CASE REPORT

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Blindness following medial maxillectomy*

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SUMMARY

Medial maxillectomy via lateral rhinotomy approach is used in the treatment for tumours affecting the lateral nasal wall. The most frequent complications are crusting, epicanthal scarring and epiphora. The authors report a rare case of blindness secondary to indirect optic neuropathy following medial maxillectomy undertaken to treat a malignant melanoma arising in the lateral nasal wall. It is important to keep in mind, the possibility of this rare complication, while patients are being counselled preoperatively for medial maxillectomy.

Key words: medial maxillectomy, complication, optic neuropathy, blindness

INTRODUCTION

Cranio-facial trauma and surgical procedures in the orbital region are important causes of traumatic optic neuropathy (Steinsapir, 1999; Villarreal et al., 2000). Nearly 10% of patients admitted for trauma related injuries suffer facial fractures. Of these fractures 45% involve one or both orbits. Blindness is an uncommon complication of facial trauma with a reported incidence of 2 to 5 percent (Girotto et al., 1998b). The causes of iatrogenic traumatic optic neuropathy include repair of orbital fracture, (0.24%) (Girotto et al., 1998b) and endoscopic sinus surgery (Bolger et al., 1999). The authors report a rare case of optic neuropathy following medial maxillectomy.

CASE REPORT

An 87-year-old woman presented to the ENT department with profuse epistaxis. Despite nasal packing the patient continued to bleed. Examination of the nose under a general anaesthetic revealed a haemorrhagic polyp arising from the superior surface of the left middle turbinate. Excisional biopsy of the polyp was done and the sphenopalatine artery was ligated to stop the bleeding. The histology was reported as amelanotic melanoma. CT scan of the paranasal sinuses revealed a mass in the left anterior ethmoidal cells, extending into the medial wall of the maxilla. The patient underwent a medial maxillectomy and ethmoid clearance via a lateral rhinotomy approach. On the first postoperative day, the patient complained of left-sided diminution of visual acuity to finger counting at a distance of 1 ft. There was mild proptosis. The afferent pupillary reflex was sluggish but the fundus examination was normal. A provisional diagnosis of intra-orbital haemorrhage causing a compartment syndrome was made. Emergency orbital fat decompression was done under general anaesthesia. Incision over the orbital periosteum did not reveal unduly raised intra-orbital pressure. On the second postoperative day the patient complained of loss of vision. Ophthalmological examination revealed that the pupillary reaction to light was absent in the left eye. Fundoscopy showed optic disc oedema with blurring of inferior margin and absence of spontaneous venous pulsations, suggestive of optic neuropathy. A visual field examination revealed complete loss of vision in the left eye. Examination of the right eye was normal.

Further investigation by MR scan showed asymmetry of the optic nerves with the left optic nerve returning higher signals in T2 weighted and STIR images but not in T1 weighted images (Figures 1, 2 and 3). There was absence of any direct injury or bony fragment pressing on the optic nerve. The patient was treated with high dose of dexamethasone. Repeated visual examinations revealed no improvement. At one year follow up, the patient is blind in the left eye, but free of loco-regional recurrence and distant metastases.

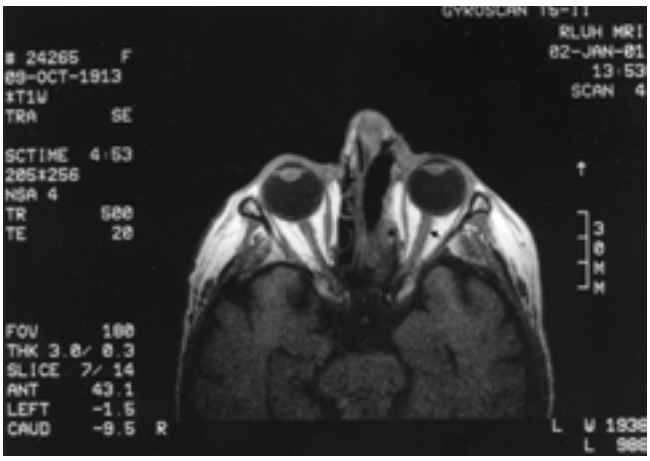

Figure 1. T1 weighted axial image showing asymmetry of the optic nerves. Arrow indicates affected side.

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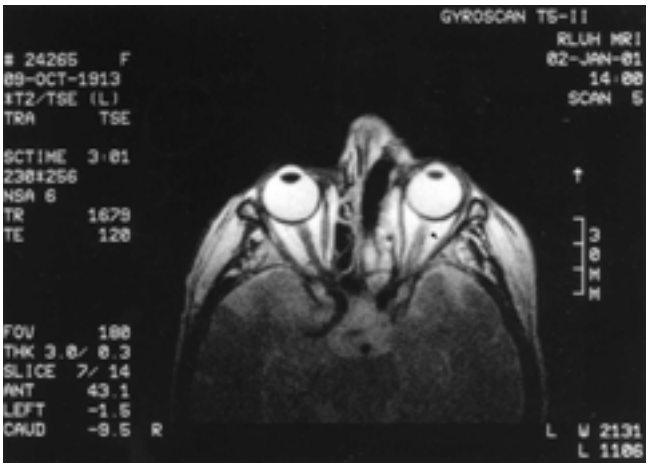

Figure 2. T2 weighted axial image showing high intensity signal return from the left optic nerve.

