Case report

Foster Kennedy syndrome

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Sri Lanka Journal of Neurology, 2014, **3**, 33-34

Index words: Foster Kennedy syndrome, meningioma, papilloedema, optic atrophy

Introduction

Foster Kennedy syndrome is a sign in neurology which is seen rarely but spoken about frequently. Since the development and wide spread availability of numerous neuroimaging modalities, this sign is a rarity in the modern world. Foster Kennedy syndrome is ipsilateral optic atrophy and contralateral papillodema caused by an intracranial mass. Since it was first described by Sir Robert Foster Kennedy back in 1969, many case reports have been published. Ophthalmologic examination and neuroimaging is essential for the diagnosis of Foster Kennedy syndrome. We present a case of unilateral blindness due to a large sphenoidal wing meningioma causing Foster Kennedy syndrome and discuss the history, etiopathogensis and clinical relevance of this sign in today's context.

Case report

A 49-year-old woman admitted to hospital with a complaint of visual impairment for about two weeks. She complained of total blindness in the left eye. She had not experienced any signifiant headache or any eye pain. She did not have emotional lability, depression nor abnormal behaviour. The patient was not on any medications. The past medical, gynaecological, family, surgical histories were not significant.

On examination, the patient was alert and Glasgow coma scale was 15. The pupillary examination revealed a left relative afferent papillary defect. Sense of smell was normal. Fundocscopy revealed left optic atrophy and right papilloedema. Extraocular eye movements were normal. All other cranial nerves were normal. Her visual acuity was 6/9 in the right eye and 1/60 in the left eye. The retinal photography shows left optic atrophy and right papilloedema (Figure 1).

All her basic blood investigations were normal. The contrast computed tomography of the head showed a large mass 5X5 cm in the left frontal region with some calcification in the middle suggestive of a meningioma. A magnetic resonance imaging confirmed the diagnosis of a meningioma arising from the lesser wing of sphenoid encasing the supraclinoid portions of the left internal carotid and compressing the intracranial portions of the left optic nerve causing mass effect (Figure 2 and 3).

The patient was started on dexamethasone for cerebral oedema and was transferred to a neurosurgical unit for surgical management. The patient is presently awaiting surgery.

Figure 1. Papilloedema in left and optic atrophy in right eye.

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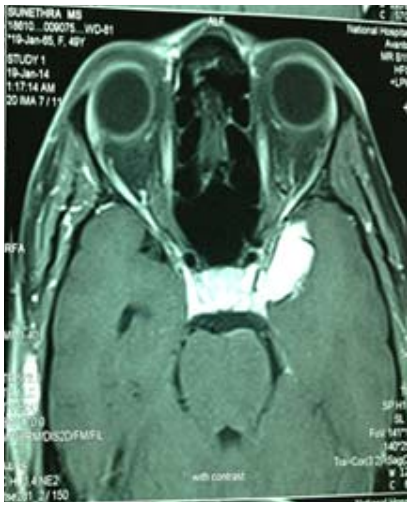

Figure 2. MRI brain showing left lesser wing sphenoid meningioma.

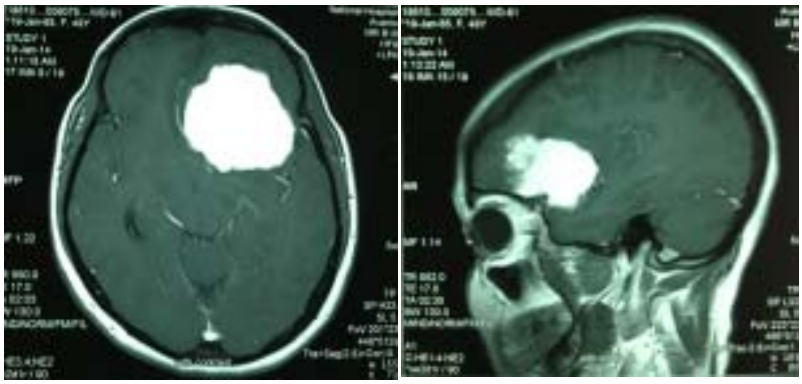

Figure 3.

Discussion

Foster Kennedy published his article titled, "Retrobulbar Neuritis as an Exact Diagnostic Sign of Certain Tumors and Abscesses in the Frontal Lobes" in 1991[1]. The six cases he discussed were tumours and abscesses of the frontal lobe and the mass can be a meningiomas of the olfactory groove, falx, sphenoidal wing or subfrontal regions, craniopharngiomas, pituitary adenomas plasmacytomas and large aneurysms[1].

In them he described the occurrence of retrobulbar neuritis and optic atrophy on the side of the lesion together with papillodema of the contralateral eye.

Mechanism

The Foster Kennedy sign represents optic atrophy and anosmia on one side, with papilloedema caused by raised intracranial pressure on the other[2]. Majority of cases occur without anosmia. The exact pathogenesis of Foster Kennedy syndrome in unclear. It is postulated that an intracranial mass causes direct pressure on the optic nerve, causing impaired ocular venous return and compression in the subarachnoid space of the intra-orbital part of the optic nerve[3]. This mechanical compression results in ipsilateral optic atrophy. Therefore the development of papilloedma is prevented in that eye. The elevated intracranial pressure causes papilloedma of the contralateral side[4,5].

Variations

Pseudo-Foster Kennedy syndrome is described as unilateral optic disc swelling with contralateral optic atrophy in the absence of an intracranial mass causing compression of the optic nerve. This occurs mainly due to bilateral sequential optic neuritis, ischaemic optic neuropathy and unilateral optic nerve hypoplasia[6,7,8,9].

Conclusion

Foster Kennedy syndrome is rare. Today any patient complaining of persistent headache undergoes neuroimaging and an intracerebral lesion is found before visual loss develops and hence the rare occurrence of this sign. Typical syndrome due to mass lesions are very unlikely to be seen today especially in the developing world, but rare cases still can come up in countries where imaging facilities are not freely available and where access to proper health care is difficult. Foster Kennedy syndrome should be considered in patients who present with unilateral visual impairment, or a junctional scotoma and headache.

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