

LETTERS TO THE EDITOR

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 with 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 hypoaesthesia for touch and pinprick was appreciable in the region of the supraclavicular branches of the cervical plexus. Thoracic fluoroscopy showed normal diaphragm motility. CT and MRI of the cervical regions were normal. EMG examination revealed total 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 nerve² 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.³ 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.⁴ There is, however, some variability as occasionally the whole trapezius is supplied by these roots.⁵

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

tions. The unusual position of our patient at the time of the collision, implying torsional components in addition to the usual hyperextension-flexion movement of the neck, may explain the rarity of this condition. The combination of these rapid movements may have produced a stretch-induced lesion of the cervical plexus fibres, which suggests a poor prognosis.

L TOSI
G ZANETTE
Servizio di Neurologia,
Ospedale "Sacro Cuore" Negrar,
Verona, Italy

- Mumenthaler M, Schliak H. *Lasionen peripherer Nerven. Diagnostik und therapie.* Stuttgart-New York: Thieme, 1987:214.
- Petrera JE, Trojaborg W. Conduction studies along the accessory nerve and follow-up of patients with trapezius palsy. *J Neurol Neurosurg Psychiatry* 1984;47:630-6.
- Soo KC, Hamlyn PJ, Pegington J, Westbury G. Anatomy of the accessory nerve and its cervical contributions in the neck. *Head Neck Surg* 1986;9:111-5.
- Coleman CC, Walker JC. Technics of anastomosis of the branches of the facial nerve with spinal accessory for facial paralysis. *Ann Surg* 1950;131:960.
- Fharer H, Ludin HP, Mumenthaler M, Neiger M. The innervation of the trapezius muscle. An electrophysiological study. *J Neurol* 1974;207:183-8.

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 most if not all these lesions are caused by cysticercosis.^{2,3} An excision biopsy of the lesion following CT guided stereotaxic 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 SELs. Attempts have been made to use a serological test in the diagnosis of these lesions, the ELISA test being the one most frequently used.³ 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 lyophilised samples were analysed at CDC using the immunoblot assay and the results are shown in the table. Of the 18

Table Clinical data of patients and results of the assay

Diagnosis	Number	Serology	
		Positive	Negative
Healthy volunteer	5	0	5
SEL*	18	1	17
Healed SEL†	5	0	5
Multiple neurocysticercosis	7	4	3
Tuberculoma	1	0	1
Total	36	5	31

*SEL = Single, small, enhancing CT lesion.
†Healed SEL = "Disappeared" or calcified.

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.⁴ One or more of seven major glycoprotein bands was recognised in serum specimens of 97% of 108 parasitologically confirmed cases of neurocysticercosis. The serum of 376 patients with heterologous infections did not show any bands (specificity = 100%). Subsequent prospective experience with the assay has shown that the number of intraparenchymal cysticerci 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 for 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 neurocysticercosis² 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. An 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 doubtful diagnosis, excision biopsy following CT guided stereotaxic 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.

VEDANTAM RAJSHEKHAR*
Department of Neurological Sciences,
Christian Medical College and Hospital,
Vellore, India
MARIANNA WILSON
PETER M SCHANTZ
Parasitic Diseases Branch,
Parasitic Diseases Division
Center for Infectious Diseases,
Centers for Disease Control,
Atlanta, Georgia 30333, USA

*Present address: Section of Neurosurgery, Dartmouth-Hitchcock Medical Center, Hanover NH 03756, USA.

- Wadia RS, Makhale CN, Kelkar AV, et al. Focal epilepsy in India with special reference to lesions showing ring or disc-like enhancement on contrast computed tomography. *J Neurol Neurosurg Psychiatry* 1987;50:1298-301.
- Chandy MJ, Rajshekhar V, Prakash S, et al. Cysticercosis causing single small CT lesions in Indian patients with seizures. *Lancet* 1989;i:390-1.
- Ahuja GK, Behari M, Prasad K, Goulatia RK, Jaikhanani BL. Disappearing CT lesions in epilepsy: is tuberculosis or cysticercosis the