eye, pain and photophobia.

### SIGNS

#### Stage 1

Neovascular glaucoma or rubeosis iridis: Initially, tiny whorls and tufts of capillaries resembling small clumps of pigment are seen around the pupil margin (Figure 1). These may or may not be accompanied by similar vessels in the angle. Intraocular pressure is usually normal.

#### Stage 2

Secondary open-angle glaucoma occurs from a proliferation of these vessels across the iris, ciliary body and angle, and the formation of a fibrovascular membrane causing elevated intraocular pressure.

#### Stage 3

This is reached when the membrane contracts and causes peripheral anterior synechiae. Secondary-angle closure and markedly elevated intraocular pressure may rapidly ensue. Other potential signs at this stage include conjunctival hyperaemia, corneal oedema, aqueous cells and flare, pathological cupping of the optic disc and visual field defects.

Careful and thorough biomicroscopy, gonioscopy and ophthalmoscopy are essential in all at-risk patients. Fluorescein angiography may be required and a B-scan ultrasound examination if the retina is obscured.

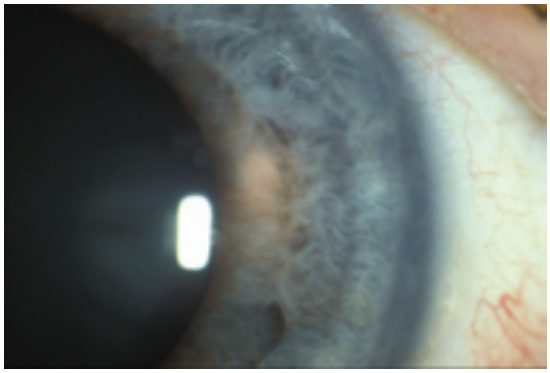

**Figure 1** Rubeosis: tiny whorls and tufts of capillaries resembling small clumps of pigment are seen around the pupil margin

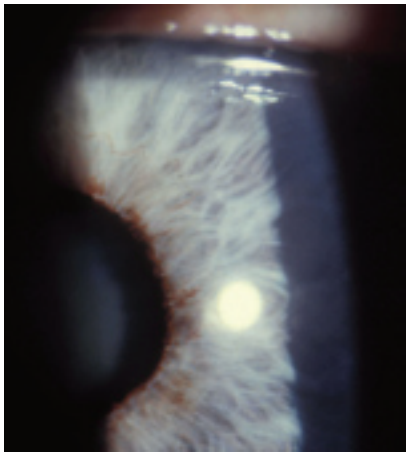

**Figure 2** Early rubeosis

### PREVALENCE

Neovascular glaucoma is relatively uncommon. Approximately 33 per cent of patients with central retinal vein occlusion develop neovascular glaucoma.

### DIFFERENTIAL DIAGNOSIS

Glaucoma – classification, acute angle closure glaucoma, inflammatory glaucoma. Conditions that may manifest raised IOP and either dilated iris vessels or new vessels such as post-surgical vascular engorgement, retinopathy of prematurity, Fuchs' heterochromic iridocyclitis and iridocorneal endothelial syndrome. Normal iris vessels are often seen in patients with light irides both with the slit lamp and on gonioscopy. These should be differentiated from new vessels.

### MANAGEMENT

This is one of the most difficult glaucomas to successfully manage especially when firmly established. Early diagnosis and treatment are essential.

#### Laser surgery

Just as new vessels in the fundus may resolve following timely pan-retinal photocoagulation, stage 1 iris vessels may similarly regress. In stage 2, pan-retinal photocoagulation is again indicated.

#### Topical medication

Control of intraocular pressure is via aqueous suppressants as in primary open-angle glaucoma, although miotics such as pilocarpine are contraindicated.

#### Incisional surgery

If intraocular pressure is inadequately controlled by medical means, a filtration procedure may be considered. Stage 3 eyes that have little useful vision and are congested and painful may gain some relief from topical steroids and atropine. Retrobulbar injection of alcohol may be considered and some eyes may need to be enucleated.

The full series of these articles will be available in the book *Posterior Eye Disease and Glaucoma A-Z* by Bruce AS, O'Day J, McKay D and Swann P. £39.99. For further information click on the Bookstore at **opticianonline.net**


- **Adrian Bruce** is a Chief Optometrist at the Victorian College of Optometry and a Senior Fellow, Department of Optometry and Vision Sciences, The University of Melbourne.
- **Justin O'Day** is an Associate Professor in the Department of Ophthalmology, The University of Melbourne and Head Of Neuro-Ophthalmology Clinic, Royal Victorian Eye and Ear Hospital.
- **Daniel McKay** is a Medical Officer at the Royal Victorian Eye & Ear Hospital.
- **Peter Swann** is Associate Professor in the School of Optometry, Queensland University of Technology.