

# Letters to the Editor

## Clinical Case Notes

### A visual field abnormality: ocular or cerebral cause?

#### ABSTRACT

The visual field can be affected by disorders involving any part of the visual pathway, from the retina to the striate cortex. The pattern of visual field damage can indicate the location of the disorder. The case is presented here of an 8-year-old girl with congenital glaucoma. Initial perimetry appeared consistent with glaucomatous field loss; however, this evolved into a homonymous quadrantanopia, indicating the presence of a cerebral lesion as the cause. Computed tomography and magnetic resonance imaging scans detected a large middle cranial fossa arachnoid cyst. Postoperatively there was resolution of the visual field defects secondary to the arachnoid cyst. The pattern of field loss was pivotal in this patient's management. Attention must be paid when caring for the patient with a chronic disease to ensure that concurrent conditions are diagnosed.

**Key words:** arachnoid cyst, congenital glaucoma, middle cranial fossa, visual field.

#### INTRODUCTION

Perimetry is an important tool in the diagnosis and management of many ocular<sup>1,2</sup> and cerebral<sup>3–7</sup> conditions. The visual field can be affected by disorders involving any part of the visual pathway.<sup>8</sup> Depending on the location of a disorder, the visual field can manifest a different and characteristic pattern of abnormality.<sup>8</sup>

We report a case of a patient with congenital glaucoma who developed a visual field defect. The field defect progressed and this led to the diagnosis of a large intracranial arachnoid cyst. The features of this condition are described.

#### CASE REPORT

Our patient is a young girl who was initially examined at the age of 6 days and was found to have bilateral buphthalmos and intraocular pressures (IOPs) of 42 mmHg in her right eye and 54 mmHg in her left eye. Her congenital glaucoma was managed by bilateral trabeculotomy and, following this, her condition remained under control until the age of 5 years. During this time her IOP was in the low 20s in her right eye and in the high teens in her left eye. Her cup-to-disc ratio (CDR) was 0.3 in the right eye, with no significant cupping in the left.

At each of her reviews, between the ages of 5 and 7 years, her visual acuity remained good at 6/6 right and left; however, she complained of right frontal and right periorbital headaches. These

were felt to be due to rising right IOP, which by now was in the low 30s and continued to remain at this level despite escalating antiglaucomatous therapy. By the age of 7 years she required lantanoprost and timolol in her right eye. Her CDR was noted to be 0.6 in the right eye and 0.1 in the left eye, so dorzolamide was added to the right eye. Computerized perimetry showed visual field loss. This was felt to represent a superior nasal step in the right eye and an early superior arcuate scotoma in the left (Fig. 1).

At the age of 8 years, she continued to report right-sided headaches and ocular discomfort. Her right IOP was in the mid 20s and her left had begun to rise into low 20s. Brimonidine was added to the combination in her right eye. Latanoprost was also commenced in her left eye. Perimetry was repeated, showing a substantial increase in the area and the depth of the visual field loss in both eyes despite improved IOP control (Fig. 2). This field loss appeared to respect the vertical meridian, but not the horizontal.

Neuro-imaging was arranged to exclude a neurological cause. An magnetic resonance imaging (MRI) scan of the patient's brain showed the presence of a large right middle cranial fossa arachnoid cyst, causing thinning and expansion of the right parietal bone, shift of midline structures and enlargement of the ventricular system (Fig. 3). A neurosurgical opinion was sought. She subsequently had marsupialization and fenestration of the arachnoid cyst. This involved a craniotomy and gentle retraction of the right temporal lobe to expose the arachnoid cyst lying deep to it. As much of the arachnoid cyst membrane was removed as possible. The neck of the cyst was explored to determine where it communicated with the basal cisterns. A large opening was then made into

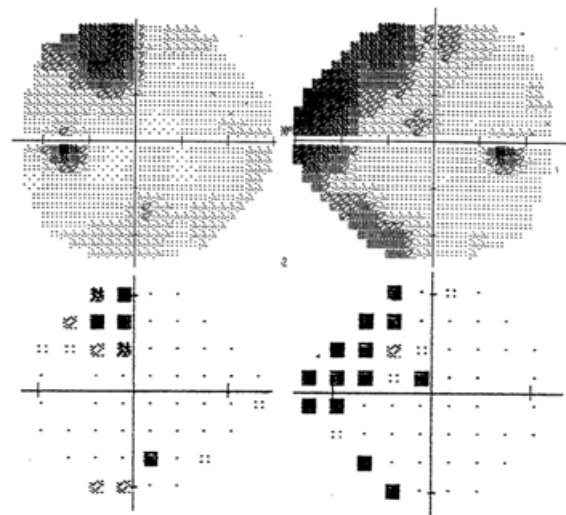


Figure 1. Grey scale and pattern deviation plots of visual fields at age 7 years.

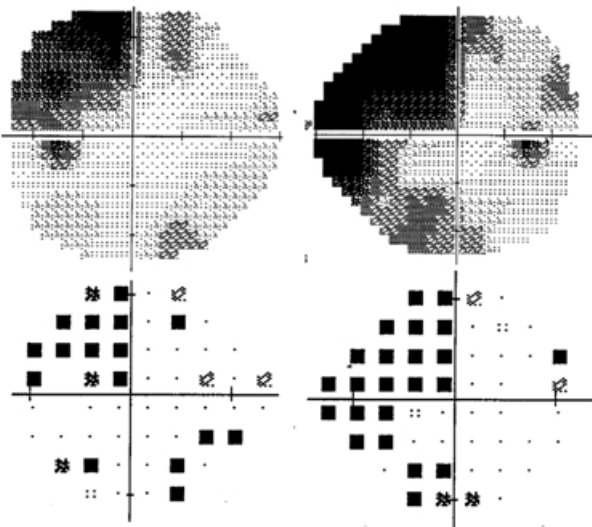


Figure 2. Grey scale and pattern deviation plots of visual fields at age 8 years, preoperatively.

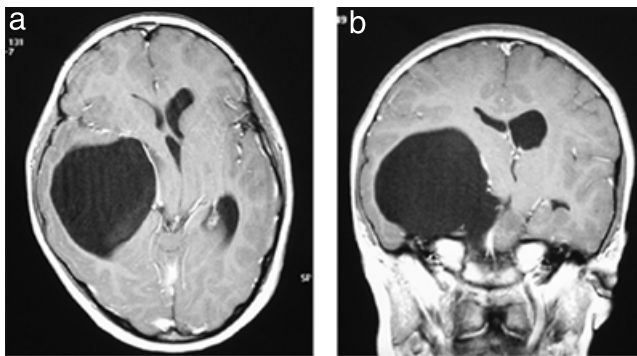


Figure 3. (a) Axial and (b) coronal T1-weighted magnetic resonance images of right middle cranial fossa arachnoid cyst at age 8 years, preoperatively.

the subarachnoid space in front of the brainstem, to try to prevent the cyst from recurring.

Four months postoperatively, her symptoms of headache and right ocular discomfort had resolved. Visual acuity remained unchanged (at 6/6 right and left), as did the optic disc appearance (Fig. 4). However, the IOP had decreased. At her most recent review it was 14 mmHg in her right eye and 15 mmHg in her left eye. Therefore her medications were gradually tapered. A postoperative visual field test showed a complete resolution of field loss in the left eye and residual superonasal field abnormalities in the right (Fig. 5). A postoperative MRI scan showed a dramatic reduction in the size of the arachnoid cyst, with re-expansion of the affected cerebral hemisphere and resolution of the midline shift (Fig. 6). Currently, she remains asymptomatic.

## DISCUSSION

The management of congenital glaucoma may pose certain challenges. Accurate assessment of the visual field may only be

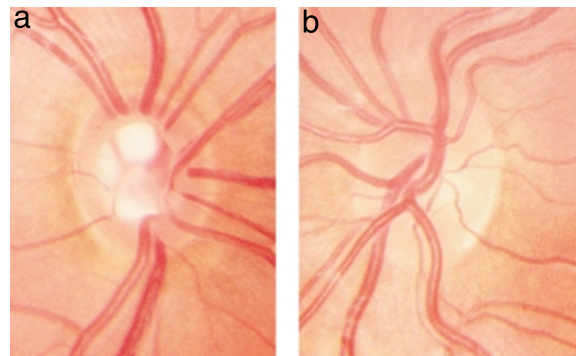


Figure 4. (a) Right and (b) left disc photographs, 4 months post-operatively.

