ities is difficult to assess, however, as the patient population is very small and the viral keratitis is possibly self-limiting anyway. Our patient did not have any improvement from either topical steroids or anti-virals.

Most of the previously reported cases of EBV keratitis have occurred in conjunction with mononucleosis;[4,5,9,10] however, 3 cases reported by Matoba and Wilhelmus in 1986 and 2 cases reported by Matoba and Jones in 1987 were without systemic symptoms. Corneal findings in the previously reported cases are broad, ranging from superficial/dendritic to stromal lesions of all depths.[1,4,5,8,10] Our patient represents another possible case of chronic EBV keratitis without systemic symptomatology, which displayed only superficial punctate staining throughout the course of the illness.

Matoba and Jones[6] in 1987 suggested that EBV keratitis might be a much more common entity than previously thought. Due to the variety of appearances of its keratitis and its propensity for causing a follicular conjunctivitis, it is easily confused with either adenoviral or herpetic lesions. These cases are usually self-limiting, however, so we do not routinely recommend Heterophile antibody screen or EBV serology. If the case is refractory or otherwise complicated, the above tests should be included in the diagnostic investigation of the keratitis.

Thygeson's superficial punctate keratitis (TSPK) is also without significant conjunctival injection, but causes no pannus and usually responds well to steroids.[11,12] There is also evidence suggesting that TSPK is responsive to trifluridine.[13] Our patient had proved unresponsive to weeks of topical steroid therapy by the time we evaluated her, and she already exhibited inferior pannus bilaterally. Additionally, her keratitis was largely unchanged by a 1 week course of Viroptic which was begun immediately after we saw her.

TSPK was first described in 1950. It is a chronic or recurring, usually bilateral condition which is accompanied by tearing, irritation and photophobia. Corneal lesions tend to exhibit microscopic coarse, granular opacities which often grouped into a larger round or oval conglomerate.[12,13] Whereas the lesions strongly resemble those of adenoviral keratoconjunctivitis, the conjunctivae in TSPK remain uninvolved. The lesions of our patient's EBV keratitis (which may or may not be accompanied by a conjunctival response) closely resembled those of TSPK. It may be that an EBV panel and/or Heterophile antibody test should be included in the investigation of TSPK in order to rule out EBV as a possible cause.

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Sir,

Nasopharyngeal Carcinoma: A Cause of Foster Kennedy Syndrome

In 1911, Foster Kennedy presented 6 cases of unilateral papilloedema with contralateral optic atrophy, often accompanied by loss of smell on the atrophic side, as pathognomonic of a space-occupying basofrontal lesion on the side of the optic atropy.[1] We describe a 33-year-old patient with nasopharyngeal carcinoma with extensive intracranial extension causing ipsilateral optic atrophy and contralateral papilloedema as a result of increased intracranial pressure.

Case Report

A 33-year-old white male patient had noted sudden visual disturbances with his right eye in the form of blurred vision and dull colours. He also complained of headaches, worst in the morning, but he denied any sickness. His wife added that he had appeared confused on a few occasions recently.

There had been a past history of left squint and amblyopia when the patient was a child. Eighteen months previous to his referral to the Eye Department, he was diagnosed as having nasopharyngeal carcinoma on the left side. It was noted then that the tumour mass had extended up towards the base of the skull through the foramen lacerum and was encroaching onto the left cavernous

