

Acute Angle Closure Glaucoma Resulting from Myelodysplasia

G S Ang, MRCOphth*, A Tey, MRCOphth**, W S Ng, MBChB*, V Subrayan, FRCOphth*

*Department of Ophthalmology, Raigmore Hospital, Inverness, **Department of Ophthalmology, Princess Alexandra Eye Pavilion, Edinburgh, United Kingdom

SUMMARY

Patients with bleeding diatheses can present in a variety of clinical situations. When these patients manifest with ocular complications, their management can be challenging. We describe a case of acute angle closure glaucoma secondary to subretinal haemorrhage, with myelodysplasia as a predisposing factor.

KEY WORDS:

Acute angle closure glaucoma, Intraocular pressure, Myelodysplasia, Age-related macular degeneration, Choroidal neovascularisation, Subretinal haemorrhage

INTRODUCTION

Angle closure glaucoma is a rare complication of subretinal neovascularisation resulting in haemorrhagic retinal detachment^{1,2}. Predisposing factors for haemorrhage are documented in most of the reported cases^{1,2}. We present a case of acute angle closure glaucoma secondary to subretinal haemorrhage, with myelodysplasia as a predisposing factor.

CASE REPORT

A 93-year old lady presented to Eye Casualty with a 5-day history of a painful red left eye associated with nausea and headache. Ten years previously, she was diagnosed with bilateral untreatable age-related macular degeneration. As her vision was 6/24 right eye and hand movements left eye, she was registered partially sighted. Her past medical history included myelodysplasia and was managed conservatively with regular red cell and platelet transfusions.

On presentation, her vision was counting fingers right eye, and no perception of light left eye. The right eye was quiet, but the left eye showed classical signs of angle closure glaucoma: congested conjunctiva, corneal oedema, shallow anterior chamber with cells and flare, mid-dilated pupil, and congested iris vessels. The intraocular pressure (IOP) was 28mmHg in that eye. Examination of the drainage angles with gonioscopy revealed that angles in the left eye were closed. However, gonioscopy in the right eye revealed open and non-occludable drainage angles. A secondary cause for the angle closure in the left eye was thus suspected. There was a dense cataract, but it was not subluxed or dislocated. She was thus commenced on treatment with intravenous acetazolamide 500mg, topical pilocarpine 4%, topical timolol

0.5%, topical apraclonidine 0.5%, and topical prednisolone 1%, which reduced IOP. On admission, she had a very low platelet count of $15 \times 10^9/l$. To exclude any secondary causes of the acute glaucoma, B-scan ultrasound was performed. In the right eye, no abnormalities were seen. In the left eye, there was massive subretinal haemorrhage occupying the posterior segment. (Figure 1) No intraocular masses were seen.

Subsequently, laser peripheral iridotomy was performed, and the anterior chamber deepened immediately. Gonioscopy after peripheral iridotomy showed open angles. The left IOP dropped to 14mmHg, and the patient was discharged home on topical pilocarpine 4% four times daily, topical prednisolone 1% twice daily, and topical Cosopt (combination of dorzolamide 2% and timolol 0.5%). There has been no recurrence to date for the past 24 months.

DISCUSSION

In the normal eye, the aqueous is produced by the ciliary body epithelium, and flows from the posterior chamber (space between the lens and iris) into the anterior chamber through the pupil. It is then drained via the trabecular meshwork at the angle formed between the sclera and peripheral iris. Angle closure glaucoma occurs when the aqueous drainage angle is occluded, obstructing aqueous outflow, and causing elevation of IOP. Acute angle closure glaucoma is an ophthalmic emergency, as prolonged raised IOP may result in irreversible ischaemic changes to the optic nerve head and other ocular structures. Management of acute angle closure glaucoma is two-fold: rapid reduction of the acutely raised IOP medically or surgically, and the prevention of future episodes. Prevention is achieved by peripheral iridotomy, where an opening in the peripheral iris is made by laser. This establishes a direct connection between the posterior and anterior chambers for aqueous flow, thus preventing further attacks.

Angle closure glaucoma as a result of haemorrhagic retinal or choroidal detachment is uncommon^{1,2}. The haemorrhagic detachment causes anterior displacement of the vitreous body and iris-lens diaphragm, with subsequent obliteration of the anterior chamber drainage angle, thus resulting in angle closure glaucoma. In age-related macular degeneration, this is caused by bleeding from choroidal neovascularisation, with consequent localised retinal detachment². In patients with impaired haemostasis, the haemorrhage continues and results in a massive generalised retinal detachment.