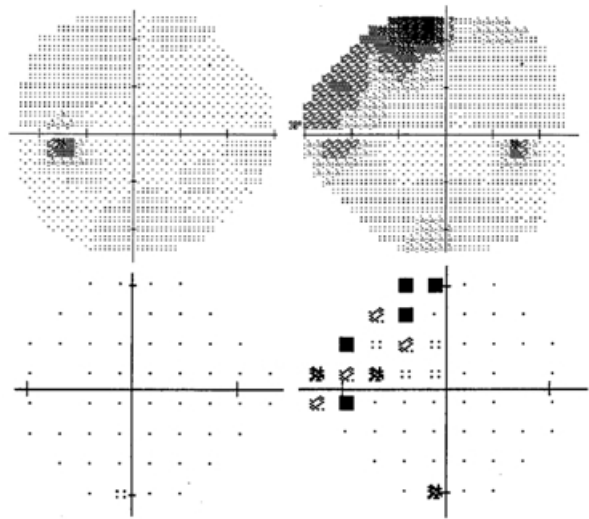


Figure 5. Grey scale and pattern deviation plots of visual fields, 4 months postoperatively.

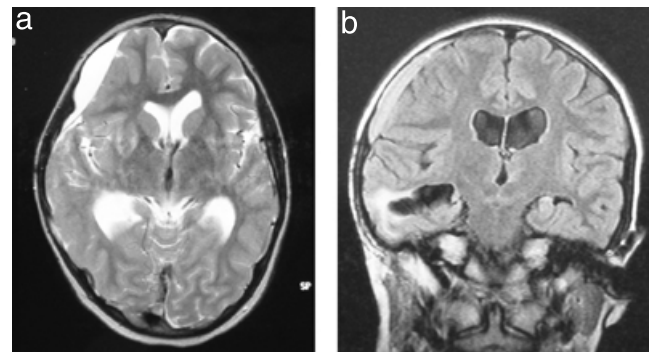


Figure 6. (a) Axial T2-weighted and (b) coronal T1-weighted magnetic resonance images of right middle cranial fossa arachnoid cyst, 6 months postoperatively.

possible when the patient becomes old enough to perform perimetry reliably.<sup>9</sup> Our patient had her first field test at the age of 7 years, although reliable fields have been performed in patients

as young as 4 years of age.<sup>9</sup> The pattern of field loss may resemble that seen in adult glaucomas,<sup>10,11</sup> with nasal step and arcuate scotoma being a common finding.<sup>9</sup> This pattern, however, is a reflection of the location of visual pathway damage. Glaucomatous optic neuropathy occurs when retinal ganglion cell axons become compromised as they pass through the lamina cribrosa.<sup>12–14</sup> This produces a pattern of field loss that respects the horizontal meridian.<sup>10</sup> However, conditions that affect the visual pathway at a retrochiasmal location may cause field loss respecting the vertical meridian but not the horizontal.<sup>4,15</sup>

Our patient had field loss that represented a left superior homonymous quadrantanopia with some degree of incongruity between eyes. A common site for a causative lesion responsible for this field loss is the occipital lobe.<sup>16</sup> However, lesions affecting the temporal lobe are more likely to produce field loss that displays incongruity between eyes.<sup>15,17,18</sup> Typically, the visual field ipsilateral to the lesion demonstrates a superonasal deficit, which is a larger and denser scotoma than that in the contralateral visual field. This may be due to fibres projecting from the retina ipsilateral to the lesion lying more laterally in the temporal lobe than those from the contralateral retina.<sup>17,18</sup> Therefore, they may be more prone to injury from extra-axial lesions in the middle cranial fossa. Lesions such as arachnoid cysts that abut the temporal lobe comprise at least one-half of adult and one-third of paediatric intracranial arachnoid cysts.<sup>19</sup> They are congenital lesions that arise during development from splitting of the arachnoid membrane.<sup>20–22</sup> Most arachnoid cysts that become symptomatic do so in early childhood.<sup>23</sup> Symptoms of arachnoid cysts may include headaches, seizures, visual field loss or hemiparesis.<sup>23,24</sup> Symptomatic cysts may require craniotomy for marsupialization and fenestration, or even cyst shunting.<sup>25–27</sup>

Retrospectively, our patient showed numerous symptoms consistent with an expanding right middle cranial fossa arachnoid cyst. However, until the visual field showed a convincing left superior quadrantanopia, her symptoms were explainable by other conditions including her glaucoma. The arachnoid cyst was associated with a displaced cerebral midline and an enlarged ventricular system. Therefore, intracranial pressure would have been expected to be raised. However, this patient never developed papilloedema. We cannot account for this, although raised intracranial pressure in the absence of papilloedema has been reported,<sup>28</sup> and features of raised intracranial pressure on computed tomography have been shown not to correlate with measured intracranial pressure.<sup>29</sup> To our knowledge, there is no association between congenital glaucoma and the occurrence of arachnoid cysts. (No previous cases were found with a Medline search using the following keywords: glaucoma, congenital glaucoma, arachnoid cyst, intracranial cyst, middle cranial fossa cyst.)

Following surgery, our patient's field loss had largely resolved, such that the left eye had a normal visual field and the right eye had only residual superonasal field loss. It may be that the field loss secondary to the arachnoid cyst had resolved<sup>30</sup> and the remaining defect in the right visual field is a superior nasal step, which reflects the degree of glaucomatous optic neuropathy in the right eye.

In summary, a case is presented of a young girl with congenital glaucoma who coincidentally developed an arachnoid cyst. The pattern of visual field loss was pivotal in her management. Care must be taken in the interpretation of visual fields in patients with glaucoma, so that disorders of the visual pathway causing field loss are not overlooked.

## ACKNOWLEDGEMENTS

We would like to thank Associate Professor Glen Gole, Associate Professor Michael Besser and Dr Peter Stein for their contribution to the management of this patient.

John Landers MB BS MPH,<sup>1</sup> K C Tang FRANZCO<sup>1</sup>  
and Stephen Hing FRANZCO<sup>1,2</sup>

<sup>1</sup>Orange Eye Centre, Orange and <sup>2</sup>Children's Hospital at Westmead, Westmead, New South Wales, Australia

## REFERENCES

- Kramer SG. The peripheral visual field in glaucoma: Re-evaluation in the age of automated perimetry. *Surv Ophthalmol* 1991; **36**: 59–9.
- Kelter JL, Johnson CA, Spurr JO, Beck RW, Optic Neuritis Study Group. Baseline visual field profile of optic neuritis: the experience of the optic neuritis treatment trial. *Arch Ophthalmol* 1993; **111**: 231–4.
- McFadzem R, Brosnahan D, Hadley D, Mutlukan E. Representation of the visual field in the occipital striate cortex. *Br J Ophthalmol* 1994; **78**: 185–90.
- Grochowicki M, Vighetto A. Homonymous horizontal sectoranopia: report of four cases. *Br J Ophthalmol* 1991; **75**: 624–8.
- Johnson CA, Kelter JL. Automated suprathreshold status perimetry. *Am J Ophthalmol* 1980; **89**: 731–41.
- Li SG, Spaeth GL, Scimeca HA, Schatz NJ, Savina PJ. Clinical experiences with the use of an automated perimeter (Octopus) in the diagnosis and management of patients with glaucoma and neurologic diseases. *Ophthalmology* 1979; **86**: 1302–12.
- McCrary JA, Feigon J. Computerized perimetry in neuro-ophthalmology. *Ophthalmology* 1979; **86**: 1287–301.
- Miller NR. Topical diagnosis of lesions in the visual sensory pathway. In: Miller NR, ed. *Walsh and Hoyt's Clinical Neuro-Ophthalmology*, Vol. 1, 4th edn. Baltimore: Williams & Wilkins, 1982; 108–52.
- de Souza EC, Berezovsky A, Morales PH, de Arruda Mello PA, de Oliveira Bonomo PP, Salomao SR. Visual field defects in children with congenital glaucoma. *J Pediatr Ophthalmol Strabismus* 2000; **37**: 266–72.
- Hart WM, Becker B. The onset and evolution of glaucomatous visual field defects. *Ophthalmology* 1982; **89**: 268.
- Heijl A, Lundqvist L. The frequency distribution of earliest glaucomatous visual field defects documented by automated perimetry. *Acta Ophthalmol* 1984; **62**: 658–64.
- Hernandez MR, Pena JD. The optic nerve head in glaucomatous optic neuropathy. *Arch Ophthalmol* 1997; **115**: 389–95.
- Quigley HA, Addicks EM, Green WR, Maumenee AE. Optic nerve damage in human glaucoma II. The site of injury and susceptibility to damage. *Arch Ophthalmol* 1981; **99**: 635–40.
- Fetchner RD, Weinreb RN. Mechanisms of optic nerve damage in primary open angle glaucoma. *Surv Ophthalmol* 1994; **39**: 23–42.
- Lessell S, Lessell AM, Glasser JS. Topical diagnosis: retrochiasmal visual pathways and higher cortical function. In: Tasman W, Jaeger EA, eds. *Duane's Clinical Ophthalmology*, Vol. 2. Philadelphia: JB Lippincott, 1993; 1–7.

