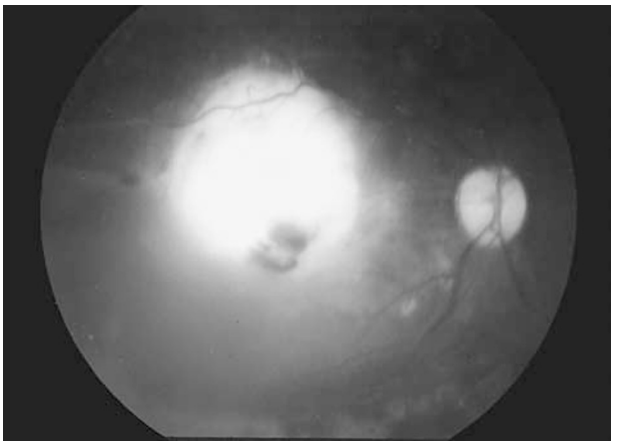

Figure 2 Postoperative fundus photograph showing reattachment of the retina with decreasing submacular lipid exudation.

More recently, Schmidt-Erfuth and Lucke[4] reported eight patients with advanced Coats' disease who were managed with scleral buckle, pars plana vitrectomy, removal of epiretinal membranes, drainage of subretinal fluid through a retinotomy and tamponade with silicone oil. All three retained useful vision (20/500–20/700), but complications from silicone oil did occur in two patients requiring removal of silicone oil in one patient.

Yoshizumi *et al*[5] reported success in three out of four patients with advanced Coats' disease or Coats'–like reaction after vitrectomy surgery. All three successful operations included internal air–fluid exchange and one unsuccessful operation did not include internal air–fluid exchange. Final visual acuity ranged from light perception to 20/300.

Our case represents the first case report of advanced Coats' disease managed with vitrectomy, membrane peeling, internal gas–fluid exchange not employing scleral buckling in which useful vision was obtained. Progressive loss of vision secondary to retinal detachment in advanced Coats' disease is avoidable. We feel scleral buckling plays a very limited role in the management of retinal detachment in Coats' disease which is due to combined exudative and tractional mechanisms. The removal of vitreous and preretinal membranes is an integral step to successful surgery, and vitreous membranes may recur.

Aggressive vitreo-retinal surgery with vitrectomy, drainage of subretinal fluid, air–fluid exchange, and pneumatic retinopexy may offer the best hope for preventing progressive loss of vision.

References

1 Haik BG. Advanced Coats' disease. *Tr AM Ophth Soc* 1991; **89**: 371–476.
2 Silodor SW, Augsburger JJ, Shields JA, Tasman W. Natural history and management of advanced Coats' disease. *Ophthalmic Surg* 1988; **19**: 89–94.
3 Wessing A. Morbus coats. In: Lund OE, Waubke TN (eds). *Gefaberkrankungen des Auges*. Enke: Stuttgart, 1983, pp 126–134.
4 Schmidt-Erfuth U, Lucke K. Vitreoretinal surgery in adanced Coats' disease. *German J Ophthalmol* 1995; **4**: 32–36.
5 Yoshizumi MO, Kreiger AE, Lewis H. Vitrectomy techniques in late-stage Coats'-like exudative retinal detachment. *Documenta Ophthalmologica* 1995; **90**: 387–394.

G Kranias[1,2] and TP Krebs[2]

[1]Dept of Opthalmology
The Christ Hospital
Cincinnati, OH, USA

[2]Opthalmic Imaging and Clinical Services
Vitreo-Retinal Surgeons
2230 Auburn Avenue
Cincinnati, OH, USA

Correspondence: G Kranias, MD
Tel: 513 381 1900
Fax: 513 287 6403
E-mail: gkranias@yahoo.com

Sir,

Optic neuritis: a rare manifestation of nasopharyngeal carcinoma

Eye (2002) **16,** 501–503. doi:10.1038/sj.eye.6700003

Nasopharyngeal carcinoma (NPC) typically presents to ophthalmologists with one or more cranial nerve palsies. The fifth and sixth nerves are the most commonly affected.[1] Although ocular symptoms with cranial nerve involvement are not uncommon in patients with NPC, they often become evident several months or years after the malignancy is diagnosed. We present a case of optic neuritis as the first clinical manifestation of NPC.

