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Foster Kennedy syndrome secondary to oligodendroglioma

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Abstract

Foster Kennedy syndrome (FKS) is rare. It is characterised by the presence of ipsilateral optic atrophy, contralateral papilloedema and ipsilateral anosmia. Since its first description in 1911, it has never been reported in oligodendroglioma. Here we discuss the first case of a patient with oligodendroglioma presenting with FKS.

Keywords

Foster Kennedy syndrome; oligodendroglioma.

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Introduction

This case report represents the first description, in the literature, of an oligodendroglioma as the cause for Foster Kennedy syndrome (FKS).

Case report

A 33-year-old, left handed gentleman was admitted in November 2002 with right eye vision deterioration over 6 weeks and headaches which were exacerbated by coughing and postural changes. Since 1996, he had a history of partial and generalised seizures secondary to an intracranial tumour. Previous magnetic resonance (MR) imaging had demonstrated a tumour with the appearance of a right subfrontal low-grade glioma (Fig. 1). The seizures commenced with an aura of odd epigastric sensations progressing to involuntary jerking of the left arm and leg with speech arrest, and the development of secondary generalisation characterised by tongue biting and incontinence. Medical management, with levetiracetam 3000 mg and Lamotrigine 300 mg daily, limited the seizures to simple, partial seizures every 1-2 weeks and secondary, generalised seizures once a month.

On examination, he had right sided vision of 6/60 with a central scotoma and optic atrophy (Fig. 2), left sided vision of 6/9 with papilloedema (Fig. 3), and right sided anosmia. MR imaging showed an increase in the size of the previously demonstrated right subfrontal tumour with surrounding oedema and minimal enhancement. A stereotactic biopsy of the lesion was undertaken and histology revealed a low-grade oligodendroglioma (Fig. 4). In view of the rapid growth of the tumour, the patient was subsequently given fractionated radiotherapy.

Discussion

FKS is a very uncommon syndrome and due to the current availability of computed tomography and MR imaging it is rarely observed^[1,2]. The syndrome was first described in

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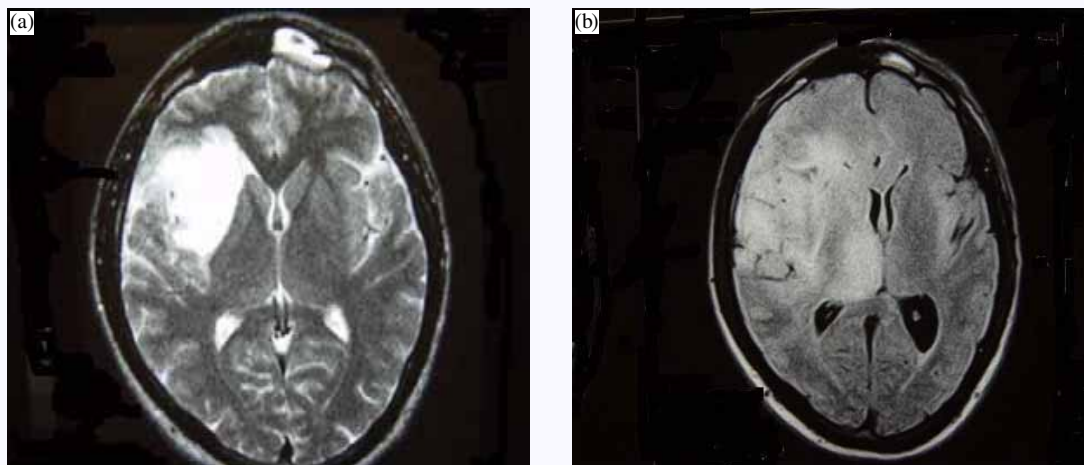


Fig. 1. T2-weighted axial MR scan showing the lesion in 1996 (a). Flair axial MR scan showing the lesion in 2002 (b).

1911 by the neurologist Foster Kennedy, who presented a series of six patients with the triad of ipsilateral optic atrophy, contralateral papilloedema and ipsilateral anosmia^[3]. It is usually caused by a large, frontal lobe tumour or a tumour arising from either the olfactory groove or the medial third sphenoidal wing. Histology invariably shows a meningioma^[2].