16. Jacobson DM. The localising value of a quadrantanopia. *Arch Neurol* 1997; **54**: 401–4.
17. Hughes TS, Abou-Khalil B, Lavin PJM, Fakhoury T, Blumenkopf B, Donahue SP. Visual field defects after temporal lobe resection: a prospective quantitative analysis. *Neurology* 1999; **53**: 167–72.
18. Egan RA, Shults WT, So N, Burchiel K, Kellogg JX, Salinsky M. Visual field deficits in conventional anterior temporal lobectomy versus amygdalohippocampectomy. *Neurology* 2000; **55**: 1818–22.
19. Felipe A, Gianuotta SL. Arachnoid cyst rupture producing subdural and intracranial hypertension. Case reports. *Neurosurgery* 1997; **41**: 951–6.
20. Van Der Meche F, Braakman R. Arachnoid cysts in the middle cranial fossa: cause and treatment of progressive and non-progressive symptoms. *J Neurol Neurosurg Psychiatry* 1983; **46**: 1102–7.
21. Mayr U, Aichner F, Bauer G *et al.* Supratentorial extracerebral cysts of the middle cranial fossa: a report of 23 consecutive cases of the so-called temporal lobe agenesis syndrome. *Neurochirurgia* 1982; **25**: 51–6.
22. Rengachary SS, Watanabe I. Ultrastructure and pathogenesis of intracranial arachnoid cysts. *J Neuropath Exp Neurol* 1981; **40**: 61–83.
23. Harsh GR, Edwards MSB, Wilson CB. Intracranial arachnoid cysts in children. *J Neurosurg* 1986; **64**: 835–42.
24. Passero S, Filisomi G, Cioni R, Venturi C, Volpini B. Arachnoid cysts of the middle cranial fossa: a clinical, radiological and follow-up study. *Acta Neurol Scand* 1990; **82**: 94–100.
25. Caldarelli M, Di Rocco C. Surgical options in the treatment of interhemispheric arachnoid cysts. *Surg Neurol* 1996; **46**: 212–21.
26. Von Wild K, Gullotta F. Arachnoid cyst of the middle cranial fossa – aplasia of temporal lobe? *Childs Nerv Syst* 1987; **3**: 232–4.
27. Richard KE, Dahl K, Sanker P. Long-term follow-up of children and juvenile with arachnoid cysts. *Childs Nerv Syst* 1989; **5**: 184–7.
28. Spence JD, Armacher AL, Willis NR. Benign intracranial hypertension without papilloedema. Role of 24-hour cerebrospinal fluid pressure monitoring in diagnosis and management. *Neurosurgery* 1980; **7**: 326–36.
29. Von Wild K. Arachnoid cysts of the middle cranial fossa. *Neurochirurgia* 1992; **35**: 177–82.
30. Gau M, Nestler A, Dietrich J, Faude F. [A retrosellar arachnoid cyst as a rare cause of homonymous hemianopsia.] *Klin Monatsbl Augenb* 1998; **212**: 480–81 (in German).

## Incomplete and complete plateau iris syndrome

### ABSTRACT

Plateau iris syndrome is considered to be a rare entity and has been classified into two subtypes, the complete and the incomplete forms. Herein a case is reported of plateau iris syndrome that had a postmydriatic rise in intraocular pressure (IOP) in one eye (complete syndrome) and no increase in IOP in the other

(incomplete syndrome). The pre- and post-mydriasis ultrasound biomicroscopic findings were documented. These findings suggest that incomplete plateau iris syndrome may be caused by a difference in the extent of closure of the angle circumferentially over the 360 degrees of the angle.

**Key words:** plateau iris syndrome, ultrasound biomicroscopy.

### INTRODUCTION

Plateau iris configuration and syndrome were reported by Wand *et al.* in 1977.<sup>1</sup> Plateau iris syndrome is defined as a postoperative condition in which iridectomy has removed the relative pupillary block but gonioscopically confirmed angle closure occurs without a shallow anterior chamber axially.<sup>1</sup> The syndrome can be complete (closure involves the whole of the meshwork and the intraocular pressure [IOP] will rise) or incomplete (closure involves only the posterior trabecular meshwork and the IOP will not increase) depending on the increase in IOP following a post-mydriatic appositional closure.<sup>2</sup> However, Epstein suggested that the IOP elevation following mydriasis may be related to functional angle closure around the circumference of the angle.<sup>3</sup>

Ultrasound biomicroscopy (UBM) is a technique that offers an objective way of assessing the anatomy of angle of the anterior chamber and the ciliary processes. UBM uses high frequency ultrasound (50–100 MHz) to produce images of the anterior segment at a high resolution (20–50 µm), that allows structural details of the angle and the ciliary body to be visualized and measured at a microscopic resolution.<sup>4–6</sup>

### CASE REPORT

A 58-year-old man with diabetes presented for a routine check up. His vision was 6/6, N6 in both eyes. The IOP on applanation tonometry was 26 mmHg in the right eye and 25 mmHg in the left eye. Gonioscopy with a Sussman goniolens revealed appositional closure of the angle over 360° in both the eyes. The angles were open to scleral spur on indentation in both the eyes.

The patient underwent an uneventful laser iridotomy (10 o'clock and 1 o'clock quadrant for right and left eyes, respectively) in both eyes on the same day. A week later his IOP was 22 mmHg right eye and 21 mmHg left eye, gonioscopy revealed the angles to be narrow but open. Dilated evaluation of the disc and the visual fields were within normal limits, and he had early non-proliferative diabetic retinopathy. Over the next 2 years his intraocular pressures in both the eyes were maintained in the low to mid twenties and the disc and the visual fields remained stable. In the meantime he had undergone four dilated evaluations. The post-dilated IOP was recorded on two of these occasions and did not show a significant increase.

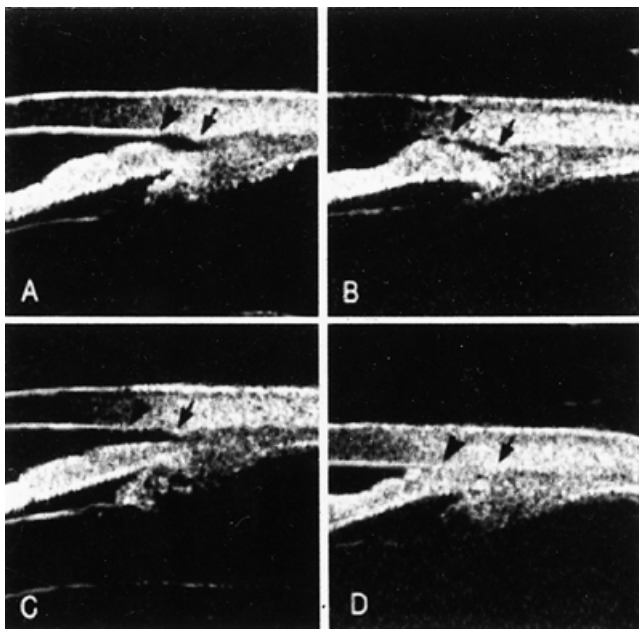
During a subsequent visit his IOP without any treatment was 22 mmHg in the right eye and 23 mmHg in the left eye. The pupils were dilated with topical 5% phenylephrine eye drops for a dilated evaluation. The IOP rose to 29 mmHg in the right eye and was 22 mmHg in the left eye following the dilation.

As part of a prospective study, we were performing UBM evaluation in primary angle closure glaucoma patients following a laser iridotomy before and after dilation. Therefore, UBM was performed on this patient.

