Angle-closure glaucoma secondary to posterior scleritis

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SUMMARY This paper reports 3 patients who presented with angle-closure glaucoma secondary to posterior scleritis. Differentiation from primary angle-closure glaucoma is important, as medical treatment is markedly different while surgical treatment is not required.

Posterior scleritis is an uncommon eye condition with protean manifestations (McGavin et al., 1976; Watson and Heyreh, 1976; Cleary et al., 1975), the treatment for which is, however, relatively constant. This paper describes 3 patients with posterior scleritis each presenting with secondary angle-closure glaucoma. We emphasise the difference in clinical features between the eyes of our patients and eyes with primary angle-closure glaucoma, and report the effects of treatment. Our patients were made worse with miotic treatment but responded rapidly to mydriatics and treatment of the scleritis.

Case reports

CASE 1
A man aged 48 presented with a 10-day history of constant pain in his right eye associated with 4 attacks of amaurosis, each attack lasting for 20 minutes after coughing.

On examination the left eye was normal. Visual acuities were 6/5 unaided in each eye. There was a small area of scleritis in the inferotemporal quadrant of the right eye (Fig. 1). The anterior chamber did not have either flare or cells. Both pupils were normal in size and reaction. The intraocular pressures were 28 mmHg and 12 mmHg in right and left eyes. Anterior chamber depths were 1.6 mm and 2.5 mm in right and left eyes (Fig. 2). Gonioscopy showed that the angle of the right eye was closed while the angle of the left eye was open and of normal width.

Examination of the fundus after dilatation of the right eye revealed a localised choroidal detachment in the inferotemporal quadrant (Fig. 3), together with a shallow annular choroidal detachment. Fluorescein angiography showed leakage of the retinal veins overlying the inferotemporal choroidal detachment (Fig. 4a, b).

The diagnosis of secondary angle-closure glaucoma was made for this patient because of the atypical history, unequal anterior chamber depths, and scleritis.

Treatment consisted of oxyphenbutazone 200 mg 3 times a day and cyclophentolate drops 1% 3 times a day. After 48 hours the scleritis had resolved, the angle of the right eye had opened, the intraocular pressure was normal, and the eye was comfortable. One week later the anterior chambers of the 2 eyes were the same depth. The choroidal swelling disappeared after 2 weeks of treatment. A repeat fluorescein angiogram of the retinal veins previously shown to leak fluorescein did not reveal any abnormality. There has been no recurrence.

CASE 2
A man aged 40 presented with a 2-day history of constant pain in his right eye. Examination showed...
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that with his own glasses (-2.75 sphere right, -2.00 sphere left) his visual acuity was 6/18 right eye, and 6/6 left eye. Addition of a further -0.75 sphere to the prescription for the right eye improved the acuity to 6/9. The left eye was normal. The right eye had a marked anterior scleritis, 3 mm of proptosis, painful eye movements, and a tender globe on palpation. The anterior chamber of the right eye showed a few cells and a mild flare. Intraocular pressures were 24 mmHg and 14 mmHg.

Fig. 2 Case 1. Slit-lamp photographs of the right and left eyes to show difference in depths of the anterior chamber (R 1.66 mm, L 2.5 mm)

Fig. 3 Case 1. Fundus painting showing choroidal swelling over areas of scleritis (arrowed). The annular choroidal detachment has not been drawn

Figs. 4a, b Case 1. Fluorescein angiograms showing retinal vessels overlying the area of choroidal swelling, to show capillary dilatation (a) and leakage (b)
200 mg 3 times a day, acetazolamide 250 mg 3 times a day cyclopentolate drops 1% 3 times a day, and dexamethasone drops 0.1% 2-hourly. After 72 hours the angle opened and the intraocular pressure fell to 10 mmHg, the scleritis was much less marked, and the eye more comfortable. After 5 days the anterior chamber depths and intraocular pressures were equal; however, the choroidal detachment persisted. As a result, systemic prednisolone was given, dosage starting with 120 mg daily for 1 week, and then reducing by 20 mg daily. With this treatment the choroidal swelling resolved. He has had no recurrence of symptoms.

**Case 3**

A man aged 40 presented with a history of a possible injury to his left eye 4 weeks previously, followed 1 week later by distorted vision with haloes affecting the vision of his left eye. Examination showed severe glaucoma with a ‘retinal detachment’. When pilocarpine drops and acetazolamide failed to lower the intraocular pressure of his left eye, he was referred to Moorfields Eye Hospital for further management.

On examination the visual acuities were found to be, unaided, 6/5 and 6/24 (6/5 with −3.00 DS) in the right and left eyes. The right eye was normal; the left eye had a diffuse anterior scleritis (Fig. 6); the anterior chamber was ‘quiet’. Intraocular pressures were 18 mmHg and 40 mmHg in right and left eyes. Anterior chamber depths were 2.00 mm and 1.5 mm in the right and left eyes (Fig. 7), while gonioscopy disclosed an open angle of normal width in the right eye and a completely closed angle in the left eye.

Angle-closure glaucoma secondary to scleritis was suggested by the association of scleritis, dissimilar angle chamber widths, and acquired myopia.

After dilatation of the left eye, it was possible to see an annular detachment of the ciliary body together with a superotemporal choroidal detachment (Fig. 8), confirmed by ultrasound (Fig. 9).

These findings confirmed the diagnosis. Treatment was started with oxyphenbutazone 200 mg 3 times a day, acetazolamide 250 mg 3 times a day, neutral adrenaline drops 1% twice daily, and hyoscine drops 0.5% twice daily.

The choroidal effusion resolved over the next 4 weeks. The intraocular pressure returned to normal, and the anterior chamber depths equalised over the next 6 weeks (Fig. 10). There has been no recurrence.

