proptosis and ocular movements improved but the vision remained counting fingers. He later underwent a transurethral resection of prostate for his prostatic symptoms. Histological examination showed poorly differentiated adenocarcinoma of the prostate.

Comment

Pituitary metastasis with extension into the orbital apex is a rare condition and, to our knowledge, this has not previously been described as a presenting feature of prostatic cancer.

Metastatic tumours of the pituitary gland are uncommon and are usually observed in widespread metastatic disease or at autopsy. Rarely do such tumours represent the onset symptoms of an underlying malignancy, as in our patient. The most common primary cancers causing pituitary metastases are breast and lung.

Radiologically, it is difficult to differentiate metastatic pituitary tumours from primary tumours.[2] However, the differentiation is important, as the treatment and prognosis are significantly different. Clinically, there are certain signs suggestive of metastatic tumour. Diabetes insipidus is an important sequela of metastatic disease, which is found in up to 20% of patients.[3,4] This is because the posterior pituitary lobe receives direct blood flow from the systemic circulation and is therefore more commonly involved than the anterior pituitary lobe, which is secondarily supplied by the pituitary portal vasculature. An ophthalmoplegia is unusual with pituitary adenomas as they rarely invade the cavernous sinus. The presence of diabetes insipidus and ophthalmoplegia should suggest a malignancy such as metastatic tumour.[5]

Prostatic carcinoma is common in elderly men and typically metastasises to bone causing osteoblastic lesions. The incidence of pituitary metastases has been reported to be between 1% and 6% amongst patients with prostatic carcinoma, but they are rarely detected in life.[6,7]

The optic neuropathies can be caused by metastatic prostate cancer to the optic canal. The mechanism is thought to be optic nerve compression resulting from diffuse bony changes. Imaging is useful in showing bone hypertrophy and deformity.[8] However, this feature was not seen in our patient, suggesting that his optic neuropathy may have resulted from direct tumour compression. Treatment with a combination of steroid and radiotherapy can improve the vision if given early.

Our patient is unusual in many respects: the site of the lesion and the absence of an underlying malignancy at presentation. In retrospect, the rapid progression of the ophthalmoplegia and visual loss should have raised the suspicion of a malignancy. Our case shows that metastatic disease should be included in the differential diagnosis of pituitary lesions.

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Sir,

Orbital apex syndrome as a presenting sign of maxillary sinus carcinoma

Orbital apex syndrome is a rare symptomatological complex characterised by proptosis and paralysis of extraocular muscles, associated with involvement of the first division of the trigeminal nerve and visual loss of various degrees. Only two cases of maxillary sinus neoplasms presenting as orbital apex syndrome have been reported in the literature.[1,2] We report, to the best of our knowledge, the first case of poorly differentiated squamous cell carcinoma of the maxillary sinus presenting unusually as orbital apex syndrome. Immunohistochemistry was used to confirm the epithelial nature of the tumour cells.

Case report

A 66-year-old man was referred to the eye clinic with an 8 week history of swelling, pain and discharge from his right eye. The patient was a chronic smoker and had no previous history of sinusitis or nasal block. The visual acuities were 6/36 NIP in the right eye and 6/6 unaided in the left eye. Examination of the right eye showed periorbital swelling, non-axial proptosis, ptosis and chemosis (Fig. 1, above). There was an afferent pupillary defect with impairment of colour vision. Corneal hypothaesia with decreased sensation along the first division of the fifth cranial nerve distribution was noted. The intraocular pressures were normal. Fundus examination showed disc oedema with normal retinal