**Table 1.** Ultrasound biomicroscopy parameters pre- and post-dilation

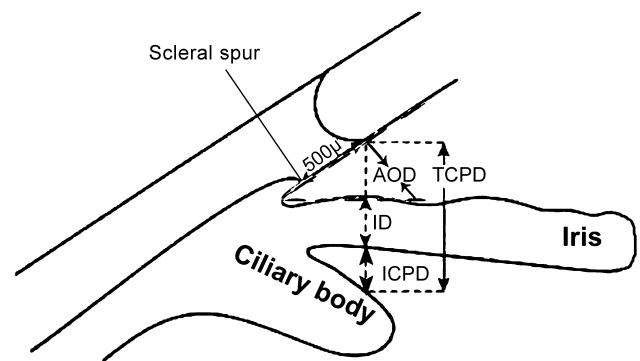
Quadrant	Angle opening distance ( $\mu\text{m}$ )		Trabecular CP distance ( $\mu\text{m}$ )		Iris distance ( $\mu\text{m}$ )		Iris CP distance ( $\mu\text{m}$ )	
	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Right eye								
Superior	231	104	648	845	370	625	47	116
Temporal	185	0	532	567	347	463	0	104
Inferior	69	0	486	602	417	602	0	0
Nasal	162	197	602	822	440	579	0	46
Left eye								
Superior	162	58	845	683	521	532	162	83
Temporal	116	0	613	544	475	544	22	0
Inferior	81	266	590	741	509	475	0	0
Nasal	231	150	799	579	486	429	82	0

CP, ciliary process; Pre, pre-dilation; Post, post-dilation.



**Figure 1.** Ultrasound biomicroscopy images of the right eye. (a) Pre- and (b) post-dilation images of the superior quadrant showing narrowing without closure of the angle. (c) Pre- and (d) post-dilation images of the temporal quadrant showing closure of the angle.

The pre- and post-dilation UBM parameters are given in Table 1. Figure 1 shows pre- and post-dilation UBM images of the right eye. The superior quadrant shows narrowing without closure of the angle (Fig. 1a,b); however, the temporal quadrant shows closure of the angle (Fig. 1c,d). A line diagram showing the parameters measured is shown in Fig. 2. Pre-dilation angle opening distance varied from  $69\ \mu\text{m}$  to  $231\ \mu\text{m}$  in different quadrants. Following dilation the angle opening distance was  $0\ \mu\text{m}$  in two quadrants of the right eye and in one quadrant of the left eye.



**Figure 2.** Line diagram showing the parameters measured. AOD, angle opening distance; ID, iris thickness; ICPD, iris ciliary process distance; TCPD, trabecular ciliary process distance.

## DISCUSSION

In the case presented here, we have looked at the UBM images in the four quadrants before and after dilation and correlated the angle morphology with the post-dilation increase in IOP. Following dilation, there was a greater increase in IOP in the right eye ( $7\ \text{mmHg}$ ) as compared to the left eye ( $1\ \text{mmHg}$ ). Ritch differentiated incomplete and complete plateau iris syndromes based on the increase in IOP following dilation.<sup>2</sup> Our case, we believe, is an example of incomplete plateau iris syndrome in the left eye and complete plateau iris syndrome in the right eye because of the IOP rise following dilation. Pathogenically, Ritch considers the basis for the differentiation between the complete and incomplete forms to be based on the height of the plateau or the level of iris stroma in relation to the angle structures.<sup>2</sup> However, Epstein suggested that circumferential differences in the extent of the angle closure might be responsible for incomplete plateau iris syndrome.<sup>3</sup> In this case, following dilation, the angle closed in two quadrants in the right eye and in one quadrant in the left eye. This correlated with the increase in IOP in the right eye. The extent of the narrowness of the angle also varied as indicated by the variable angle opening distance (Table 1).

This concept of incomplete and complete variants of the plateau iris syndrome being produced by the differences in the extent of angle closure over the circumference of the angle is reported here with UBM documentation for the first time to the best of our knowledge.

G Chandra Sekhar MD, K Sreelatha Onam BOpt and  
Vallam Kunjam BOpt  
VST Centre for Glaucoma Care, LV Prasad Eye Institute,  
Hyderabad, India

## REFERENCES

1. Wand M, Grant WM, Simmons RJ, Hutchison BT. Plateau iris syndrome. *Trans Am Acad Ophthalmol Otolaryngol* 1977; **83**: 122–9.
2. Lowe RF, Ritch R. Angle-closure glaucoma: clinical types. In: Ritch R, Shields MB, Krupin T, eds. *The Glaucomas*. St. Louis: CV Mosby, 1996; 827–9.
3. Epstein DL. Angle closure glaucoma. Evaluation and treatment after iridectomy. In: Epstein DL, Allingham RR, Schuman JS, eds. *Chandler and Grants Glaucoma*, 4th edn. Philadelphia: Lea & Febiger, 1996; 267–73.
4. Pavlin CJ, Harasiewicz K, Eng P *et al*. Ultrasound biomicroscopy of anterior segment structures in normal and glaucomatous eyes. *Am J Ophthalmol* 1992; **113**: 381–9.
5. Pavlin CJ, Ritch R, Foster FS. Ultrasound biomicroscopy in plateau iris syndrome. *Am J Ophthalmol* 1992; **113**: 390–95.
6. Pavlin CJ, Foster FS. Plateau iris syndrome: Changes in angle opening associated with dark, light and pilocarpine administration. *Am J Ophthalmol* 1999; **128**: 288–91.

## Glaucoma from topical corticosteroids to the eyelids

### ABSTRACT

Raised intraocular pressure and glaucoma have rarely been associated with use of periorbital corticosteroids for dermatological conditions such as blepharitis and eczema. Three cases are described in which periorbital topical corticosteroids appear to have resulted in raised intraocular pressure or glaucoma. Topical corticosteroids used for dermatological conditions around the face and eyes are often regarded as being fairly innocuous with regard to ocular side-effects. This case series demonstrates that secondary open-angle glaucoma can be a sight-threatening consequence, and periorbital steroids should therefore be used cautiously and sparingly, particularly in those with a family history of glaucoma. Intraocular pressure may not always return to normal upon cessation of the drug.

**Key words:** blepharitis, corticosteroid, dermatitis, glaucoma, ocular hypertension, topical.

### INTRODUCTION

Both topical ocular corticosteroids and systemic corticosteroids are recognized by ophthalmologists as potentially causing raised

intraocular pressure (IOP) or glaucoma as a side-effect.<sup>1–4</sup> However, topical steroid creams or ointments used for periorbital dermatological conditions such as dermatitis are less well-recognized in having this risk, or are employed by other medical practitioners less aware of this potential.<sup>5–10</sup> We present a series of three cases highlighting this risk.

## CASE REPORTS

### Case 1

A 40-year-old Caucasian man presented in September 1996 with pressures of 40 mmHg in the right eye and 51 mmHg in the left eye, in the context of chronic use of betamethasone dipropionate cream to the eyelids for presumed psoriasis. There was no other significant ocular history or family history of ocular disease or glaucoma. Visual acuity was 6/5 on the right and 6/6<sup>-2</sup> on the left. Anterior segments were normal apart from mild left posterior subcapsular cataract. Angles were open with light trabecular pigmentation and no peripheral anterior synechiae (PAS). No intraocular inflammation was seen. Optic discs demonstrated cup-to-disc ratios of right 0.4 and left 0.8.

The patient initially responded well to cessation of topical steroid and commencement of timolol 0.5% b.d. His IOPs remained normal and stable over the following 5 years. In April 2001, on routine follow up, his IOPs were noted to be 16 mmHg on the right and 29 mmHg on the left. He had recommenced topical steroid use to his eyelids. Despite commencement of latanoprost one drop nocte in both eyes, his pressures remained elevated after 1 month. In addition, he was found to have significant inferonasal visual field loss in the left eye, and had developed a left relative afferent pupillary defect. Given the combination of progressive optic neuropathy and visual field loss, with uncontrolled IOP, he proceeded to a left trabeculectomy with 5-fluorouracil (FU). He achieved a good postoperative result, with stable visual field and optic disc without ongoing topical antihypertensive therapy. A dermatological opinion on the skin condition suggested a diagnosis of steroid rosacea, with no evidence of psoriasis on the face or scalp. The condition responded well to full cessation of topical periorbital steroid use. Progressive left posterior subcapsular cataract will require surgery.

### Case 2

A 71-year-old Caucasian man had a history of left traumatic glaucoma from a long-past blunt injury at football. This had required left trabeculectomy and later cataract extraction, and finally required timolol 0.5%/pilocarpine 4% combination therapy for IOP control at right 19 and left 20 mmHg. He presented in July 2001 with IOPs of right 21 and left 25.5 mmHg having used topical hydrocortisone 1% to the lids for presumed allergic dermatitis. He had used the cream for at least several weeks but had ceased it 2 weeks prior to presentation. An uncle and possibly also his mother had glaucoma. Examination showed an intraocular lens (IOL), past YAG laser capsulotomy, low filtration bleb and a Grade IV angle with no PAS or recession. Two sphincter ruptures from the previous trauma were noted. Cup-to-disc ratios were right 0.5 and left 0.6.

