Transient angle-closure glaucoma after retinal vein occlusion

Report of two cases

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Glaucoma may be a contributory factor in the aetiology of retinal vein occlusion or may result from this condition. When glaucoma occurs secondary to retinal vein occlusion, it is usually considered to be due to vascularization of the anterior chamber angle and closure of the angle by goniosynechiae (Duke-Elder and Dobree, 1967; Ballantyne and Michaelson, 1970).

The two cases of retinal vein occlusion reported below exhibited marked shallowing of the anterior chamber, pupillary block, and transient angle-closure glaucoma, without vascularization of the angle.

Case reports

Case 1, a 64-year-old man with polycythaemia vera, complained of deterioration of vision in the right eye for a period of 6 weeks.

Examination

The visual acuity was counting fingers in the right eye, the intraocular pressure was 32 mm. Hg, and there was hypermetropia of 2.5 D. Slit-lamp examination showed marked shallowing of the anterior chamber. There was a complete occlusion of the central retinal vein with marked venous engorgement, oedema of the optic disc, and many haemorrhages in the posterior pole. Gonioscopy revealed a narrow angle, only the anterior part of the trabeculae being visible.

In the left eye the visual acuity was 6/6, the intraocular pressure 17 mm. Hg, and the anterior chamber angle was wide open.

The depth of the anterior chamber was measured with Goldmann's attachment for the Haag Streit 900 slit lamp, using the technique described by Lowe (1966).^{*} The anterior chamber depth was 2.25 mm. in the right eye and 3 mm. in the left.

Treatment

The intraocular pressure in the right eye dropped to 18 mm. Hg after the instillation of one drop of 2 per cent. pilocarpine; 2 days later while pilocarpine treatment was continued, the anterior chamber depth was 2.25 mm. and the angle was slightly wider, the posterior trabeculae being visible in parts of the angle.

Tonography, performed 12 hours after the instillation of pilocarpine into the right eye, gave the following results:

$P_0 = 21$, $C = 0.18$ in the right eye

$P_0 = 9$, $C = 0.28$ in the left eye.

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^{*}The maximum observer's error with this method is 0.08 mm. (Lowe, 1971, personal communication, and our own unpublished material)

Progress

The anterior chamber in the affected eye gradually became deeper and the intraocular pressure remained normal: 7 months after the onset of the symptoms the anterior chamber depth was 2.7 mm. in the right eye and 2.95 mm. in the left; the intraocular pressure was 18 mm. Hg in the right eye, without treatment. On gonioscopy the scleral spur was visible all round and the ciliary body could be seen in part of the angle; no abnormal vessels were visible in the angle.

Case 2, a 55-year-old man, was examined because of blurred vision in the right eye for 1 week.

Examination

The visual acuity in the right eye was 2/60 and the intraocular pressure 25 mm.Hg. The eye was emmetropic. Slit-lamp examination gave normal results. There was a complete occlusion of the central retinal vein with marked venous engorgement and many large confluent haemorrhages in the posterior pole. Gonioscopy revealed a narrow, irregular angle, the anterior part of the trabeculae being just visible; no abnormal vessels were visible in the angle.

In the left eye the visual acuity was 6/6, the intraocular pressure was 16 mm.Hg, and the anterior chamber angle was wide open.

The depth of the anterior chamber was 2.6 mm. in the right eye and 3.1 mm. in the left.

Treatment

Pilocarpine was given three times a day to the right eye; the ocular tension became normal, and 1 week later the anterior chamber depth was 2.45 mm.

Progress

2 months later the patient returned to the clinic with a red, painful eye, having stopped using pilocarpine 10 days earlier. The visual acuity in the right eye was hand movements, the intraocular pressure 52 mm.Hg, and the anterior chamber depth 2.95 mm.; there was rubeosis of the iris and gonioscopy revealed an angle closed by broad synechiae with many clearly visible new vessels. Tonography in the left eye at this stage revealed $P_0 = 22$, $C = 0.38$.

Discussion

The two cases of central retinal vein occlusion reported above exhibited a transient elevation of the intraocular pressure associated with shallowing of the anterior chamber and closure of the chamber angle, without visible vascularization of the angle. In both cases the intraocular pressure became normal and the angle opened when treatment with pilocarpine was instituted. In Case 1 the anterior chamber gradually deepened and the intraocular pressure remained normal even when pilocarpine was discontinued. In Case 2 the intraocular pressure remained normal for 6 weeks with pilocarpine treatment, but intractable glaucoma developed later due to neovascularization of the angle.

Samuels (1935) suggested that glaucoma after retinal vein occlusion might be due to swelling of the vitreous pushing the lens forward, but he did not demonstrate any shallowing of the anterior chamber in his cases.

It has been shown by fluorescein photography (Eisner, 1965) that retinal vein occlusion is associated with transudation of fluid from the retinal vessels into the retina and subsequently into the vitreous, or into the retrovitreal space in the case of posterior vitreous detachment.

In the two cases of retinal vein occlusion reported above, there was probably a transudation of fluid from the retinal vessels into the vitreous cavity causing a forward

displacement of the lens, pupillary block, and angle-closure glaucoma. This type of glaucoma after retinal vein occlusion has not been described before.

It is important to perform gonioscopy early in the management of cases of retinal vein occlusion. In cases in which the angle is narrow, it may be justified to institute miotic therapy even if the intraocular pressure is normal.

Summary

In two eyes with occlusion of the central retinal vein there was a transient angle-closure glaucoma without vascularization of the angle, probably due to transudation of fluid from the retinal vessels into the vitreous, forward displacement of the lens, and pupillary block. This type of glaucoma after retinal vein occlusion has not been described previously.

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