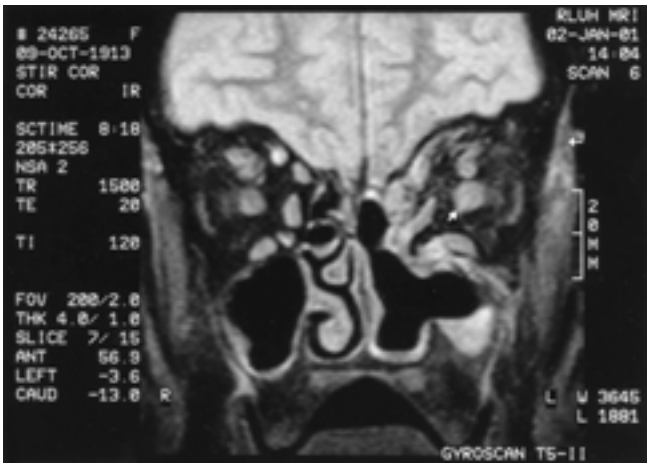

Figure 3. Stir coronal image showing high intensity signal return from the left optic nerve.

DISCUSSION

Proximity of orbit puts the eye at risk in all the operations involving the lateral nasal wall. This is well known to the surgeon using the endoscope to deal with sino-nasal pathologies. Orbital complications after medial maxillectomy and lateral rhinotomy include epiphora, dacrocystitis, ectropion, diplopia and orbital haemorrhage (Bernard, 1989; Thompson et al., 1990; Weisman, 1995). Blindness after medial maxillectomy is an extremely rare complication.

Optic nerve trauma (Steinsapir, 1999) is visual loss caused by deformational forces that affect the optic nerve. When the injury is the result of contact between the optic nerve and an object (such as a bullet or a sinus endoscope), it is classified as *direct* injury. In contrast, *indirect* injury to the optic nerve occurs when energy is transferred through the bones of the skull or by globe rotation into the optic nerve.

During medial maxillectomy, the osteotomies through the medial and inferior wall of the orbit and the pterygomaxillary fissure may result in atypical propagation of the fracture lines into the optic canal due to close anatomical proximity (Rontal et al., 1979). This has been also investigated in an experimental set up on cadavers (Girotto et al., 1998a). The rarity of optic nerve damage is probably attributed to the secure position of the nerve in the optic canal made up of compact bone and the controlled fracturing of the relatively weaker areas of the maxilla. Orbital haemorrhage can give rise to a compartment syndrome, which can cause optic nerve damage by compression. (Steinsapir, 1999). Moreover, holographic interferometry has confirmed that blunt stresses applied to the malar and suprabrow forehead are transmitted to the optic canal (Anderson et al., 1982).

In our patient, the compartment syndrome was excluded by the findings at the orbital fat decompression operation while direct transection injury of the optic nerve was ruled out by the MR scan.

Diagnosis of indirect optic neuropathy was made based on fundoscopic findings. Indirect traumatic optic neuropathy is defined by Walsh and Hoyt as “traumatic loss of vision without external or initial ophthalmoscopic evidence of injury to the eye or its nerve”(Walsh and Hoyt, 1969). It typically occurs with a delay of hours/days/weeks after the initial trauma. The visual loss can be sudden and complete or progressive and partial, but in all cases the fundoscopic examination appears normal at the outset.

Treatment of indirect optic nerve damage is controversial (Cook et al., 1996; Levin et al., 1999). The prognosis in cases where there is total visual loss is poor regardless of the treatment. In the cases of delayed incomplete or progressive visual loss high dose steroids and optic nerve decompression have been proposed as possible treatment options.

The indications for optic nerve decompression include (Villarreal et al., 2000):

1. Delayed progressive partial visual loss non responsive to treatment with megadose of steroids.
2. Initial improvement in vision while receiving steroids followed by deterioration in visual acuity.
3. Subtotal or delayed visual loss in patients in whom CT/MRI shows a retro bulbar haematoma or displaced fracture fragments.

Currently, there is no evidence that surgical or medical treatment is more effective than observation alone. It is possible that selected patients might benefit from a more aggressive treatment and therefore it is reasonable to decide to treat or not to treat on an individual patient basis (Levin et al., 1999).

It is important to emphasize the other options available – such as endoscopic means - to approach lateral nasal wall tumours, and that this approach is oncologically valid and might extend a greater chance of avoiding trauma to the optic nerve.

Ophthalmologists have long recognized orbital haemorrhage as the cause of blindness following simple routine procedures like retrobulbar anaesthesia and blepharoplasty Awareness of, the possibility of indirect trauma as a cause for blindness, by surgeons working around the eye is very essential. Pre-operative counselling by surgeons should include this rare complication, which may happen secondary to procedures done in or around the orbit although they involve relatively minor mechanical forces (Gross et al., 1981).

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