This article was accepted: 10 May 2007

Corresponding Author: G S Ang, Specialist Registrar Ophthalmology, Eye Clinic, Aberdeen Royal Infirmary, Foresterhill, Aberdeen AB25 2ZN



Fig. 1: B-scan ultrasound display of the left eye showing massive subretinal haemorrhage occupying the posterior pole and resulting in haemorrhagic retinal detachment

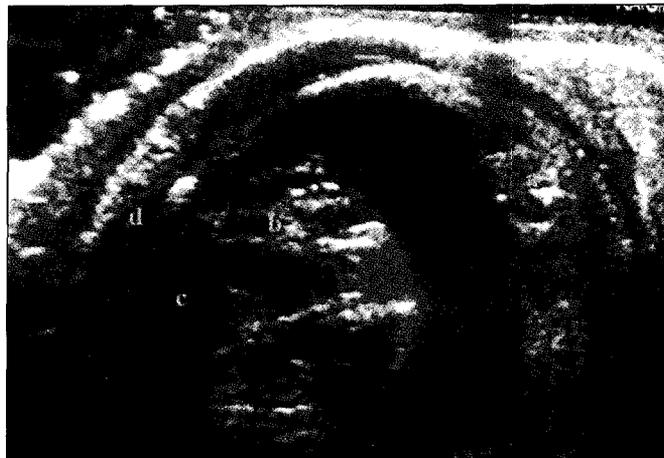


Fig. 2: B-scan ultrasound display of the left eye showing massive subretinal haemorrhage occupying the posterior pole and resulting in haemorrhagic retinal detachment

Massive haemorrhage from oral anticoagulants and systemic bleeding tendencies has been previously documented¹. Myelodysplasia is a group of acquired bone marrow disorders, usually occurring in the elderly, and is due to stem cell defects, characterised by increasing bone marrow failure with abnormalities of all myeloid cell lines. This results in pancytopenia, and patients usually present with anaemia, infection or bleeding. In our patient, her low platelet count resulted in impaired haemostasis, and she had superficial bruising throughout her body.

When faced with a patient with angle closure glaucoma, it is important to establish if it is primary or secondary. In our patient, we suspected a secondary cause because gonioscopy in the unaffected eye showed wide-open drainage angles. As funduscopy was not possible due to the corneal oedema and dense cataract, B-scan ultrasound was performed. Although massive retinal detachment with subretinal haemorrhage was shown and there were no other intraocular masses seen, it was not possible to completely rule out a small neoplastic lesion. Intraocular tumours should always be considered as a cause of secondary angle closure glaucoma. Based on our patient's history of age-related macular degeneration, myelodysplasia resulting in impaired haemostasis, and negative echographic findings, we concluded that the source of bleeding was likely to be from choroidal neovascularisation.

Treatment of patients with angle closure glaucoma secondary to a massive subretinal haemorrhage associated with impaired haemostasis can be difficult. In our patient, the intraocular

pressure was controlled sufficiently with medical treatment and laser peripheral iridotomy. This was often not possible in the other reported cases, which subsequently required further laser or surgical intervention to reduce IOP^{1,2}. Laser iridotomy should be performed with great care to avoid the iris blood vessels, to avoid causing massive hyphaema in patients with bleeding tendencies. Despite the prophylactic peripheral iridotomy, there is a possibility that our patient may experience a recurrence in the future if there is further massive subretinal haemorrhage.

In conclusion, although uncommon, clinicians should consider the possibility of secondary acute angle closure glaucoma in a patient with bleeding diathesis who has a painful red eye, especially when associated with a headache. Although these eyes generally have poor visual potential, referral to the ophthalmologist should be made promptly. Management should be aimed at providing symptomatic relief by lowering the intraocular pressure, and preventing further recurrences.

REFERENCES

1. Chen SN, Ho CL, Ho JD, Guo YH, Chen TL, Chen PF. Acute angle-closure glaucoma resulting from spontaneous hemorrhagic retinal detachment in age-related macular degeneration: case reports and literature review. *Jpn J Ophthalmol* 2001; 45: 270-5.
2. Pesin SR, Katz LJ, Augsburger JJ, Chien AM, Eagle RC Jr. Acute angle-closure glaucoma from spontaneous massive hemorrhagic retinal or choroidal detachment. An updated diagnostic and therapeutic approach. *Ophthalmology* 1990; 97(1): 76-84.
3. Brown GC, Tasman WS, Shields JA. Massive subretinal hemorrhage and anticoagulant therapy. *Can J Ophthalmol* 1982; 17: 227-30.