Despite numerous combinations of topical therapy, IOP control could not be regained, field loss increased in the left eye and repeat

trabeculectomy with 5-FU was performed in October 2001. At review in early 2003, IOP was right 23 and left 10 mmHg on treatment of levobunolol and brinzolamide to the right eye only.

### Case 3

A 55-year-old Caucasian man presented in October 2000 with a 10-day history of blurred left vision. He had no history of previous episodes or ocular inflammation. A vague family history suggested progressive vision loss in his mother. He suffered from severe blepharitis, and had been using mometasone 0.1% to the eyelids for 6–9 months prior to presentation. Visual acuities were 6/4 bilaterally, and IOPs were noted to be right 44 mmHg and left 75 mmHg. There was associated corneal oedema but no evidence of intraocular inflammation. Drainage angles were Grade IV bilaterally, with no trabecular pigmentation or PAS. His cup-to-disc ratios were right 0.4 and left 0.5. Perimetry revealed a reproducible loss at one test location in the inferonasal quadrant in the left eye.

He achieved a rapid response to oral acetazolamide 250 mg q.i.d., timolol 0.5% mane and latanoprost nocte. He was instructed to cease the topical steroid use, and in mid-2001 was controlled on timolol 0.25% alone at IOPs of right 19 and left 18 mmHg. However, reapplication of steroid cream resulted in symptoms of ocular 'heaviness' with IOP right 37 and left 30 mmHg. His IOP returned to normal with final cessation of steroid cream, and in early 2003 IOP was right 16 and left 16 mmHg on no topical antihypertensive agents, with stable discs and fields.

### DISCUSSION

Steroid-induced glaucoma is a well-known entity that appears to be heterogeneous in its effect with regard to which individuals experience an elevation in IOP, the degree of elevation of IOP and the fall or otherwise in IOP after cessation of steroid; however, certain patterns have been detected.<sup>1,11,12</sup> The 'steroid response' – the marked elevation in IOP induced by steroids in some individuals – is more commonly found in people with primary open-angle glaucoma or a family history of glaucoma.<sup>1,2,11–13</sup>

At a biochemical level, binding of glucocorticoids to glucocorticoid receptors triggers cell type-specific mRNA changes in human trabecular meshwork cells that result in synthesis of new proteins. These new proteins in turn influence activities of various cytoplasmic proteins, the synthesis of cell surface and extracellular molecules, cytokine production, and growth factors.<sup>14</sup> It remains unclear as to which of these alterations in the activities of components of the human trabecular meshwork cells actually cause a rise in IOP, but several authors have suggested increased aqueous outflow resistance secondary to glycosaminoglycan accumulation in the trabecular meshwork.<sup>1,12</sup>

Severe elevation of IOP following steroid use is seen in 5–6% of normal volunteers as compared to 50% or more of patients with open-angle glaucoma.<sup>1,2</sup> The risk of developing steroid glaucoma is dependent on the concentration,<sup>4</sup> potency, dosage and duration of topical treatment, although these findings may not apply to systemic therapy.<sup>15</sup> The IOP may not return to normal upon cessation of the topical steroid<sup>15,9</sup> (Cases 1 and 2), and a tendency for the pressure to remain elevated in those patients who have been on therapy of long duration has been noted.<sup>16</sup> A steroid response has also been noted in an eye with previous anterior segment trauma.<sup>17</sup>

Topical dermatological corticosteroid potency can be arbitrarily divided into four groups: class I, mild (e.g. hydrocortisone 0.5–1%); class II, moderate (e.g. betamethasone valerate 0.02–0.05%); class III, potent (e.g. mometasone furoate 0.1%); and class IV, very potent (e.g. halcinonide).<sup>18</sup> Our cases involved a range of different steroid potencies, from mild in case 2, to potent in cases 1 and 3. The potency of topical ocular corticosteroids is less well defined, but common potent topical steroids include dexamethasone, betamethasone and prednisolone, while less potent preparations include the progesterone-like agents, such as fluorometholone and medrysone.<sup>19</sup>

Awareness of the possibility of glaucoma from steroids taken by novel routes has recently been heightened by the publication of results related to inhaled betamethasone,<sup>13,20</sup> however, glaucoma induced by corticosteroids applied to the periorbital area remains a rarely recognized entity. Postulated mechanisms for raised IOP or glaucoma in this setting are: direct absorption through the skin of the eyelids; absorption from spill of steroid ointment over the lid margin; or systemic absorption of corticosteroid with distribution to the eye via the circulation.<sup>5</sup>

The prevalence of glaucoma or ocular hypertension in those using topical periorbital steroid has not been studied in any structured way and reports have generally been sporadic cases. In our cases 1 and 3, steroid use was certainly chronic, and in both cases potent steroid formulations were implicated. However, despite up to 9 months of use of mometasone in case 3, and with dangerously high IOPs on presentation, IOP has finally been controlled without topical hypotensive therapy, perhaps in contrast to an earlier report.<sup>16</sup> Normal tension glaucoma would be considered in a differential diagnosis on future appraisal of this eye for the first time, and this possibility should figure in assessment of patients with normal tension glaucoma.<sup>21</sup> There is some suggestion that an eye, once 'primed' for a steroid response, will produce a repeat response more quickly,<sup>1</sup> perhaps as in case 2.

This case series highlights the risk of glaucoma and the potential for permanent ocular damage as a result of topical periorbital corticosteroid use. In each case the steroid therapy was commenced by a non-ophthalmologist, and in at least one case the therapy was inappropriate due to incorrect diagnosis of the skin condition. Patients may also be highly motivated to continue steroid use because of their skin condition, and compliance with instructions to cease must be repeatedly sought. We have also shown that steroid use via this route can be a cause for apparent loss of control in established glaucoma, and changes in medication should be asked for specifically in this setting. Use of topical steroid should be for a specific indication, at the lowest dose and frequency of application, and for the shortest duration of therapy to control the condition being treated, so as to avoid this essentially silent but potentially blinding side-effect. This series provides a timely reminder of the need for regular ophthalmic assessments to screen for glaucoma in all patients on any long-term corticosteroid preparation, particularly if there is evidence of a family history of glaucoma.

Helen M Garrott MB BS and Mark J Walland FRANZCO  
*Department of Ophthalmology, Royal Melbourne Hospital,  
Melbourne, Victoria,  
Australia*

## REFERENCES

1. Armaly MF. Effect of corticosteroids on intraocular pressure and fluid dynamics I. Effects of dexamethasone in the normal eye. *Arch Ophthalmol* 1963; **70**: 98–105.
2. Armaly MF. Effect of corticosteroids on intraocular pressure and fluid dynamics II. Effects of dexamethasone in the glaucomatous eye. *Arch Ophthalmol* 1963; **70**: 492–9.
3. Becker B, Mills DW. Corticosteroids and intraocular pressure. *Arch Ophthalmol* 1963; **70**: 500–7.
4. Podos SM, Krupin T, Asseff C, Becker B. Topically administered corticosteroid preparations. *Arch Ophthalmol* 1971; **86**: 251–4.
5. Kitazawa Y. Increased intraocular pressure induced by corticosteroids. *Am J Ophthalmol* 1976; **82**: 492–5.
6. Cubey RB. Glaucoma following the application of corticosteroid to the skin of the eyelids. *Br J Dermatol* 1976; **95**: 207–8.
7. Vie R. Glaucoma and amaurosis associated with long term application of topical corticosteroids to the eyelids. *Acta Venerol* 1980; **60**: 541–2.
8. Zuger C, Sauters D, Levit F. Glaucoma from topically applied steroids. *Arch Dermatol* 1976; **112**: 1326.
9. Aggarwal RK, Potamitis T, Chong NHV, Guarro M, Shah P, Kheterpal S. Extensive visual loss with topical facial steroids. *Eye* 1993; **7**: 664–6.
10. thoe Schwartzberg GWS, Buys YM. Glaucoma secondary to topical use of steroid cream. *Can J Ophthalmol* 1999; **34**: 222–5.
11. Armaly MF. The heritable nature of dexamethasone-induced ocular hypertension. *Arch Ophthalmol* 1966; **75**: 32–6.
12. Spaeth GL, Rodrigues MM, Weinreb S. Steroid-induced glaucoma: A. Persistent elevation of intraocular pressure B. Histopathological aspects. *Trans Am Ophthalmol Soc* 1977; **75**: 353–81.
13. Mitchell P, Cumming RG, Mackey DA. Inhaled corticosteroids, family history, and risk of glaucoma. *Ophthalmology* 1999; **106**: 2301–6.
14. Polansky JR, Fauss DJ, Nguyen TD. Ophthalmic corticosteroids and steroid glaucoma mechanisms. *Ophthalmic Clinics NA* 1995; **8**: 215–28.
15. Godel V, Feiler-Ofry V, Stein R. Systemic steroids and ocular fluid dynamics. II. Systemic versus topical steroids. *Acta Ophthalmol* 1972; **50**: 664–76.
16. Espildora J, Vicuna P, Diaz E. Cortisone-induced glaucoma: a report on 44 affected eyes. *J Fr Ophthalmol* 1981; **4**: 503–8.
17. Spaeth GL. Traumatic hyphema, angle recession, dexamethasone hypertension, and glaucoma. *Arch Ophthalmol* 1967; **78**: 714–21.
18. Mashford ML, Fischer G, Marks R et al. *Therapeutic Guidelines: Dermatology*. Melbourne: Therapeutic Guidelines Limited, 1999; 21.
19. Terek ME, Spaeth GL. *The Glaucomas: Concepts and Fundamentals*. Philadelphia: Lippincott Williams & Wilkins, 2000; 104.
20. Opatowsky I, Feldman RM, Gross R, Feldman ST. Intraocular pressure elevation associated with inhalation and nasal corticosteroids. *Ophthalmology* 1995; **102**: 177–9.
21. Sugar HS. Low tension glaucoma: a practical approach. *Ann Ophthalmol* 1979; **11**: 1155–71.