The aetiological mechanism of this syndrome is unclear. Foster Kennedy originally hypothesised that ipsilateral optic atrophy resulted from direct pressure on the optic nerve, and the contralateral papilloedema from long-standing elevated intra-cranial pressure. An analysis of reported cases showed that in 22% the above applied, in 33% there was bilateral optic nerve compression, in 5% there was long-standing, increased intracranial pressure and in 40% the mechanism was unclear^[2-7]. This case report supports the original

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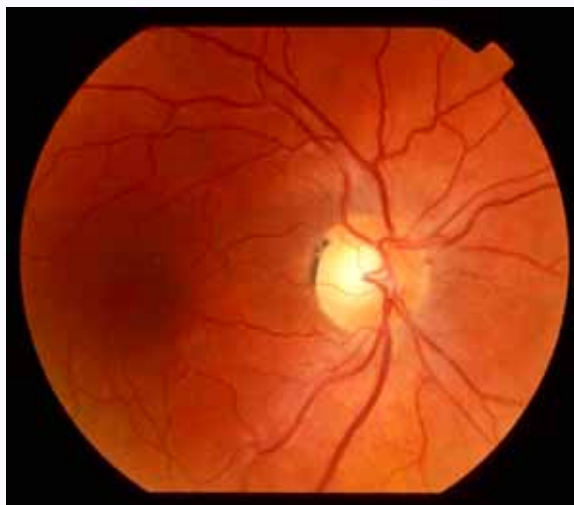


Fig. 2. Right fundus showing optic atrophy.

hypothesis of Foster Kennedy, as there was direct compression of the right optic nerve and clinical features of raised intracranial pressure.

The interesting aspect of this case report is the histology. A literature search reveals that FKS caused by an oligodendroglioma has never previously been reported.

Oligodendrogliomata are relatively rare primary brain tumours composed of neoplastic oligodendrocytes. They generally present with seizures and are associated with a long natural history and unpredictable biological behaviour^[8-10]. In cases where surgical resection is not possible, surgical intervention in the form of resection or needle biopsy is the mainstay of treatment. Radiotherapy represents one of the standard adjuvant treatment modalities in cases of low-grade oligodendrogliomata. Chemotherapy is reserved

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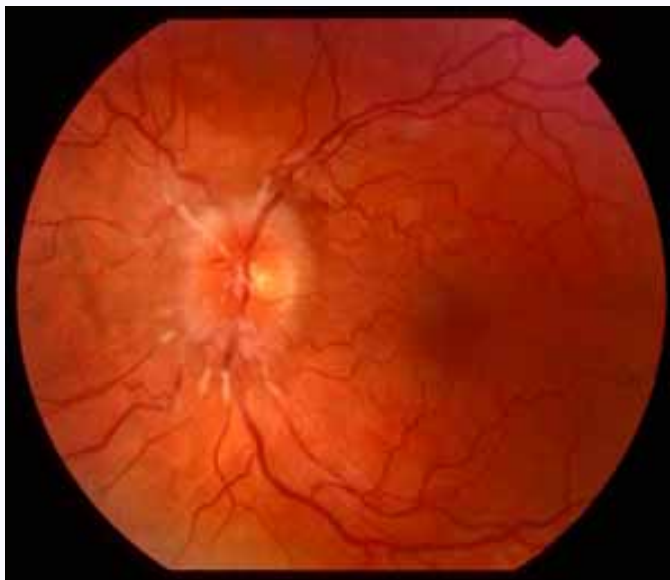


Fig. 3. Left fundus showing papilloedema.

for those with recurrence following radiotherapy. The median survival periods range from 8 to 10 years in cases of low-grade oligodendrogliomata. Large series have reported no plateau in survival, so radiotherapy has been proposed to optimise surgery and to delay recurrences. However, there has been no randomised trial assessing the optimal timing and the beneficial role of radiotherapy. Some advocate radiotherapy at an early stage of the disease, while others follow a non-aggressive management, with irradiation only at the time of progression^[8,10-12].

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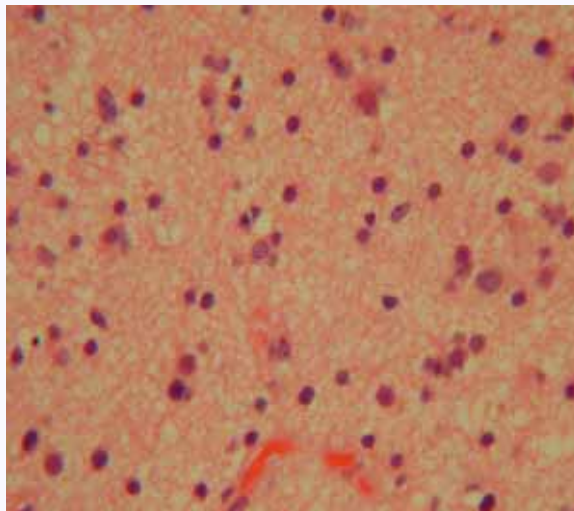


Fig. 4. Histology demonstrating oligodendroglioma with no high-grade features.

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