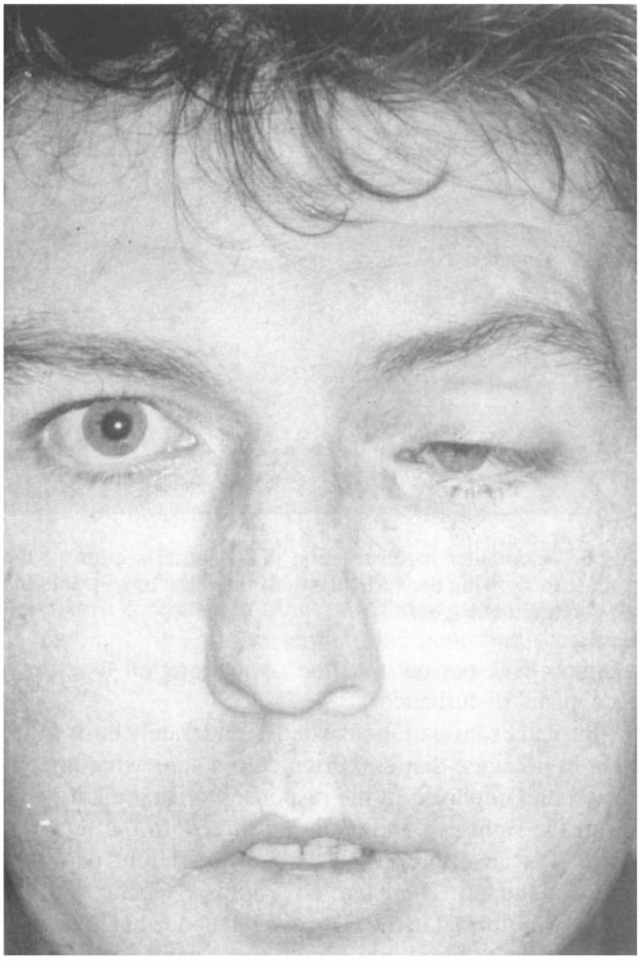

Fig. 1. *The patient. Note the left partial ptosis, divergent squint, and wasting of the temporalis and masseter muscles.*

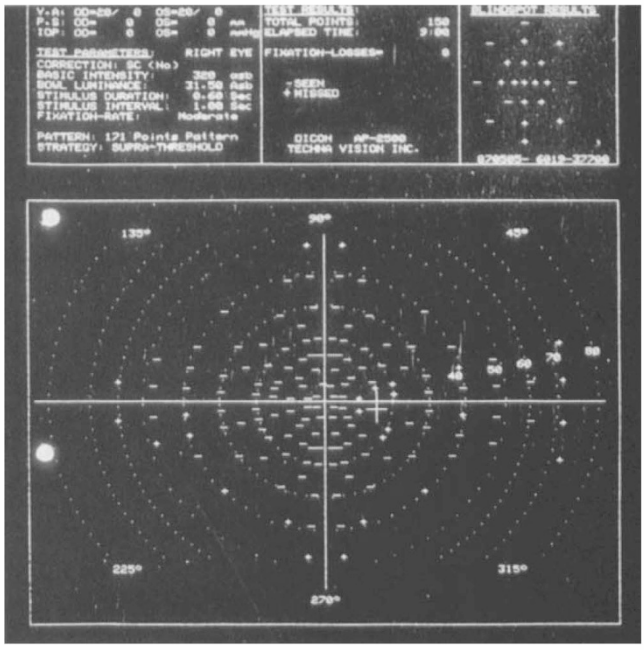

Fig. 2. *Dicon field testing shows extension of the blind spot of the right eye.*

