This 64-year-old woman presented to a general practitioner in 2010 with a decrease in vision of the right eye for 2 weeks with no other symptoms such as vomiting or headache. At physical examination, her limb powers were full and her reflexes were normal with downgoing Babinski reflex. The visual acuity of her right eye was hand movements and that of her left eye was 0.6 with hyperopic spectacle correction, and she had normal intra-ocular pressures. The visual field was otherwise unremarkable. Given such a clinical picture, the ophthalmologists were consulted and a full examination was done.

At examination of her fundi, the right optic disc was pale (Fig 1) and the left optic disc showed features of disc swelling of hyperaemia with blurred margin (Fig 2). Her smell sensation was preserved. A clinical diagnosis of Foster Kennedy syndrome was made. The patient was referred to the neurosurgeons for further investigations. Computed tomographic scan of the brain showed a right middle cranial fossa mass with extension into the right cavernous sinus (Fig 3). Magnetic resonance imaging of the brain with contrast showed an extra-axial lesion over the right sphenoid ridge, with avid homogenous contrast enhancement and significant mass effect causing compression on the pons and midbrain and with midline shift (Fig 4). Dexamethasone was started
at full dose with 4 mg 4 times a day and debulking surgery was performed. The tumour was encasing the internal carotid artery, so careful dissection of the tumour was done. Histology showed that the tumour was a meningioma.

Foster Kennedy syndrome was first described in 1911 by Dr Robert Foster Kennedy.1 This syndrome is a combination of optic atrophy of one eye and papilloedema of the other eye. Several reports of this phenomenon have been published and multiple intracranial pathologies have been described, including meningioma, pituitary adenoma, and oligodendrogliomas.2,4 The most common hypothesis is direct compression of the optic nerve by the tumour causing optic atrophy in one eye and papilloedema in the fellow eye due to increased intracranial pressure.

This patient's presentation differed from the usual clinical presentation in that she did not have any symptoms of increased intracranial pressure such as headaches, nausea, or vomiting. Her reason for presentation was visual disturbance. The ophthalmologists were able to make the correct diagnosis and the patient was treated accordingly. Despite the fact that Foster Kennedy syndrome is uncommon, awareness by different specialists is important to provide a better outcome for patients.

References

Answers to CME Programme

Hong Kong Medical Journal February 2011 issue

Hong Kong Med J 2011;17:11–9

I. Hepatocarcinogenesis of regenerative and dysplastic nodules in Chinese patients

A


B


Hong Kong Med J 2011;17:20–5

II. Total knee arthroplasty for primary knee osteoarthritis: changing pattern over the past 10 years

A


B