## Recovery of vision after bilateral arteritic central retinal artery occlusion

### ABSTRACT

A case is reported of a patient with bilateral central retinal artery occlusion secondary to giant cell arteritis. After treatment the vision recovered in one eye but remained poor in the other eye. Treatments employed are discussed with particular attention drawn to differences in the treatment of each eye and suggestions are made for treatment of similar cases.

**Key words:** central artery occlusion, giant cell arteritis, low molecular weight heparin, temporal arteritis.

### INTRODUCTION

Giant cell arteritis (GCA) is a well recognized cause of visual loss. Such visual loss usually occurs because of anterior ischaemic optic neuropathy but less commonly (in approximately 8.3–15.2% of eyes)<sup>1,2</sup> can be related to central retinal artery occlusion (CRAO). Conversely, 1–2% of CRAO are associated with GCA and almost invariably have a poor outcome.<sup>3</sup> We present an unusual case of bilateral central retinal artery occlusion in association with giant cell arteritis in which vision recovered in one eye but not in the other eye.

### CASE REPORT

A 75-year-old man was referred with a 3-week history of headache and intermittent jaw ache as well as fluctuating left vision for 10 days.

On examination visual acuity was 6/6 in each eye. Pupil reactions were normal. Visual fields were full to confrontation. Fundus examination was unremarkable in each eye. Temporal arteries were thought to be thickened and non-pulsatile to palpation but were non-tender.

Investigations revealed ESR of 70 mm/h, C-reactive protein of 100 mg/L and platelet count  $513 \times 10^9/L$  (normal  $150\text{--}500 \times 10^9/L$ ).

Giant cell arteritis was diagnosed. The patient was admitted to hospital and high dose intravenous steroids (1 g/day methylprednisolone in two divided doses) were administered. GCA was later confirmed with temporal artery biopsy.

The next morning the patient described rapid bilateral simultaneous visual loss while lying in bed. At that time ophthalmic examination revealed hand movement vision only in each eye and markedly attenuated retinal blood vessels consistent with central retinal artery occlusion. There was no frank retinal oedema observed, presumably because the eyes were examined almost immediately after the onset of symptoms and coagulative necrosis of the retina had not had time to develop. There were no optic disc haemorrhages or swelling present.

Immediate treatment consisted of ocular massage, intravenous acetazolamide 500 mg, topical ocular hypotensives (timolol 0.5%, apraclonidine 0.5%) and inhaled carbogen (5% CO<sub>2</sub>) for 2 h. In addition anterior chamber paracentesis was performed – in the left



eye within 15 min of the onset of loss of vision and in the right eye approximately 40 min later. The patient was continued on high dose intravenous steroids and treatment with subcutaneous low molecular heparin (LMWH) was instituted (Clexane 75 mg SC thrice daily).

Six hours after the onset of visual loss the right visual acuity remained at hand movements only but the left visual acuity had improved to 6/6. The next day the left fundus examination was unremarkable but the right retina had attenuated arterioles and was pale with a foveal cherry red spot consistent with an established central retinal artery occlusion.

Three months after the onset of visual loss the right visual acuity remained unchanged at hand movements only and the left visual acuity was stable at 6/6. The right fundus showed optic atrophy but the left fundus examination was unremarkable.

## DISCUSSION

Giant cell arteritis is well-known for its potential to cause severe visual loss, which may be unilateral or bilateral. Usually the visual loss is secondary to anterior ischaemic optic neuropathy but can occasionally be secondary to CRAO. In such circumstances the visual prognosis is almost universally poor.<sup>3</sup>

The decision for aggressive manage of CRAO was based on past anecdotal evidence of a positive response to paracentesis, carbogen and pharmacological lowering of IOP.<sup>4</sup> A recent prospective trial of aggressive stepwise therapeutic management of non-arteritic CRAO showed improvement over controls,<sup>5</sup> however, even with appropriate treatment, visual loss can still ensue.<sup>4,5</sup> The striking closeness of corticosteroid treatment then deterioration of symptoms in giant cell arteritis, as seen in this case, has been previously described.<sup>6-8</sup> Although corticosteroid treatment remains the standard for GCA and vision improvement sometimes occurs after steroid treatment,<sup>9,10</sup> there may be some evolution of ischaemia with steroids. Attention is drawn to the high platelet count in this patient, also well reported in cases of GCA.<sup>11-13</sup> Therefore active anticoagulation or thrombocyte aggregation inhibition was considered in the context of potential evolving ischaemia. Heparin and steroids have been described in the treatment of arteritic anterior ischaemic optic neuropathy and central artery occlusion with anecdotal success.<sup>14,15</sup> Complete arterial occlusion can occur with progression of the hyperplastic process or may be a result of superimposed arterial thrombosis. It has been postulated that systemic anticoagulation might be beneficial during the initial phase of corticosteroid treatment because of recent evidence that anticardiolipin antibodies are present in a higher frequency in patients with GCA.<sup>15</sup>

Low molecular weight heparin was instituted in this case and has the added advantage of prevention of deep vein thrombosis.<sup>16</sup> In addition, a combination of steroid and LMWH has been reported as being beneficial in a previous case of cerebral angitis secondary to GCA.<sup>17</sup> LMWH has a larger elimination half-life than unfractionated heparin, so is sufficiently predictable with the standard dose that monitoring is not routinely required.<sup>18</sup> LMWH is at least as safe and effective as heparin.<sup>18</sup>

In this case we used multiple treatment modalities for the bilateral CRAO our patient experienced. The outcome was good vision in one eye and poor vision in the other eye. The only difference in treatment in the two eyes was the non-simultaneous anterior chamber paracentesis: only the eye that received this treatment earlier recovered useful vision. It also seems possible that

the institution of treatment with LMWH had a positive effect on the outcome in the good eye and such treatment should at least be considered in cases of visual loss secondary to GCA in the future. Finally this case suggests that it is possible to reverse visual loss secondary to CRAO in GCA and any such cases presenting should be treated aggressively as for a nonarteritic CRAO with early paracentesis and consideration given to the possible addition of LMWH to the standard high-dose steroid regimen. Ideally a prospective controlled treatment trial of arteritic CRAO cases would be performed; however, owing to the infrequency of this presentation, anecdotal publications give the only available evidence at present.

