lead to a greater urinary loss of calcium. <sup>12</sup> They also inhibit osteoblast maturation and synthetic capability, reducing the amount of bone formed. <sup>13</sup> The routine use of osteoporosis prophylaxis by calcium supplementation <sup>5,7,9</sup> and HRT for postmenopausal women <sup>8</sup> is now accepted as good practice. <sup>3</sup> This study relates to high-risk patients under the care of an ophthalmologist while on long-term oral steroids for various ophthalmological conditions.

We found that none of those patients managed solely in the eye clinic received any direct instructions on calcium supplements or other forms of osteoporosis prevention. It is suggested that the ophthalmologist is unaware of this potential side effect. We have not investigated whether the GP or community pharmacist may provide this information, although we think it unlikely.

We are aware of a number of prevention studies in the area of steroid-induced osteoporosis, although their role in prevention is not yet known. Osteoporosis has become a more recognised clinical entity with the advent of bone density scans (DXA) and may in the future become important medicolegally.

In our department it is now policy to recommend all patients on corticosteroids to supplement their diet with calcium and vitamin D. This is achieved by a verbal discussion with patients when they are seen and a leaflet given when patients collect their prescriptions from the pharmacy. We also confirm that patients are on HRT if indicated and where appropriate we collaborate in care with a physician.

P. R. Hodgkins<sup>1</sup> R. G. Hull<sup>2</sup> A. Vakalis<sup>1</sup> A. Cole<sup>1</sup> C. Hallet<sup>1</sup> A. R. Evans<sup>1</sup> M. N. Jeffrey<sup>1</sup>

Departments of <sup>1</sup>Ophthalmology and <sup>2</sup>Rheumatology Queen Alexandra Hospital Portsmouth UK

Correspondence to: Mr P. Hodgkins Department of Ophthalmology Queen Alexandra Hospital Cosham Portsmouth PO6 3LY UK

#### References

1. Consensus development conference: prophylaxis and treatment of osteoporosis. Osteoporosis Int 1991;1: 114–7.

- 2. Sambrook PN, Jones G. Corticosteroid osteoporosis. Br J Rheumatol 1995;34:8–12.
- 3. Barlow DH (Chairman). Advisory group on osteoporosis: report. London: Department of Health, November 1994.
- 4. Sambrook PN, Birmingham J, Kelly P, *et al.* Prevention of steroid osteoporosis: a comparison of calcium, calcitriol, and calcitonin. N Engl J Med 1993;328: 1747–52.
- Reid IR, Grey AB. Corticosteroid osteoporosis. In: Reid DM, editor. Baillières clinical rheumatology: osteoporosis, vol 7. London: Bailliere Tindall, 1993: 573–87.
- 6. Reid IR, King RA, Alexander CJ, Ibbertson HK. Prevention of corticosteroid induced osteoporosis with biphosphonate. Lancet 1988;1:143–6.
- 7. Reid IR, Grey AB. Calcium supplements in the prevention of steroid induced osteoporosis. Am J Clin Nutr 1986;44:287–90.
- 8. Lukert BP, Johnston BE, Robinson RG. Estrogen and progesterone replacement therapy reduces glucocorticoid induced bone loss. J Bone Miner Res 1992;7: 1063–9.
- 9. Peat ID, Healy S, Reid DM, Ralston SH. Steroid induced osteoporosis: an opportunity for prevention. Ann Rheum Dis 1995;54:66–8.
- Klein RG, Arnaud SB, Gallagher JC, DeLuca HF, Riggs B. Intestinal absorption in exogenous hypercortisolism: role of 25-hydroxyvitamin D and corticosteroid dose. J Clin Invest 1977;60:253–9.
- Lukert BP, Raisz LG. Glucocorticoid induced osteoporosis: pathogenesis and management. Ann Intern Med 1990;112:352–64.
- 12. Reid IR, Ibbertson HK. Evidence of decreased tubular reabsorption of calcium in glucocorticoid treated asthmatics. Hormone Res 1987;27:200–4.
- Dempster DW. Bone histomorphometry in glucocorticoid induced osteoporosis. J Bone Miner Res 1989;7:137–41.