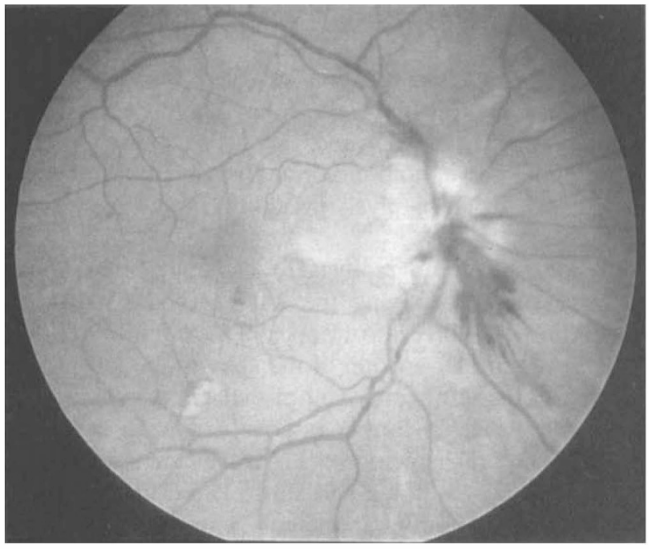

Fig. 3. *Right fundus showing papilloedema.*

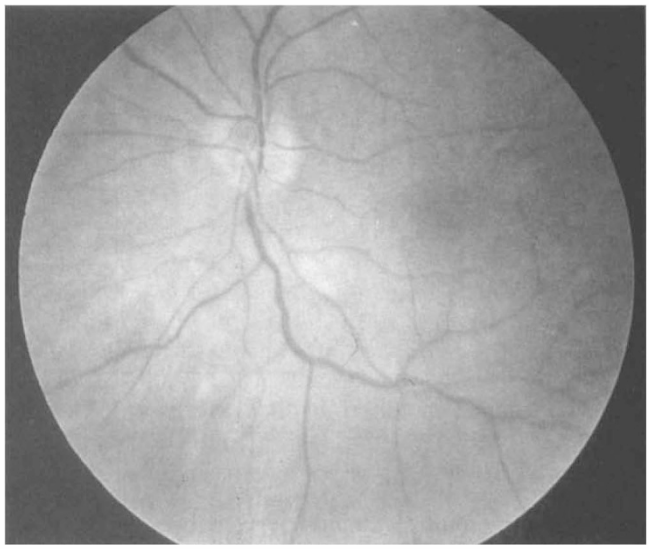

Fig. 4. *Left fundus showing a pale disc.*

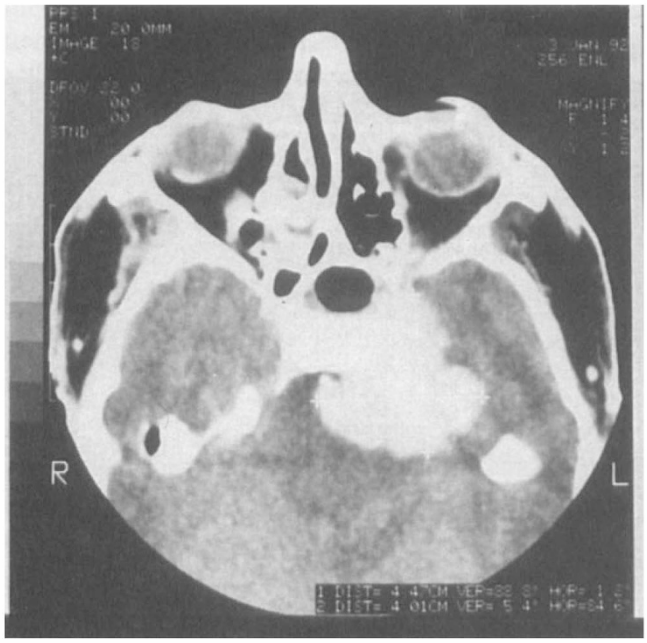

Fig. 5. Brain CT scan with contrast showing the tumour mass occupying the left cavernous sinus.