Andrew F Riley MB ChB and Neil S Aburn FRANZCO  
Ophthalmology Department, Wellington Hospital, Wellington,  
New Zealand

## REFERENCES

- Gonzalez-Gay MA, Garcia-Porrúa C, Llorca J *et al*. Visual manifestations of giant cell arteritis. Trends and clinical spectrum in 161 patients. *Medicine (Baltimore)* 2000; **79**: 283-92.
- Hayreh SS, Podhajsky PA, Zimmerman B. Ocular manifestations of giant cell arteritis. *Am J Ophthalmol* 1998; **125**: 509-20.
- Kwok AK, Lam DS, Liew CT. Bilateral arteritic central retinal artery occlusion in a Chinese patient. *Aust NZ J Ophthalmol* 1998; **26**: 175-6.
- Atebara NH, Brown GC, Cater J. Efficacy of anterior chamber paracentesis and carbogen in treating acute non-arteritic central retinal artery occlusion. *Ophthalmology* 1995; **102**: 2029-34.
- Rumelt S, Dorenboim Y, Rehany U. Aggressive systematic treatment of central retinal artery occlusion. *Am J Ophthalmol* 1999; **128**: 733-8.
- Cornblath WT, Eggenberger ER. Progressive visual loss from giant cell arteritis despite high-dose intravenous methylprednisolone. *Ophthalmology* 1997; **104**: 854-8.
- Chevalet P, Barrier J, Pottier P *et al*. A randomised, multicenter, controlled trial using intravenous pulses of methylprednisolone in the initial treatment of simple forms of giant cell arteritis: a one year follow-up study of 164 patients. *J Rheumatol* 2000; **27**: 1484-91.
- Staunton H, Stafford F, Leader M, O'Riordan D. Deterioration in giant cell arteritis with corticosteroid therapy. *Arch Neurol* 2000; **57**: 581-4.
- Lui GT, Glaser JS, Schatz NJ, Schatz J, Lawton Smith J. Visual morbidity in giant cell arteritis. Clinical characteristics and prognosis for vision. *Ophthalmology* 1994; **101**: 1779-85.
- Chan CC, Paine M, O'Day J. Steroid management in giant cell arteritis. *Br J Ophthalmol* 2001; **85**: 1061-4.
- Bhatti MT, Tabandeh H. Giant cell arteritis: diagnosis and management. *Curr Opin Ophthalmol* 2001; **12**: 393-9.
- Foroozan R, Danesh-Meyer H, Savino PJ, Gamble G, Mekari-Sabbagh ON, Sergott RC. Thrombocytosis in patients with biopsy-proven giant cell arteritis. *Ophthalmology* 2002; **109**: 1267-71.
- Liozon E, Herrmann F, Ly K *et al*. Risk factors for visual loss in giant cell (temporal) arteritis. A prospective study of 174 patients. *Am J Med* 2001; **111**: 211-17.
- Mangat HS. Retinal artery occlusion. *Surv Ophthalmol* 1995; **40**: 145-54.

15. Buono LM, Foroozan R, de Virgiliis M, Savino PJ. Heparin therapy in giant cell arteritis. *Br J Ophthalmol* 2004; **88**: 298–301.
16. Van Aken H, Bode C, Darius H *et al*. Anticoagulation: the present, the future. *Clin Appl Thromb Hemost* 2001; **7**: 195–204.
17. Rondeau M, Weber J, Storck D. A case of giant cell arteritis associated with suspected cerebral angiitis. *Eur J Intern Med* 1993; **2**: 186–7.
18. Rang HP, Dale MM, Ritter GM, Gardner P. *Pharmacology*, 4th edn. London: Churchill Livingstone, 2001; 318–20.

## Scleral buckle intrusion

### ABSTRACT

A case is reported of a 72-year-old man with an intruded scleral buckle presenting 30 years after retinal detachment repair. The lesion was noted incidentally and occupied the superotemporal retinal periphery. The diagnosis was confirmed by B-scan ultrasonography and external examination of the globe. The patient was treated conservatively. No clinical change was evident after 12 months follow up.

**Key words:** retinal cyst, retinal detachment, retinoschisis, scleral buckle intrusion.

### INTRODUCTION

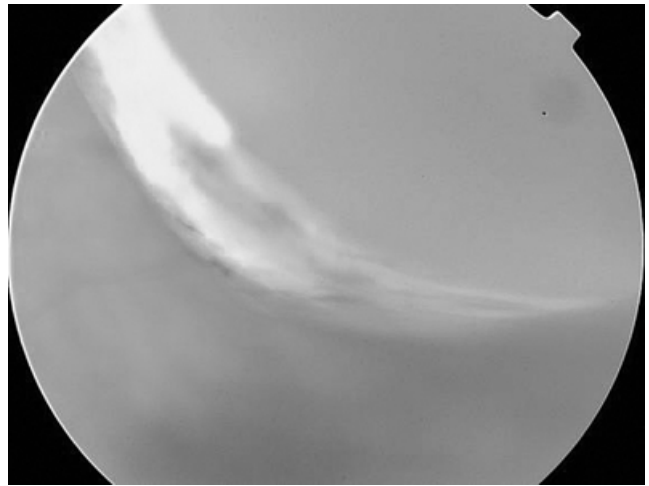
Scleral buckle intrusion is a very uncommon postoperative complication of the scleral buckling technique.<sup>1</sup> Correct diagnosis of an intruded scleral buckle has important therapeutic implications, as ocular hypotony and endophthalmitis are potential sequelae.<sup>2</sup>

### CASE REPORT

A 72-year-old man who presented with an acute right retina detachment was noted incidentally to have an unusual peripheral retinal lesion in his left eye. He gave a history of left retinal detachment 30 years previously that was repaired with scleral buckling. The vision in the left eye had remained unaltered since then.

On examination visual acuity was 6/60 in his right eye and 6/24 in his left eye. Intraocular pressures were 15 mmHg in the right eye and 15 mmHg in the left eye. Bilateral anterior segment examination was unremarkable. On fundus examination of the right eye there was a rhegmatogenous retinal detachment involving the macula. In the left eye there was an unusual lesion observed in the superotemporal retinal periphery, which had been previously diagnosed as a retinal cyst (Fig. 1). The retina was otherwise attached. B-scan ultrasound showed the lesion to be echo-lucent and having a 'cystic appearance' (Fig. 2).

On external examination of the left globe there was a depression in the contour of the sclera in the area of the presumed cyst. A diagnosis of intruded scleral sponge was made. The operative notes were obtained showing that the scleral buckle material was Miragal placed under a scleral trapdoor flap. The intruded scleral sponge was managed conservatively and has remained unchanged over 2 years.



**Figure 1.** Fundus photograph of right eye showing an intruded scleral buckle in the superotemporal retinal periphery.



**Figure 2.** B-scan ultrasound showing the echo-lucent appearance of the intruded scleral buckle.

### DISCUSSION

Scleral buckle intrusion and erosion is a complication of retinal detachment repair. In one reported series it occurred in approximately 4 of 4400 cases.<sup>1</sup> An association with myopia exists because of the related scleral thinning and altered scleral strength. The surgical technique used in this case of 'scleral trapdoor' might be associated with an increased risk of intrusion due to the thinned scleral bed. The type of buckle used is also related to the risk of intrusion; however, this complication has been noted in all explant materials.<sup>3</sup>

The appearance of the lesion on B-scan ultrasonography was very similar to that of a retinal cyst or retinoschisis. In this case the

diagnosis was made by thorough history taking, which revealed previous retinal detachment surgery, and by examining the contour of the external globe. Differential diagnoses include retinal cyst, retinoschisis, retinal detachment and choroidal effusion.

Scleral buckle erosion and intrusion may cause visual symptoms and may result in retinal detachment, vitreous haemorrhage, retinal pigment epithelium atrophy and endophthalmitis.

An intruding or eroding scleral buckle may be left undisturbed unless there is recurrent detachment or haemorrhage, or a significant threat to the integrity of the ocular structures.<sup>3</sup> Treatment of a retina re-detachment is likely to require vitrectomy surgery. Repair of the scleral defect may require scleral patch grafting. In the absence of these complications regular monitoring of the patient is necessary to ensure that the situation remains stable.

Nikhil Kumar BMed MPH,<sup>1</sup>  
Alina Zeldovich MB BS BSc(Med)<sup>1</sup> and  
Andrew Chang PhD FRANZCO<sup>1,2</sup>

<sup>1</sup>Sydney Retina Clinic and <sup>2</sup>Vitreoretinal Unit, Sydney Eye Hospital, Sydney, New South Wales, Australia

## REFERENCES

1. Nguyen QD, Lashkari K, Hirose T, Pruett RC, McMeel JW, Schepens CL. Erosion and intrusion of silicone rubber scleral buckle. Presentation and management. *Retina* 2001; 21: 214–20.
2. Deramo VA, Hauptert CL, Fekrat S, Postel EA. Hypotony caused by scleral buckle erosion in Marfan syndrome. *Am J Ophthalmol* 2001; 132: 429–31.
3. Roldan-Pallares M, del Castillo Sanz JL, Awad-El Susi S, Refojo MF. Long-term complications of silicone and hydrogel explants in retinal reattachment surgery. *Arch Ophthalmol* 1999; 117: 97–201.

