Trapezius muscle atrophy after whiplash injury: accessory nerve or cervical plexus lesion?

We have observed the rare occurrence of trapezius muscle atrophy after whiplash injury. Only one similar case has been reported previously.1

A 24 year old woman was waiting at traffic lights in her car and was changing a cassette when her right arm outstretched when another car ran into the back of hers. She was not wearing a safety-belt. Immediately she felt pain and tingling on the back of the neck but no other symptoms. Later that day she developed nausea and dizziness. The same day a neurological examination was normal. There was no radiological evidence of fracture or dislocation of the cervical spine. A soft collar did not control the neck ache.

One month later, she noticed a lowering of her right shoulder. Six months later examination revealed weakness and atrophy of the right trapezius: with her arms at her side, the right shoulder drooped and the scapula was slightly winged and laterally displaced. Abduction of the arms made these defects worse and increased the prominence of the rhomboid muscles. Muscle tests showed a global involvement of the right trapezius, more marked in the middle and lower parts. The sternomastoid and the other shoulder girdle muscles were not affected. Slight hypesthesia for touch and pinprick was appreciable in the region of the supraclavicular branches of the cervical plexus. Thoracic fluoroscopy showed normal diaphragmatic motility. CT and MRI of the cervical regions were normal. EMG examination revealed denervation of the middle and lower parts of the right trapezius and a reduced recruitment of low amplitude, and polyphasic potentials in the upper part at full effort. There was no spontaneous activity. Conduction studies along the right accessory nerve1 showed a slight delay of the motor action potential derived from the upper part of the trapezius (4.2 ms; normal range 2.1-3.7 ms), whereas no motor responses could be evoked in the middle and lower parts. There was no clinical improvement the following year.

The accessory nerve, after supplying the sternomastoid muscle with the contributions from C2 or C2-C3, often divides and forms a plexus with the fibres from C3-C4, or C4 alone, before entering the deep surface of the trapezius.1 A complete lesion of the accessory nerve in the posterior triangle, frequently observed in neck surgery, results in a partial denervation of the whole muscle, the upper trapezius being clinically more affected.1 The third and fourth cervical roots, in addition to containing the afferent fibres, provide motor innervation mostly to the lower trapezius through the cervical plexus.1 There is, however, some variability as occasionally the whole trapezius is supplied by these roots.1

In this case, the clinical and electro-physiological findings are consistent with a prominent lesion of the radicular contribu-

Cysticercus immunoblot assay in Indian patients with single small enhancing CT lesions

Single small enhancing lesions (SEL) are frequently seen on CT scans of Indian patients with seizures.1 Their aetiology has been disputed for a number of years. Recently it has been shown that not all these lesions are caused by cysticercosis.2,3 An excursion biopsy of the lesion following CT guided stereotactical localisation is the preferred method of obtaining a definitive pathological diagnosis. Since this procedure carries some morbidity and the disease process in most patients is benign and self-limiting, biopsy may not be desirable in the majority of individuals with SEL. Attempts have been made to use a serological test in the diagnosis of these lesions, the ELISA test being the one most frequently used.2 In early 1989, the Centers for Disease Control (CDC), Atlanta, reported an improved assay for detecting anti-cysticercus antibodies in the serum or CSF of patients with cysticercosis.4 The test uses lentil-lectin affinity purified antigens in an immunoblot assay. We have assessed its use in the diagnosis of patients with SEL.

Serum samples were drawn from 36 Indian patients whose clinical data are shown in the table. The immunoblot assay was analysed at CDC using the immunoblot assay and the results are shown in the table. Of the 18 patients with SEL only one had a positive result. Two patients with SEL had an excision biopsy and in both the biopsy showed a cysticercus granuloma. The other four positive results were in patients with multiple lesions. There were three patients with multiple lesions who had a negative result.

Preliminary evaluation of the CDC immunoblot assay had shown very high sensitivity.1 One or more of seven major glycoprotein bands was recognised in serum specimens of 97% of 108 parasitologically confirmed cases of neurocysticercosis. The sera of 376 patients with heterogenous infections did not show any bands (specificity = 100%). Subsequent prospective experience with the assay has shown that the number of 10 markers in the diagnosis is the most important factor determining test sensitivity. Seropositivity in patients with single, active lesions has been 71% and 36% in serum and CSF, respectively (M Wilson, unpublished data). One reason the low sensitivity of this test in patients with single lesions could be the poor systemic immune response. It is possible that the serum antibody levels in these patients are below the threshold required to obtain a positive result with the test.

The serological results reported here on seizure patients with CT abnormalities and other characteristics identical to those previously identified as neurocysticercosis1 demonstrate that the immunoblot assay is of little use for diagnostic confirmation of SEL. A negative result in a patient with SEL does not exclude a diagnosis of cysticercosis.

In Indian patients with seizures and a well defined, rounded SEL a confident diagnosis of cysticercosis can be made. However, it is in patients with a lobulated, ill-defined lesion larger than 10 mm that the diagnosis becomes doubtful. Even in the latter group anticonvulsants alone can be given and a CT scan obtained after eight to 12 weeks to look for a change in the size or configuration of the lesion and the surrounding oedema. If early diagnosis is the ideal and our results indicate that serological tests do not provide an answer in the majority of patients. Thus in patients with negative serology and a doubtful diagnosis, excision biopsy following CT guided stereotactical localisation seems to be the only method of arriving at a diagnosis.

We thank Drs Anna Oommen and V Sivakumar for their help in the collection and processing of the sera.