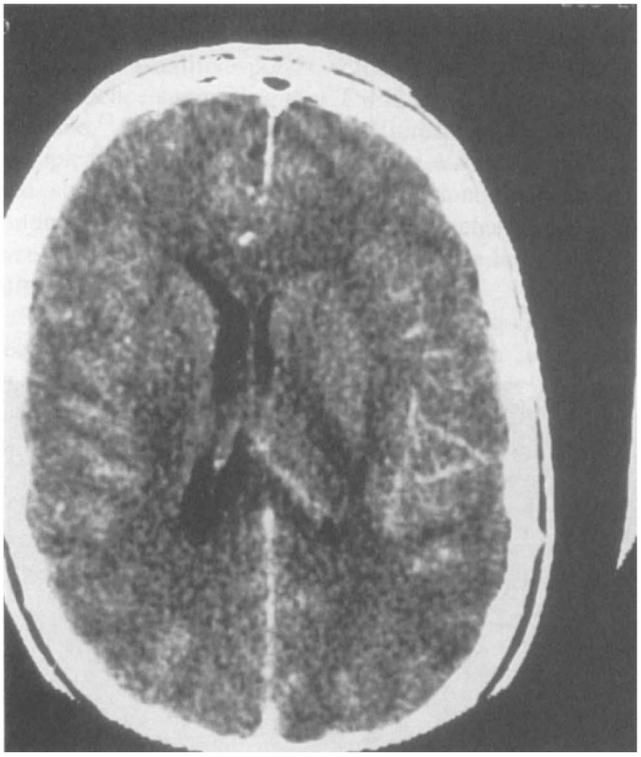

Fig. 7. *Brain CT scan showing brain oedema with dilatation of the ventricles causing shift of midline structures.*

sinus, causing multiple cranial nerve palsies. The tumour was treated by intravenous infusion of 5-fluorouracil and carboplatins, followed by a course of radiotherapy. Following this treatment the tumour mass had significantly diminished in size. This was followed by a relatively

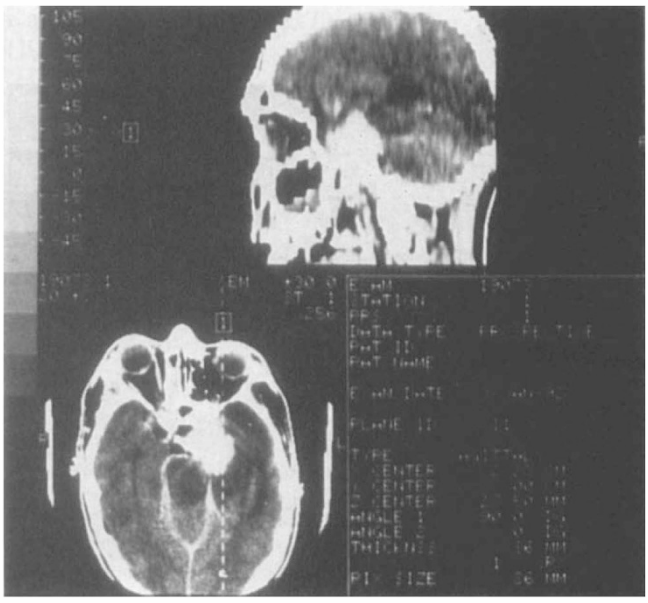

Fig. 6. Computer reconstruction of a sagittal section of the brain scan showing the vertical extension of the tumour into the left cerebral hemisphere.

symptom-free period until the patient started to experience visual disturbances.

Physical examination revealed a moderately built white male in no acute distress, oriented but somewhat unconcerned and euphoric in his responses. Visual acuity was 6/9 in the right eye and counting fingers in the left eye. There was an efferent and a relative afferent papillary defect on the left. There was no proptosis. Ocular motility testing was normal in the right eye but abnormal in the left eye as a result of multiple cranial nerve paralysis (Fig. 1). Dicon perimetry revealed an enlarged blind spot in the field of the right eye (Fig. 2). The fundus examination of the right eye showed elevated and hyperaemic disc with haemorrhages and cotton wool spots (Fig. 3). However, the left fundus examination showed a pale optic disc (Fig. 4).

Neurological examination revealed multiple cranial nerve palsies on the left side. There were partial oculomator, trochlear and abducent nerve palsies. Trigeminal nerve, both motor and sensory, was affected. There was facial paralysis in addition to cochlear nerve damage.

A computed tomography (CT) scan of the brain showed a large mass occupying the left cavernous sinus and extending anteriorly into the apex of the left orbit (Fig. 5). There was further erosion of the apex of the left petrous ridge and the left lateral wall of the sphenoid sinus. The scan also revealed further tumour extension superiorly into the cerebral hemisphere (Fig. 6), where much associated oedema was noted and shift of mid-line structures with dilatation of third and lateral ventricles (Fig. 7).

