Clinical Reports

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

Wai S. Poon, A. Ahuja¹ and A.K.C. Li

Departments of Surgery and ¹Diagnostic Radiology and Organ Imaging, Prince of Wales Hospital, Chinese University of Hong Kong, Shatin, N.T., Hong Kong

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.^{1–3} 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.³ 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.^{3,4}

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 radio-

graph 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

Correspondence: Professor A.K.C. Li, M.A., M.D., B.Chir. (Cantab.), F.R.C.S.E., F.R.A.R.C.S., F.A.C.S. Accepted: 3 August 1992

Figure 1 Contrast-enhanced CT of a 5 year old girl 2 weeks after being treated for tuberculous meningitis when both her mental state and vision deteriorated. The optochiasmatic apparatus was encased by a tuberculoma where multiple foci of low density suggestive of micro-abscesses were demonstrated (left side panel). Contrast-enhanced CT when the patient had completed a 12 month course of anti-tuberculous therapy. The tuberculoma had resolved. Residual meningeal thickening in the region of previous tuberculoma was demonstrated (right side panel).

significant change of the optochiasmatic tuberculoma or ventricular size. Again surgical decompression was considered and prepared while high-dose corticosteroids prescribed (intravenous dexamethasone 10 mg 6 hourly).

Next morning after two doses of intravenous dexamethasone 10 mg, her visual acuity improved dramatically to 10/400 in the right, and 20/100 in the left eye. Dexamethasone 10 mg 6 hourly was maintained for 2 weeks when improvement of visual acuity continued (20/40 in the right, 20/100 in the left eye). Oral dexamethasone 5 mg 6 hourly was prescribed for a further 8 weeks followed by a slow tailing off regimen lasting 4 weeks. When she completed her 'high-dose and prolonged' corticosteroid therapy, 4 months after the onset of her illness, she was Cushingoid and grossly obese; visual acuity of the right eye was 20/25, left eye 20/40. Anti-tuberculous therapy was continued for a total of 12 months (except streptomycin which was prescribed for 3 months). Repeat contrast-enhanced CT showed resolution of the optochiasmatic tuberculoma (Figure 1b). At one year, her Cushingoid appearance had resolved, she attended normal school, her visual field and acuity were close to normal (20/25 in the right eye, 20/20 in the left).

Discussion

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.^{5,6} The diagnosis of optochiasmatic tuberculoma is fac-

ilitated 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.³ 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.^{4,7-9} 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.¹⁰ An exaggerated host reaction against tuberculous protein is thought to play an important role.³ 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 corticosteroids therapy may be successful in saving sight. This can be carried safely if emergency surgical decompression is prepared and can proceed when so indicated.

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Useless hand of Oppenheim – magnetic resonance imaging findings

R.J. Coleman, L. Russon, K. Blanshard and S. Currie

Departments of Neurology and Neuroradiology, St James's University Hospital, Leeds, UK

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-

mus was also noted. Subsequent sensory disturbances in the limbs and episodes of leg weakness led to a clinical diagnosis of multiple sclerosis, supported by bilaterally delayed visual evoked responses and raised cerebrospinal fluid immunoglobulin concentrations. The current symptoms had begun with numbness of the left hand spreading over a few days to affect the left arm, trunk and leg. He then lost all use of his left hand and involuntary movements developed.

On examination he had a functionally useless left hand. There were continuous, slow, rhythmic flexion movements of the fingers on the left with a similar movement at the left wrist. The movements were present equally at rest and with the arms outstretched. The movements were involuntary and the patient could not feel the hand moving; they increased when the patient was distracted or closed his eyes. Joint position sense was absent in the fingers, wrist and elbow on the left and was impaired at the left shoulder. In addition there was subjective impairment of light touch sensation over the whole of the left upper limb and vibration sense

Correspondence: R.J. Coleman, M.D., M.R.C.P., Department of Neurology, Aberdeen Royal Infirmary, Foresterhill, Aberdeen AB9 2ZB, UK.

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