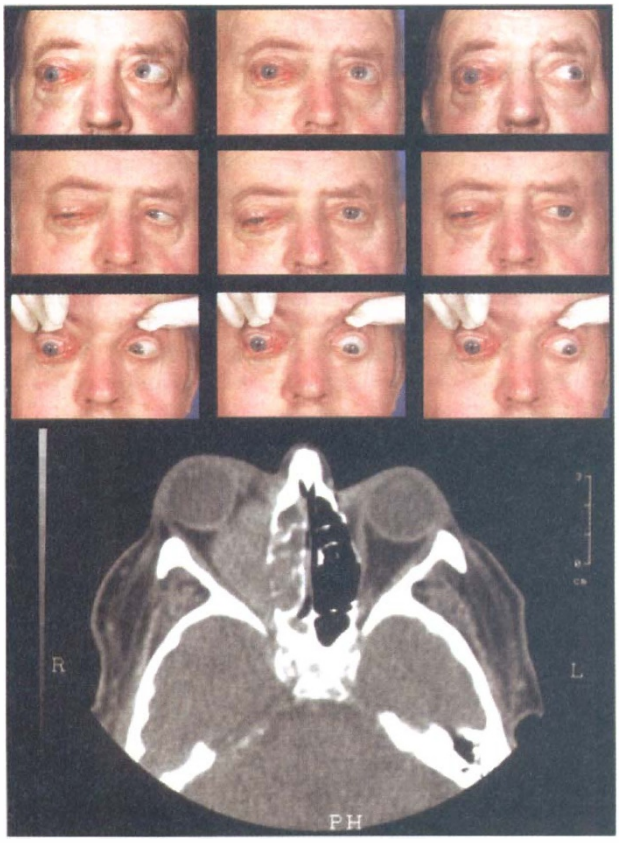

Fig. 1. *Above: Clinical photograph showing proptosis and restriction of extraocular movements of the right eye. Below: Axial CT scan showing a mass lesion extending to the apex of the right orbit.*

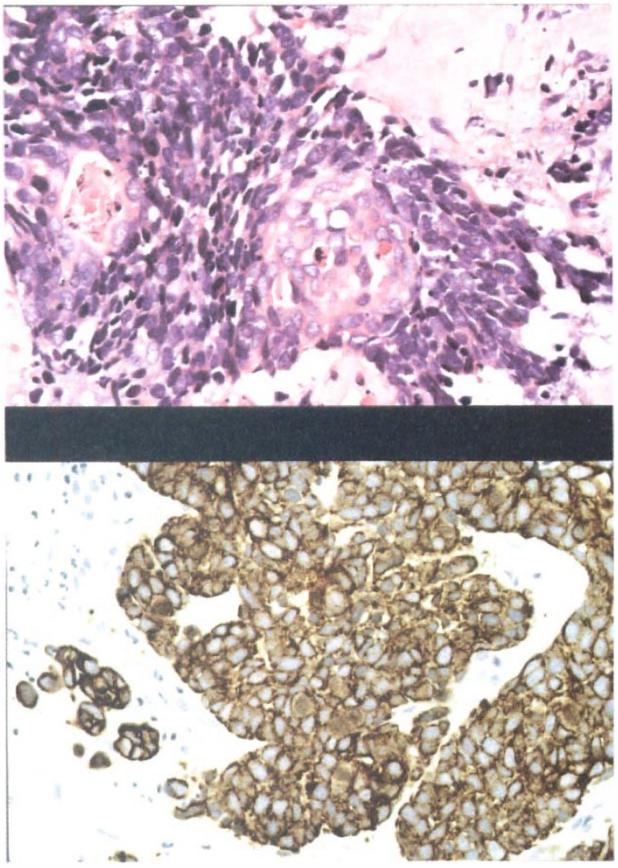

Fig. 2. *Above: Light micrograph showing sheets of pleomorphic cells around blood vessels with areas of necrosis (haemotoxylin and eosin, original magnification ×200). Below: Immunohistochemical staining with CAM 5.2 showing positive staining (brown colour) for cytokeratin (original magnification ×200).*

vasculature. The extraocular movements were restricted in all positions of gaze (Fig. 1, above). The left eye was normal on examination.

Computed tomography showed a contrast-enhancing mass lesion arising from the right maxillary sinus, eroding the floor of the right orbit, involving the right retrobulbar space and extending to the apex of the orbit (Fig. 1, below). This lesion had also involved the right nasal cavity, ethmoidal sinuses, inferotemporal and pterygopalatine fossa. The patient underwent a biopsy of the maxillary sinus tumour through a Caldwell–Luc approach.

Histopathological examination with haematoxylin and eosin showed a neoplasm composed of confluent sheets of large pleomorphic cells with scanty cytoplasm. Numerous mitotic figures were noted and foci of necrosis were present. Occasional nests of cells had a small amount of eosinophilic cytoplasm (Fig. 2, above). Immunohistochemical markers showed positive staining for the epithelial marker CAM 5.2 (Fig. 2, below), confirming the diagnosis of poorly differentiated carcinoma of probable squamous type. As the tumour was found inoperable, palliative radiotherapy was planned.

Comment

The symptomatological complex of proptosis, painful ophthalmoplegia and involvement of the fifth cranial nerve has been described under various names besides the initial one of Rochon–Duvigneaud syndrome or superior orbital fissure syndrome. These include Tolosa–Hunt syndrome (Tolosa 1954, Hunt 1961), syndrome de la paroi externe du sinus caverneux (Fois, 1992), cavernous sinus syndrome of Jefferson (1938) and parasellar syndrome (Thomas and Yoss, 1970).[3] Orbital apex syndrome is differentiated from the superior orbital fissure syndrome by the clinical involvement of the optic nerve.

