Clinical Reports

Optochiasmatic tuberculoma causing progressive visual failure: when has medical treatment failed?

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Summary: A 5 year old girl with tuberculous meningitis developed progressive visual failure during in-patient anti-tuberculous chemotherapy due to an optochiasmatic tuberculoma. This was successfully managed by prolonged high-dose corticosteroids and continued anti-tuberculous therapy resulting in complete visual and psychosocial recovery.

Introduction

Intracranial tuberculoma can develop or enlarge in patients treated for tuberculosis during supervised anti-tuberculous chemotherapy. A course of corticosteroids and continued anti-tuberculous therapy for a longer period of time than uncomplicated cases usually result in complete resolution of these lesions. However, despite high-dose corticosteroids and adequate anti-tuberculous treatment, intracranial tuberculoma can still give rise to raised intracranial pressure or compression of vital structures such as the anterior optic pathway causing progressive visual failure, or posterior fossa causing brainstem compression or hydrocephalus. Under such circumstances, timely surgical decompression is necessary to preserve life or important functions such as vision.

We recently managed a child with tuberculous meningitis who developed an optochiasmatic tuberculoma causing progressive visual loss. Surgical decompression was planned but not required because high-dose corticosteroids and continued anti-tuberculous therapy resulted in complete recovery.

Case report

A 5 year old girl presented with a 10 day history of a pyrexial illness associated with headache, nausea and vomiting. On examination, she was febrile with neck rigidity but fully conscious. Pupils, ocular movements and fundi were normal. Chest radiograph was normal. Cerebrospinal fluid (CSF) examination revealed a white cell count of 250/mm³ (60% polymorphs and 40% lymphocytes), glucose 0.8 mmol/l and protein 1.8 g/l (normal range 0.15–0.45 g/l). Ziehl-Nielsen staining of the CSF showed numerous acid-fast bacilli. A confident diagnosis of tuberculous meningitis was made and anti-tuberculous therapy commenced immediately which consisted of daily streptomycin 300 mg, isoniazid 200 mg, rifampicin 300 mg, pyrazinamide 500 mg and pyridoxine 10 mg.

Two weeks following in-patient supervised treatment of tuberculosis, she became drowsy and uncooperative complaining of a severe headache. The pupils were fixed and fully dilated. Fundi were normal. It was impossible to examine for visual field, acuity and ocular movements. Plain and contrast-enhanced computed tomography of the brain (CT) demonstrated an extensive optochiasmatic tuberculoma (Figure 1a) but there was no evidence of hydrocephalus. Intravenous dexamethasone 2 mg 6 hourly was prescribed. The clinical impression at the time was that she was probably blind from optochiasmatic compression of the tuberculoma. Surgical decompression might be necessary if corticosteroids failed.

At 48 hours after corticosteroids treatment, her headache, drowsiness and confusion resolved, both pupils were reacting to light (right pupil size 3 mm, left pupil size 5 mm) and the visual acuity of both eyes was 20/200. Her general condition and visual functions continued to improve. At 2 weeks after corticosteroids treatment, while the general condition and conscious level continued to improve, her visual acuity rapidly deteriorated to having no light perception in the right eye and 20/800 in the left eye. Repeat contrast-enhanced CT did not show...
Visual failure during the course of anti-tuberculous therapy may be due to drug toxicity, arachnoiditis and tuberculoma of the optochiasmatic apparatus, and 'cold' abscess of the sphenoidal jugum. The diagnosis of optochiasmatic tuberculoma is facilitated by contrast-enhanced axial CT. Coronal CT in cooperative patients demonstrates the relationships between the compressive lesion and the optic nerves and chiasm which is useful information when surgical decompression is considered.

Most intracranial tuberculoma resolve if adequately treated with anti-tuberculous therapy and a course of corticosteroids. Urgent surgical decompression should be carried out when medical treatment fails. In the context of tuberculoma compromising the anterior optic pathway, timely surgical decompression can give rise to favourable visual recovery. The present case can be regarded to have 'failed medical treatment' on dexamethasone 2 mg 6 hourly. Further trial of a higher dose corticosteroids was in preparation for surgical intervention rather than definitive treatment.

The pathogenesis of developing or enlarging tuberculoma in successfully treated tuberculosis is unknown. An exaggerated host reaction against tuberculous protein is thought to play an important role. The mechanism of corticosteroids in controlling intracranial tuberculoma is more likely to be diminution of this host–organism reaction rather than reduction of associated oedema. The dramatic visual improvement on high-dose corticosteroids in the present case is consistent with this immunological mechanism.

In conclusion, when optochiasmatic tuberculoma fails to respond with conventional medical treatment, high-dose and prolonged corticosteroid therapy may be successful in saving sight. This can be carried safely if emergency surgical decompression is prepared and can proceed when so indicated.
Useless hand of Oppenheim – magnetic resonance imaging findings

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Summary: A patient with multiple sclerosis developed a useless, deafferented left hand, as described previously by Oppenheim. Magnetic resonance imaging demonstrates that this is caused by an ipsilateral plaque of demyelination in the posterior columns of the cervical cord.

Introduction

In 1911 Oppenheim summarized his experiences of ‘the different types of multiple sclerosis’. Among these he described the occurrence of ‘sudden numbness and awkwardness of one arm’ in which ‘the sense of posture is most seriously affected’; he suggested that ‘this apparent acute posterior myelitis cervicalis is but a stage of disseminated sclerosis’.1 This clinical sign has become referred to as the ‘useless hand of Oppenheim’ and we report a patient in whom the site of the lesion responsible has been demonstrated.

Case report

A 38 year old man complained that he had lost the use of his left hand. Six years earlier he had presented with optic neuritis at which time nystig-
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