Sir,

# Transposition of Homonymous Hemianopia after Craniopharyngioma Surgery

Craniopharyngiomas are a cause of significant visual morbidity in both children and adults. Visual deficit results either from progressive tumour expansion and compression of optic nerves, chiasm and tracts or from intra-operative injury to distended optic pathways and the surrounding vasculature. Rapid and full recovery of dense visual field defects after surgical decompression is well documented in association with craniopharyngiomas<sup>1-3</sup> and pleomorphism of visual fields is a characteristic feature during both disease progression and treatment of individual tumours.4 We present an unusual case, whereby resolution of a right homonymous hemianopia and the development of a left homonymous hemianopia occurred simultaneously, as a result of one operation to debulk a giant craniopharyngioma.

# Case Report

A 13-year-old boy presented with a 6 week history of headaches, listlessness, anorexia and weight loss. Just

prior to diagnosis he became increasingly confused and disorientated and had visual hallucinations. Referral was made initially to a psychiatrist, who noted a right hemiparesis; consequently, computed tomography and magnetic resonance imaging of the brain were performed. These revealed an extensive craniopharyngioma occupying the suprasellar cistern and encroaching subfrontally; to both middle fossae but predominantly the left; and posteriorly into the interpeduncular cistern (Fig. 1a). The optic chiasm lay stretched and displaced between two large tumours cysts.

Clinical examination prior to surgery revealed a right hemiplegia with a right homonymous hemianopia confirmed on Goldmann perimetry (Fig. 2a). Visual acuity was 6/6 in each eye and colour vision was normal. There was no relative afferent pupillary defect and funduscopy showed mild temporal pallor of the right optic disc.

At surgery, subtotal tumour resection was performed via a right frontal craniotomy. Although access to the left side of the tumour was limited, decompression of the left subfrontal and middle fossa cysts were achieved (Fig. 1b). To effect a satisfactory tumour clearance on the right, this necessitated

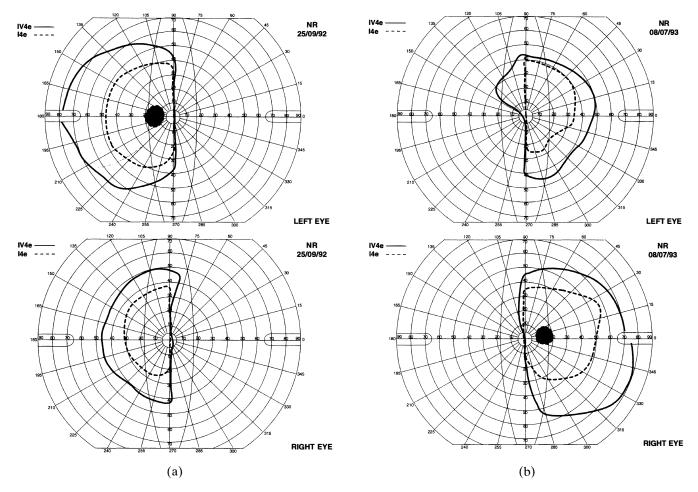
extensive dissection around the right internal carotid artery and the tumour capsule being stripped away from the right anterior temporal lobe. A cerebral vasodilator (nimodipine) was given prophylactically in the immediate post-operative period. On the fifth post-operative day the patient suffered left-sided focal seizures and a transient monoparesis of the left arm. The right-sided hemiplegia had resolved. On repeat ophthalmological testing 12 days post-operatively, visual acuity was 6/9 in each eye and Goldmann visual field assessment (Fig. 2b), performed by the same operator as pre-operatively, now demonstrated recovery of the right hemianopia and the presence of a dense left homonymous hemianopia.