The differential diagnosis of orbital apex syndrome includes orbital space-occupying lesions, cavernous sinus lesions and tumours arising from the nasopharynx, sinuses, orbit and parasellar region.[4] Carcinomas of the maxillary sinus are uncommon. They comprise of 0.2–0.5% of all systemic malignancies, 3% of all cases of head and neck carcinoma, and 80% of all cases of paranasal sinus carcinoma.[5] Johnson *et al.*[6] reviewed 79 patients with sinus and paranasal tumours and found that 47 patients (59%) demonstrated orbital involvement. The maxillary sinus was the most common site of origin of the paranasal tumours and the sphenoid sinus the least common. Squamous cell carcinoma was the most common paranasal tumour to invade the orbit, followed by inverting papilloma, osteoma and adenocarcinoma. In their series the most common ophthalmic presenting sign was proptosis. None of their patients presented with orbital apex syndrome. Maxillary sinus neoplasms presenting as orbital apex syndrome have been uncommonly reported in the literature.[1,2] Gore *et al.*[1]

reported a case of mucoepidermoid carcinoma presenting as orbital apex syndrome in a 33-year-old man with a 2 month history of maxillary discomfort, fullness and purulent discharge. McDonald *et al.*[2] reported a case of adenoid cystic carcinoma which presented as orbital apex syndrome in a patient with preceding history of facial numbness.

This case is unique both in that the patient had no ENT symptoms prior to his presentation to the ophthalmologist and as regards the short history of his symptoms, during which time the tumour had grown to inoperable dimensions. Orbital apex syndrome can be a presenting sign of maxillary sinus squamous cell carcinoma. Prompt ENT referral is recommended in these cases.

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Sir,

Clear tectonic penetrating graft using glycerine-preserved donor cornea

Corneal blindness is more prevalent in the developing countries than in the Western world. The demand for donor eyes for corneal transplantation greatly surpasses the available supply of viable tissue. In the developed world, newer and better preservative media such as optisol have been introduced.[1] But in the developing countries, donor corneas stored in McCarey Kaufman (M-K) medium and a moist chamber at 4 °C are mostly used for penetrating keratoplasty.[2] Paucity of quality donor tissues often compels the corneal surgeons in the developing world to use glycerine-preserved corneas for lamellar or emergency tectonic penetrating keratoplasty. Clear lamellar grafts have been reported with the use of donor cornea preserved in glycerine for extended periods up to 23 years.[2] To our knowledge there is no reported case of a tectonic penetrating graft having remained clear, using donor cornea preserved in glycerine. The authors report on a clear tectonic penetrating graft using donor cornea preserved in glycerine (50%) at room temperature (30 °C) for 4 weeks.

Case report

A 32-year-old woman presented to our Cornea Service with complaints of pain, redness and watering in the left eye for 4 weeks. The patient had been diagnosed as having a perforated bacterial corneal ulcer by the referring ophthalmologist. She had been prescribed ciprofloxacin 0.3% eye drops 2-hourly and cyclopentolate hydrochloride 1% eye drops 8-hourly. She was not a contact lens wearer. She did not volunteer a history of trauma, use of topical corticosteroids or other predisposing factors for infective keratitis. She had had a similar attack of pain, redness and diminution of vision in the left eye at the age of 3 years. Her symptoms had then subsided with topical antibiotic drops. The patient had then developed an opacity in the inferior half of the left cornea. Poor vision led to a gradual divergence of the left eye.

On presentation, she had best-corrected visual acuity of 6/6 in the right eye and counting fingers at 30 cm in the left eye. The patient had a left divergent squint of 40 prism dioptres. Slit-lamp biomicroscopy of the left eye revealed circumciliary congestion and a large mid-peripheral corneal perforation (4.2 × 4.2 mm) in the inferior half of the cornea. Iris plugged the site of the perforation and a thin layer of epithelium covered the iris surface. There was an area of corneal thinning (approximately 0.75 mm wide) all around the corneal perforation. In addition, there was an opacity in the inferior half of the cornea (Fig. 1). The anterior chamber was shallow. A positive Seidel's test confirmed an

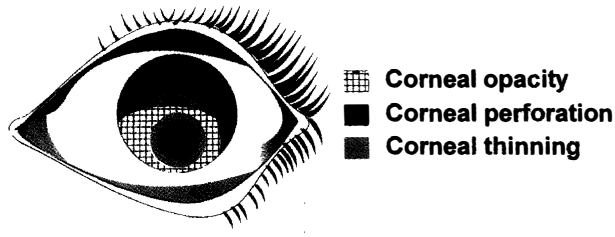

Fig. 1. *Diagram showing corneal perforation surrounded by corneal thinning and opacity.*