Patient was then re-referred for further treatment.

Discussion

The original cases of Foster Kennedy were due to baso-

frontal tumour. The optic atrophy is commonly felt to result from optic nerve compression and the contralateral papilloedema from increased intracranial pressure.[1,2] Another mechanism suggests that Foster Kennedy syndrome is due to bilateral direct optic nerve compression by a midline basal mass or less commonly by long-standing increased intracranial pressure without direct compression of either nerve.[3]

Since the early cases of Foster Kennedy syndrome, many cases have been reported in the literature caused by other tumours, especially meningiomas such as olfactory groove and sphenoid ridge meningiomas, with gliomas occasionally reported.[4–7] To our knowledge, nasopharyngeal carcinoma is rarely reported in the literature as a cause of Foster Kennedy syndrome.

Other terms have been used in the literature to describe atypical cases of Foster Kennedy syndrome. 'Pseudo Foster Kennedy syndrome' has been used to describe cases caused by non-compressive pathology such as anterior ischaemic optic neuropathy and optic neuritis.[8] 'Pseudo-pseudo Foster Kennedy syndrome' has been used to describe a case caused by two different pathologies such as meningioma and ischaemic optic neuropathy causing optic atrophy in one eye and swollen optic disc in the other.[9]

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Sir,

Apraclonidine in the Management of Glaucomatocyclitic crisis

Glaucomatocyclitic crisis (Posner–Schlossman syndrome) is a unilateral inflammation of the uveal tract in which signs of an acute increase in intraocular pressure predominate. As the aetiology is doubtful, numerous treatments have been suggested, the main aim being to reduce the exceptionally high intraocular pressure which, left untreated, will cause permanent optic nerve damage.

Apraclonidine hydrochloride 1%, a clonidine derivative and a peripheral alpha-adrenergic agonist, was developed to lower intraocular pressure while minimising systemic side effects. It has specific receptor-binding and physico chemical properties that limit its access to the central nervous system. In normal human volunteers it produces a significant fall in intraocular pressure.[1] Apraclonidine hydrochloride 1% is being used to reduce the intraocular pressure elevation after anterior segment laser surgery. It is effective in eliminating the large, acute elevation in intraocular pressure after argon laser trabeculoplasty.[2,3] It can also be used as an adjunctive glaucoma therapy.[4]

Case Report 1

The patient was a 37-year-old Malay woman referred by her general practitioner with a diagnosis of acute congestive glaucoma of the left eye. She complained of left-sided headache, and mild pain and redness of the left eye with slight blurring of vision for the preceding 3 days. On questioning she said that she saw haloes from the day of onset. This was her first episode.

Examination showed that her vision was 6/6 part, but she said that she felt as though she was seeing through water. Slit lamp examination did not show any significant oedema of the cornea. There were six unpigmented precipitates on the posterior surface of the cornea. There were no obvious precipitates at the angle. Aqueous did not show significant flare or cells. Gonioscopy revealed that the chamber angle was wide open. The pupil reacted less briskly compared with the fellow eye. The iris was similar in character when compared with the fellow eye. Posterior segment was normal. The intraocular pressure in the left eye was 50 mmHg and in the right eye was 14 mmHg.

At 9.50 a.m. the patient was asked to lie down and 1 drop of apraclonidine 1% was instilled in the conjunctival sac. The pressure was monitored every hour until 10.00 p.m. and again at 8.00 a.m. the next morning. Within 1 hour the pressure dropped from 50 mmHg to 18 mmHg. It remained at 18 mmHg for 6 hours and then rose to 22 mmHg during the seventh hour. Another drop of apraclonidine was instilled. Within an hour the pressure went down to 18 mmHg and in another hour to 14 mmHg, and remained the same throughout 3 days of monitoring. Examination on the third day showed the fields were normal. There was no significant fluctuation of pressure on regular follow-up.

Case Report 2

The patient was a 25-year-old Chinese woman. She was