Case report

A 55-year-old Taiwanese woman presented with pain and decreased vision in the right eye of about one week duration. There were no other systemic symptoms, in particular symptoms suggestive of NPC. There was no significant past medical or ocular history. General physical examination was normal. Best-corrected visual acuity was 6/10 in the right eye and

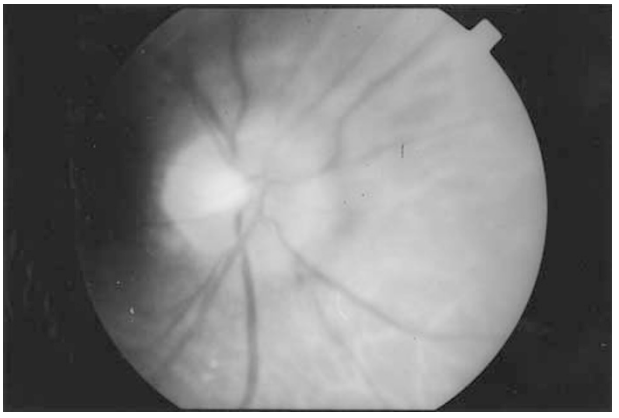

Figure 1 Colour fundus showed a hyperemic disc swelling, especially in the nasal side.

6/6 in the left eye with a normal anterior segment. Intraocular pressures were within normal limits. Examination of the left fundus was unremarkable. Posterior segment examination on the right showed mild disc swelling. There was a right relative afferent pupillary defect. The ocular motility was full. A computed tomography (CT) scan was arranged, but the patient refused for personal reasons.

After 3 weeks the visual acuity in the right eye had deteriorated to 6/60 and colour vision had also decreased. The fundus examination of the right eye revealed moderate disc swelling (Figure 1). The Goldmann visual field demonstrated a constriction of the right visual field. A diagnosis of optic neuritis was made and the patient was given 1 g of intravenous methylprednisolone for 3 days and then commenced on oral prednisolone 60 mg daily on a reducing regime. The visual acuity in the right eye improved to 6/7.5 after 3 weeks. The patient was reviewed and was finally persuaded to undergo the CT examination, which demonstrated a space-occupying lesion in the sphenoid sinus (Figure 2) and erosion of the skull base.

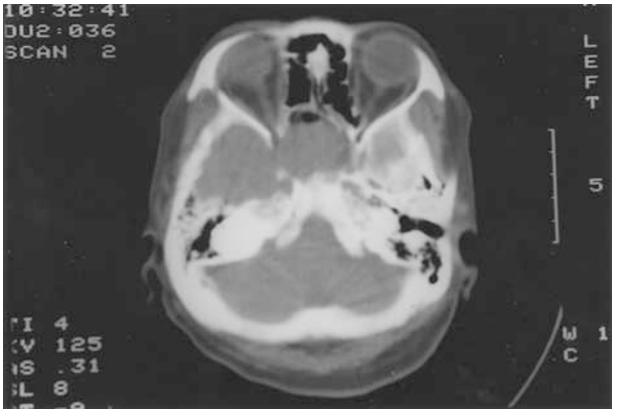

Figure 2 Brain CT scan disclosed a space-occupying lesion situated in the sphenoid sinus.

There was no evidence of tumor infiltration to the orbital cavity. Nasopharyngoscopy showed a tumor mass over the roof of the nasopharynx on the right with skull base invasion. Tissue biopsy revealed a 'non-keratinizing squamous cell carcinoma'. The patient was referred for further management of the NPC with radiotherapy and chemotherapy and followed up in the ophthalmic clinic for 6 months. During this period the patient regained 6/6 vision and normal colour vision in the right eye. One year after completing the radiotherapy and chemotherapy the patient developed right sixth and seventh cranial nerve palsies.

Comment

NPC has a striking geographic distribution, with especially high rates in southern China, Hong Kong, and Taiwan. It commonly manifests as a neck mass, nasal obstruction, nasal bleeding, and occasionally hearing loss. Abducens or oculomotor nerve palsy can often be the presenting ocular feature. These are primarily due to tumor invasion of the cavernous sinus and/or skull base. However, early invasion of the optic nerve is very rare.[2,3] Optic nerve involvement often occurs in the late stage of the disease process and is usually associated with direct tumor invasion of the orbit. In the present case, there were no extraocular signs or evidence of direct tumor invasion around the optic nerve.

Two cases of optic neuritis have been reported previously as the initial presentation of NPC.[4,5] Presad and Doraisamy[4] reported five cases of NPC with optic nerve involvement, one of which manifested initially as a retrobulbar optic neuritis due to extension of the tumor along the medial orbital wall to involve the optic nerve. Hoh *et al*[5] described a case of NPC, which presented initially as optic neuritis. No evidene of a tumor mass around the optic nerve or any histological evidence of tumor infiltration was documented. A possible remote effect of NPC was postulated.

In conclusion, the present report highlights that optic neuritis, a common cause of acute visual loss, can be a rare initial manifestation of NPC. Although it is unclear whether this complication is due to a paraneoplastic effect, clinicians should be aware that NPC can present as retrobulbar/optic neuritis.

Acknowledgements

No public and private support. None of the authors have any commercial interest in the material mentioned herein.