**Discussion**

Posterior scleritis is an uncommon condition accounting for only 0.08% of referrals to eye departments (Williamson, 1974).
CLINICAL FEATURES OF POSTERIOR SCLERITIS

Posterior scleritis may in itself be difficult to diagnose. The features suggesting this diagnosis include unilateral signs of diffuse anterior scleritis, proptosis, and limitation of eye movements secondary to scleral thickening together with an exudative retinal detachment. The association of an exudative retinal detachment and scleritis was first made by Pürtscher (1891). Watson (1974) described the appearance of shifting subretinal fluid with underlying choroidal and scleral involvement as a greyish-white mass ('abscess') at either the equator or posterior pole. Annular choroidal detachments were reported in 2 of 12 patients with posterior scleritis in a recent series (Cleary, 1975), although shallowing of the anterior chamber was not noted.

ANGLE-CLOSURE GLAUCOMA AND SCLERITIS

Glaucoma has not been noted as a frequent complication of scleritis. Watson and Hayreh (1976) reported open-angle glaucoma in 11.62% (35 eyes), all of which had anterior scleritis alone. McGavin et al. (1976) found bilateral closed-angle glaucoma in 1 patient with rheumatoid episcleritis which settled after bilateral peripheral iridectomies. In addition, these authors found 9 eyes (18.7%) with rheumatoid scleritis and raised intraocular pressure (which by inference, though not stated, had open angles), and in most of these cases the glaucoma appeared to settle with the scleritis.

Apart from these large series of eyes with scleritis there have been isolated reports of what may have been angle-closure glaucoma secondary to scleritis. Brockhurst et al. (1960) mentioned in discussion 1 patient who developed angle-closure glaucoma associated with an extensive choroidal detachment. Gass (1967) described a case of angle-closure glaucoma secondary to inflammatory pseudotumour. This was a case of a 55-year-old woman with a 44-year history of progressive visual disturbance, serous...
retinal detachment, diffuse choroidal and ciliary body thickening, secondary narrow-angle glaucoma, and mild proptosis (he also mentioned two other cases presenting with similar symptoms). Histological examination showed infiltration of choroid and ciliary body by masses of predominantly immature lymphocytes, reticulum cells, and plasma cells. There was retinal pigment epithelial metaplasia, uveal tract infiltrate into the optic nerve, sclera, episclera, and retrobulbar tissues. Hogan and Zimmerman (1962) diagnosed chronic reactive lymphoid hyperplasia in 2 eyes which showed similar histopathological findings.

As well as his earlier histopathological report Gass (1974) reported a case with clinical features similar to our patients: a 31-year-old man who complained of a red eye and misty vision for 2 weeks. Examination revealed angle-closure glaucoma associated with posterior scleritis, which extended from the limbus to behind the equator in the superotemporal quadrant of the eye. There was a large subretinal mass in the inferotemporal quadrant with overlying subretinal fluid. Fluorescein angiography showed 'staining on the surface of the mass' and eventual staining of the subretinal fluid. The angle opened and the choroidal mass disappeared within 5 days of beginning treatment with subconjunctival and systemic corticosteroids.

Phelps (1974) reported 2 cases with recurrent attacks of unilateral angle-closure glaucoma. He considered that the likely diagnosis was recurrent cyclitis or scleritis with associated spasm and swelling of the ciliary body. Although Phelps's cases differed from those reported here, in that his patients suffered recurrent attacks and did not have evidence for posterior scleritis, it is likely that they fell into the same clinical spectrum as ours. Both of Phelps's
cases were treated successfully with local and systemic steroids and cycloplegics rather than miotics.

MECHANISM OF THE GLAUCOMA
Secondary angle-closure (pupil block) glaucoma was suggested for each of our cases by the association of a unilateral shallow anterior chamber, anterior segment inflammation, and anterior choroidal effusion. Kolker and Hetherington (1976) list a number of possible causes for this type of glaucoma—miotics, iris bombe, subluxed or swollen lens, panretinal photocoagulation, and malignant glaucoma—all of which were excluded in our cases by examination. The diagnosis of ‘scleritis’ was made by history, physical signs, and response to specific anti-inflammatory treatment. The cause of the shallow anterior chamber in each case would appear to be anterior rotation of the ciliary body at the scleral spur following development of the choroidal effusion. Such a mechanism was demonstrated by Chandler and Grant (1965), and thought to be the cause of angle closure following scleral buckling for detachment surgery (Smith, 1967; Sebastyan et al., 1962).

Reversal of the angle closure and deepening of the anterior chamber occurred within 24 hours after treatment with cycloplegics. The glaucoma resolved within 7 days while on anti-inflammatory treatment. This slight delay might reflect a co-existent trabecular obstruction induced by the scleritis, as in the cases noted by Watson and Hayreh (1976) in eyes with anterior scleritis; it might also suggest that in our cases the abnormally high intraocular pressures were caused in part by secondary angle-closure glaucoma and in part by secondary open-angle glaucoma.

Treatment
Recognition that angle-closure glaucoma can be secondary to posterior scleritis is important, as treatment is different from that given for primary angle closure. Case 3 was initially treated with pilocarpine without success. As pilocarpine induces shallowing of the anterior chamber (Poinooasawamy et al., 1976) the glaucoma may even have been made worse. In contrast treatment with cycloplegics appeared to be successful in deepening the anterior chamber, breaking pupil block, and allowing the anterior chamber angle to open.

We found that treatment needed to be directed towards the scleritis—topical steroids, oxyphenbutazone, and, where necessary, systemic steroid, as well as the glaucoma—cycloplegics and where necessary acetazolamide. The approach was uniformly successful in bringing about a rapid resolution of the condition.

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For permission to use Fig. 4 we are indebted to W. B. Saunders Co Ltd.

References