## Anterior and posterior vitreous cysts

### ABSTRACT

Vitreous cysts are rare structural entities of uncertain aetiology. Although most do not produce troublesome visual symptoms, they can cause diagnostic difficulties in clinical practice. Two different types are described and the possible aetiologies of congenital and acquired vitreous cysts are discussed.

**Key words:** congenital, cysts, pigmented, vitreous.

### INTRODUCTION

Vitreous cysts are rare structural entities of uncertain aetiology. They can occur in the anterior or posterior vitreous, and be either pigmented or non-pigmented. Two different types of vitreous cysts are described in the following case reports.

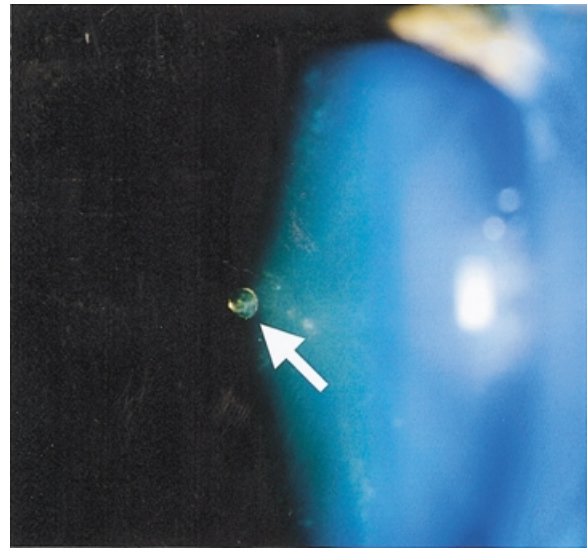


Figure 1. Anterior non-pigmented vitreous cyst attached to posterior surface of lens capsule.

## CASE REPORTS

### Case 1

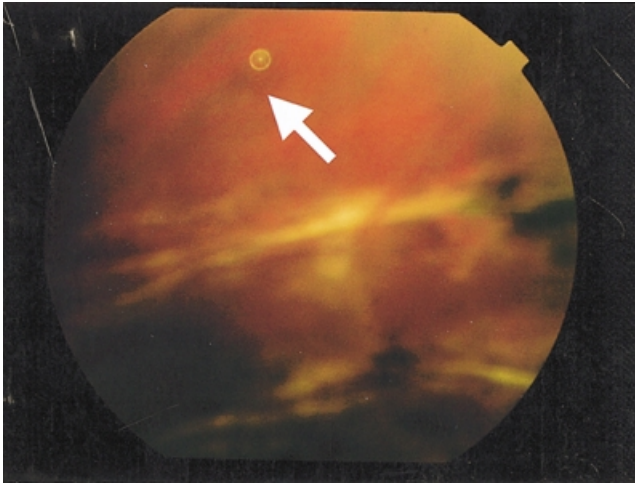
A 30-year-old Caucasian man presented with a history of floaters in his right eye for the preceding few months. He was highly myopic with congenital nystagmus. His corrected visual acuity was 6/12 in the right eye and counting fingers in the left eye, which had undergone a retinal detachment repair using a scleral buckle 14 years previously.

In the left eye, a 2-mm non-pigmented spherical 'cyst' was observed posterior to the lens. It was mobile within the anterior vitreous but attached to the centre of the lens by a thin strand (Fig. 1). Bilateral posterior vitreous detachments were present, and also evidence of myopic macular degeneration and previous retinal surgery. His right eye symptoms were thought to be due to the vitreous detachment. After 1 year the cyst had remained stable and he was discharged with instructions to report any new or increasing symptoms.

### Case 2

A 47-year-old Caucasian man began to complain of a floater in his amblyopic left eye. He had been hit with a cricket bat on this side at the age of 9 years requiring some sutures to the side of his face. At the age of 17 it became apparent that the vision on this side had been poor for some time due to a chronic retinal detachment. Surgery was not performed on this eye at any time and the corrected visual acuity was 6/36 on the left and 6/6 on the right with a small myopic prescription.

A clear round 'cyst' floating freely in the superior posterior vitreous was noted in the left eye (Fig. 2). After 1 year the cyst remained stable and he was discharged with instructions to report any new or increasing symptoms.



**Figure 2.** Traumatic posterior non-pigmented vitreous cyst free-floating in superior vitreous.

## DISCUSSION

Both cases caused initial diagnostic difficulties; however, a literature search suggested these were vitreous cysts. These structures were first described in 1899<sup>1</sup> and by 1998 only 50 cases had been reported in the literature.<sup>1,2</sup>

Cysts of the anterior and posterior vitreous, either pigmented or non-pigmented, have been described. They may be classified into primary congenital vitreous cysts, or secondary cysts resulting from trauma, intraocular surgery, uveitis or retinal conditions.<sup>1</sup> Free-floating cysts lying between the lens and the anterior hyaloid are extremely rare.<sup>3</sup>

Cysts are rarely removed, thus their aetiology is unclear. Primary congenital vitreous cysts are thought to be remnants of the hyaloid artery system and may be attached to the disk or posterior lens surface by a stalk. One cyst that was removed during vitrectomy revealed heavily pigmented cells, with tight junctions and apical microvilli.<sup>4</sup> It was felt to have originated from the hyaloid system because of its location within Cloquet's canal, the presence of a Mittendorf's dot and the unusual presence of immature melanosomes within the pigment epithelial-type cells. Other histopathological studies have indicated a variable origin: one cyst suggested retinal pigment epithelial origin,<sup>5</sup> and yet another was composed of retinal tissue and thought to have resulted from several procedures for retinal detachment surgery.<sup>6</sup>

Cyst size varies between 0.15 and 12 mm, as a unilateral or bilateral feature in both emmetropic and myopic eyes.<sup>2,7,8</sup> They

may be highly mobile in liquefied vitreous (e.g. Case 2, most likely a secondary traumatic posterior non-pigmented vitreous cyst), or be attached to the posterior lens surface and partially mobile (e.g. Case 1, possibly a congenital anterior non-pigmented vitreous cyst or the result of retinal surgery).

The cysts may be asymptomatic, or cause transient infrequent visual obscurations, or new symptoms of floaters if their position is altered by a posterior vitreous detachment in an otherwise asymptomatic individual.

Previous reports suggest that most cysts do not enlarge, thus only symptomatic cysts should be treated. Options include laser photocystotomy (Argon<sup>9</sup> or Nd:YAG<sup>10</sup>) or vitrectomy. Patients may be discharged after a period of observation with instructions to report any subsequent increase in symptoms.

An awareness of the possibility of encountering vitreous cysts as a cause of floaters can allow the clinician to reassure patients about the nature and long-term effects of these unusual structures.

**Niro Narendran BSc MRCOphth, Eddie Doyle MRCOphth  
and Alistair Laidlaw MD FRCOphth**  
*Department of Ophthalmology, St Thomas' Hospital, London, UK*

## REFERENCES

1. Lusky M, Weinberger D, Kremer I. Vitreous cyst combined with bilateral juvenile retinoschisis. *J Pediatr Ophthalmol Strabismus* 1998; **25**: 75–6.
2. Jones WL. Free-floating vitreous cyst. *Optom Vis Sci* 1998; **75**: 171–3.
3. Sugar HS, Bogorad D, Richards BW. Free-floating cysts of the retrolental space. *Arch Ophthalmol* 1990; **108**: 483–4.
4. Nork TM, Millecchia LL. Treatment and histopathology of a congenital vitreous cyst. *Ophthalmology* 1998; **105**: 825–30.
5. Orellana J, O'Malley RE, McPherson AR, Font RL. Pigmented free-floating cysts in two young adults. *Ophthalmology* 1985; **92**: 297–302.
6. Asiyo-Vogel MN, el-Hifnawi el-S, Laqua H. Ultrastructural features of a solitary vitreous cyst. *Retina* 1996; **16**: 250–54.
7. Santos L, Gonzalez F, Capeans C, Suarez A, Sanchez-Salorio M. Bilateral vitreous cysts in an 80-year-old man. *Br J Ophthalmol* 1995; **79**: 865–6.
8. Steinmetz RL, Straatsma BR, Rubin ML. Posterior vitreous cyst. *Am J Ophthalmol* 1990; **109**: 295–7.
9. Awan KJ. Biomicroscopy and argon laser photocystotomy of free-floating vitreous cysts. *Ophthalmology* 1985; **92**: 1710–11.
10. Ruby AJ, Jampol LM. Nd:YAG Treatment of a posterior vitreous cyst. *Am J Ophthalmol* 1990; **110**: 428–9.