The patient underwent a second craniotomy using a left-sided approach with further tumour clearance, followed by cranial irradiation. Subsequent visual field assessments have shown persistence of the complete left homonymous hemianopia, confirmed by pattern visual evoked responses. At assessment 10 months from initial surgery, visual acuity was normal (6/6 in each eye), colour vision was mildly impaired in the left eye (15 of 21 Ishihara plates seen) and both optic discs were uniformly atrophic. Neurological examination was otherwise normal. Magnetic resonance imaging revealed residual tumour blend-





**Fig. 1.** (a) Pre-operative axial T2-weighted magnetic resonance image demonstrating a multi-lobulated, predominantly cystic craniopharyngioma extending from the suprasellar cistern subfrontally; to both, but predominantly the left, middle fossae; and as far posteriorly as the interpeduncular cistern. (b) Post-operative axial T1-weighted magnetic resonance image demonstrating much reduced tumour bulk and, in particular, slackening of the left middle fossa cyst with associated dilatation of the left temporal horn.



**Fig. 2.** Visual field assessment performed by Goldmann perimetry pre-operatively (a) and at follow-up 10 months after initial surgery (b), demonstrating transposition of a right homonymous hemianopia (a) to a left homonymous field defect (b). The isopters tested are shown.

ing with the right optic nerve, right side of the optic chiasm and floor of the third ventricle. There was no evidence of cerebral ischaemic damage although magnetic resonance angiography demonstrated a smaller right middle cerebral artery compared with the left side and no filling of the first part of the right anterior cerebral artery.

## Discussion

Two mechanisms must have occurred in our patient at the time of surgery to result in recovery of the right homonymous hemianopia and development of a contralateral defect. Although tumour was impinging on anterior visual pathways at presentation, there was little evidence of optic nerve and chiasmal dysfunction. However, the right hemiplegia and magnetic resonance images showing extensive left-sided retrochiasmal involvement suggested compression by tumour of the left optic tract. Complete recovery of field loss and even of blindness by relief of compression on optic pathways can be extremely rapid<sup>2</sup> and has been attributed to reversibility of conduction block which is due mainly to an ischaemic

interruption of local energy supply.<sup>3</sup> In our case, recovery of the right homonymous hemianopia was seen with relief of cyst distension and focal neurological signs resolved.

Intra-operative manipulation of distended, compressed and poorly vascularised optic pathways may incur additional visual deficit. Damage to the internal carotid arteries and branches leading to haemorrhage or spasm may also occur at surgery, particularly if craniopharyngioma is densely adherent to these structures and removal is attempted.<sup>5</sup> In our case, the development of a dense left homonymous hemianopia after surgery suggests a vascular aetiology, and irritative vasospasm of the anterior choroidal artery causing isolated infarction of the right optic tract may have occurred during dissection of the tumour capsule from the region of the right internal carotid artery and anterior temporal lobe. Although no direct vessel damage was recorded at surgery, the patient was felt to be at risk of arterial vasospasm as a cerebral vasodilator was given in the immediate post-operative period. Only partial interruption of this vessel or its branches is likely to have occurred as magnetic resonance imaging showed no signal changes to suggest infarction and there was no accompanying focal neurological deficit.<sup>6</sup> However, magnetic resonance angiography revealed distortion of the vasculature on the right side following surgery.

Whilst diagnostic features of an optic tract lesion can include a contralateral relative afferent pupillary defect and 'bow-tie' optic atrophy, 7.8 these were not seen in our patient, but involvement by tumour of both optic nerves and chiasm are likely to have complicated these signs. It is perhaps more surprising that visual acuity and colour vision have been so well preserved.

We are grateful to the Child Growth Foundation for financial support for C. J. DeV.