References

1 Turgut M, Erturk O, Saygi S, Ozcan OE. Importance of cranial nerve involvement in nasopharyngeal carcinoma. A clinical study comprising 124 cases with special reference to clinical presentation and prognosis. *Neurosurg Rev* 1998; **21**: 243–248.
2 Carlin L, Biller J, Laster DW, Toole JF. Monocular blindness in nasopharyngeal cancer. *Arch Neurol* 1981; **38**: 600.
3 Kao LY, Chuang HC, Liang YS. Visual loss as the initial presentation of nasopharyngeal carcinoma. *J Clin Neuro-ophthalmol* 1993; **13**: 24–26.
4 Prasad U, Doraisamy S. Optic nerve involvement in nasopharyngeal carcinoma. *Eur J Surg Oncol* 1991; **17**: 536–540.
5 Hoh ST, Teh M, Chew SJ. Paraneoplastic optic neuropathy in nasopharyngeal carcinoma—report of a case. *Singapore Med J* 1991; **32**: 170–173.

C-C Tsai[1,2], H-C Ho[1,2], H-C Kau[2,3], S-C Kao[1,2] and W-M Hsu[1,2]

[1]Department of Ophthalmology, Veterans General Hospital-Taipei, Taiwan

[2]Department of Ophthalmology, National Yang-Ming University, Taiwan

[3]Department of Ophthalmology, Veterans Hospital-Taoyuan, Taiwan

Correspondence: C-C Tsai
E-mail: cctsai@vghtpe.gov.tw

Sir,

Surgically induced necrotising scleritis: report of a case presenting 51 years following strabismus surgery

Eye (2002) **16,** 503–504. doi:10.1038/sj.eye.6700033

Necrotising scleritis is a destructive inflammatory disease of the sclera with serious ocular complications including keratitis, peripheral corneal ulceration and perforation with poor visual prognosis. It is usually associated with systemic diseases in particular the collagen vascular disorders and autoimmune conditions. Surgically induced scleral necrosis (SINS) is a rare form of necrotising scleritis occurring at a site of previous ocular surgery.

We report a case of unilateral necrotising scleritis and peripheral ulcerative keratitis occurring 51 years after ocular surgery. Forty years is the previously longest reported interval.[1] Systemic immunosuppression was required to stabilize the progressive necrosis. Healing was promoted with the use of a silicone hydrogel bandage contact lens.

Case report

A 59-year-old Caucasian woman presented with a history of episodic pain in her left eye and a small dark spot developing on the nasal side of the sclera over a period of 12 months. Her past medical history consisted only of two strabismus operations at the age of 7 and 8 on the affected eye. No written records were available. At initial presentation her visual acuity was 6/6 in both eyes. A focal area of scleral thinning with localised injection on the nasal side of the left eye was noted. The right eye was normal.

Following the diagnosis of anterior scleritis the patient was commenced on topical ketorolac and prednisolone acetate 1% which resulted in moderate symptomatic improvement after 2 months. Oral flurbiprofen was added to help with symptoms but on further review 2 months later, the scleral lesion was increasing in size with recurrent and persistent discomfort in the eye. At this stage there was increased thinning of the sclera with the underlying uveal tissue appearing more prominent. The adjacent peripheral cornea showed an area of guttering associated with loss of epithelium (Figure 1). Careful examination of the bulbar conjunctiva over the lateral rectus revealed subconjunctival scarring consistent with previous surgery. There was no evidence of retained suture material.

A revised diagnosis of necrotising anterior scleritis was made at this stage. Systemic immunosuppression was commenced in the form of a pulse of methylprednisolone 500 mg followed by prednisolone 20 mg, reduced to 10 mg maintenance dose within a week. Topical prednisolone phosphate was continued.

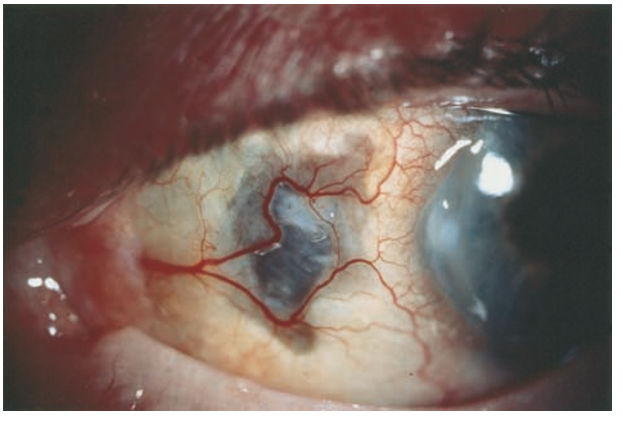

Figure 1 Medial view of the left eye showing a large area of scleral thinning at the site of previous ocular surgery with associated peripheral ulcerative keratitis.