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Table

<table>
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<th>Diagnoses</th>
<th>Number</th>
<th>Positive</th>
<th>Negative</th>
</tr>
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<tbody>
<tr>
<td>Healthy volunteers</td>
<td>5</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>SEL†</td>
<td>18</td>
<td>17</td>
<td>1</td>
</tr>
<tr>
<td>Healing SEL†</td>
<td>5</td>
<td>0</td>
<td>5</td>
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<tr>
<td>Multiple neurocysticercosis</td>
<td>7</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Total</td>
<td>36</td>
<td>31</td>
<td>5</td>
</tr>
</tbody>
</table>

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3 Abhishek GB, Behari M, Prasad K, Goulartia RK, Jailkhani BL. Disappearing CT lesions in epilepsy: is tuberculous or cysticercus the
Chronic hemidystonia following acute dystonic reaction to thiethylperazine

Thiethylperazine is a phenothiazine neuroleptic drug that blocks postsynaptic dopamine D2 receptors and is extensively used for the treatment of vertiginous syndromes. Its extrapyramidal side effects include acute and tardive dystonic reactions and a Parkinsonian syndrome. The acute reactions are usually self-limiting and brief and can be treated with anticholinergic therapy. We report an unusual case of a chronic hemidystonia which began immediately after treatment with thiethylperazine.

A 47-year-old man, with no previous personal or family history of neurological illness, was evaluated in December 1988 in our unit because of involuntory movements and postures of his right limbs. When he was aged 40 he developed a vertiginous syndrome (retrospectively diagnosed as “vestibular neuritis”) and was treated with thiethylperazine 6.5 mg three times daily. When he was aged 41, he developed dystonic movements involving the right side, which was associated with bilateral piperidin 5 mg intravenously. Twenty-four hours later, however, he developed dystonic postures and movements of the right limb, neck and trunk that persisted until the day of his admission. Apart from his movement disorder, the general and neurological examination were normal. A blood cell count, biochemistry, urinalysis and copper studies in plasma and urine, were all normal, and serological tests for syphilis were negative. EEG, cranial CT and MRI showed no abnormalities. The dystonic movements improved moderately with piperidin 8 mg/day, although he has a persistent action dystonia of his right limbs, which is more noticeable during walking.

Dystonia has been classified according to distribution as focal, multifocal, segmental, or generalised, and when its distribution involves the ipsilateral arm, leg, and face, the term “hemidystonia” is applied. The presentation of dystonia in the basal ganglia in our patient on CT and MRI, together with the onset of this clinical picture immediately after thiethylperazine exposure, implies that hemidystonia may be caused by a dystonic unmasking (in a subject possibly already predisposed to develop idiopathic dystonia) by phenothiazines.

Acute dystonic reactions after exposure to phenothiazines or other neuroleptic agents usually disappear after withdrawal of the relevant drug and/or anticholinergic therapy. Nevertheless, some cases have been reported in the literature of prolonged or chronic dystonia. The series of tardive dystonia by Burke et al includes 42 patients with dystonia after exposure to antipsychotic drugs. Dystonia developed after a mean interval of exposure of 3.7 years. In none of these cases nor in recent series of tardive dystonia, was the distribution ever hemidystonic. Our case therefore appears to be the first report of hemidystonia occurring as a direct or indirect side effect of a neuroleptic drug.

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Bilateral metastases in the cerebellopontine angle

Only 0.2% of the pontocerebellar angle (PCA) tumours are metastases. To our knowledge, we report the first case of bilateral metastasis.

In 1974, a 64-year-old woman presented with a malignant melanoma of the left leg which was treated by exeresis and chemotherapy. An inguinal lymphadenopathy was found in 1976 followed by a subcutaneous metastasis on the right deltoid region six years later were treated surgically.

From June to July 1978 the patient experienced a right-sided hearing loss, an unsteady gait with vertigo, a swallowing impairment and dysphonia. The brain CT scan and the CSF analysis were unremarkable. The patient was admitted to our hospital on 20 July because of a right facial palsy and left hypoaesthesie.

On the right side, there was involvement of the VIth and VIIth cranial nerves. On the left, the Vth, VIIth and VIIIth cranial nerves were involved slightly. The patient appeared ill but on skin examination there was no evidence of malignant melanoma.

The CT scan (fig a) showed two hyperdense lesions which on MRI (fig b) were isointense during T1 and hypointense during T2. The first image, which was round, was in the right PCA and disrupted the brain stem and the cerebellum without invading them; the second, which was oval and smaller in size, was in the left PCA, without mass effect.

On 10 August 1987 a soft, haemorrhagic, yellowish, self contained (2 cm x 3 cm) tumour was found in the right PCA, disrupting the last cranial nerves, forcing back the brain stem on the median line, and spreading onto the jugular foramen. An identical tumour on the left (1.5 cm diameter) did not press on the brain stem but spread along the last cranial nerves without disrupting them.

The histopathological examination showed that they were metastases of an undifferentiated cancer; cells of epithelial type were associated with long cells which looked like sarccoma. On electron microscopy, there was no melanosoma, but small stick inclusions in the ergastoplasm cisterns (75% of cells), of variable orientation, often crossed and made from straight microtubules, suggesting a melanoma origin.

The patient died in October 1987. A necropsy examination could not be performed.

Cornillet reported the first case of a metastasis developed in the PCA; the primary lesion was an oropharynx epithelium. Since then, most of the unusual published cases are isolated ones, originating from histologically differing types as such: lung, breast, oropharynx carcinoma, carcinoma of probably colon origin, histiocytic malignant lymphoma, malignant histiocytoma, malignant fibrous xanthoma. No case of melanoma was reported.

In our case, there were several inclusions of straight microtubules in the ergastoplasm cisterns on histopathological examination, similar to those observed in 6% of cases of

Figure 1A CT scan with contrast: two hyperdense lesions of the right and the left CPA; B: MRI (T2 weighted): the two lesions are hypointense.
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