C.J. DeVile, MA, MRCP T. Lavy, FRCS, FRCOphth C. Timms, DBO(T) W. Harkness, FRCS D. Taylor, FRCS, FRCOphth

The Hospital for Children NHS Trust Great Ormond Street London UK

#### References

- 1. Pierre-Kahn A, Sainte-Rose C, Renier D. Surgical approach to children with craniopharyngiomas and severely impaired vision: special considerations. Pediatr Neurosurg 1994;21(Suppl 1):50–6.
- 2. Al-Wahhabi B, Choudhury AR, Al-Moutaery KR, Aabed M, Faqeeh A. Giant craniopharyngioma with blindness reversed by surgery. Childs Nerv Syst 1993;9:292-4.
- 3. Frisén L, Sjöstrand J, Norrsell K, Lindgren S. Cyclic compression of the intracranial optic nerve: patterns of visual failure and recovery. J Neurol Neurosurg Psychiatry 1976;39:1109–13.
- 4. Kennedy HB, Smith RJS. Eye signs in craniopharyngioma. Br J Ophthalmol 1975;59:689–95.
- 5. Hoffman HJ. Surgical management of craniopharyngioma. Pediatr Neurosurg 1994;21 (Suppl 1):44–9.
- 6. Ward TN, Bernat JL, Goldstein AS. Occlusion of the anterior choroidal artery. J Neurol Neurosurg Psychiatry 1984;47:1048–9.
- 7. Savino PJ, Paris M, Schatz NJ, Orr LS, Corbett JJ. Optic tract syndrome: a review of 21 patients. Arch Ophthalmol 1978;96:656–63.
- 8. Newman SA, Miller NR. Optic tract syndrome: neuro-ophthalmologic considerations. Arch Ophthalmol 1983; 101:1241–50.

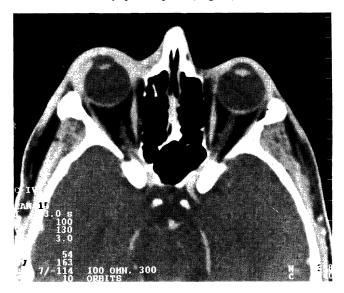
#### Sir.

# Neisseria gonorrhoeae: A Previously Unreported Cause of Pre-septal Cellulitis

Orbital cellulitis typically occurs following dental or sinus infections. Limitation of infection to the preseptal tissues, at least initially, occurs when factors such as infected lid lesions, trauma or conjunctivitis are causal. The frequency of conjunctivitis as the precipitant for pre-septal cellulitis is not known; however, *Moraxella* has been isolated from conjunctival swabs in a patient with pre-septal cellulitis. *Neisseria gonorrhoeae* can cause acute purulent conjunctivitis in neonates (one cause of ophthalmia neonatorum), children and adults. Though rare in adults, rapid progression to ulcerative keratitis and even perforation can occur, especially with penicillinase-producing strains. <sup>2–5</sup> *N. gonorrhoeae* has not been reported as a cause of pre-septal cellulitis.

## Case Report

A 31-year-old homosexual man presented with a 2 day history of acute purulent conjunctivitis in his right eye. He had been started on fusidic acid eye drops by a casualty department on the previous day. There was no history of trauma, foreign body injury or lid lesions. On examination his visual acuity was 6/9 right eye, 6/5 left eye. There was moderate periorbital erythema and swelling, with associated marked conjunctival injection, chemosis and purulent discharge. Right eye movements in all directions were limited by pain. Pupil responses were normal and there was no obvious proptosis. Ocular examination was otherwise normal. A presumptive diagnosis of right orbital cellulitis was made. He was therefore admitted and commenced on intravenous Magnapen (Beecham; flucloxacillin 500 mg/ampicillin 500 mg) q.d.s. and intravenous metronidazole 500 mg t.d.s. A CT scan was performed the day after admission because initial treatment response was poor. This demonstrated the inflammatory changes to be exclusively pre-septal (Fig. 1). There was no



**Fig. 1.** CT scan of head and orbits showing pre-septal soft tissue involvement in the right eye. There is apparent proptosis due to lid oedema and rotation of the film. (Provided by the Department of Medical Illustration, St James's University Hospital.)