Ligature injury to the accessory nerve

Sir: We report a 64 year old woman who experienced a flu-like illness and within 2 weeks noted a “knot” on the right side of her neck. She saw her physician who biopsied the mass. The procedure note indicates that bleeding occurred, clamps applied to achieve hemostasis, and vessels subsequently ligated. The biopsy was reported as “lymphoid tissue”. Three days later she noted soreness in her right shoulder which spread over her chest wall. Over the ensuing week she was unable to abduct her right arm without considerable pain. Two months after onset the patient was seen by a neurologist with complaints of right shoulder and ipsilateral chest wall pain aggravated by abduction. Neurological examination revealed wasting of the right trapezius muscle. She could not abduct her right arm beyond 80° and mild winging of the right scapula was present. The remainder of the neurological examination was normal. There was a well-healed transverse scar approximately 3 cm in length over the midportion of the posterior border of the right sternocleidomastoid muscle.

Ten weeks after biopsy electromyological studies were done. Accessory nerve conductions were performed at standard distances according to the method of Cherington. Stimulation of the right (proximal to the scar) and left accessory nerves at the posterior borders of the sternocleidomastoid muscles and recording with surface electrodes from the upper portions of the trapezius muscles revealed motor latencies of 5.0 and 1.8 ms, respectively. The amplitude of the evoked compound muscle action potential (CMAP) measured on the left side was 10 mV but on the right side was 0.3 mV (that is, 3% of normal side). Needle electrode examination of the right upper and middle trapezius muscle showed some fibrillations at most insertions. The motor unit potentials (MUPs) were markedly decreased in number and minimally increased in duration, amplitude and complexity. MUPs were rapidly recruited at minimal effort. Electrical studies of the right ulnar and median nerves plus needle electrode examination of other right upper extremity muscles was normal.

Clinical and electromyological tests were compatible with a right accessory neuropathy. Despite evidence of denervation, conduction studies indicated some nerve continuity. The patient underwent surgical exploration. Two non-absorbable sutures, 1 cm apart, were found “stran-gulating” the nerve just as it divided into two branches. The ligatures were removed and continuity of the nerve confirmed.

The day following surgery the patient was able to abduct her right arm beyond 90°. Two months after surgery she had gained considerable strength in her trapezius muscle bulk increased, and her pain greatly diminished. Functionally, she could abduct her arm to a vertical position. Electrophysiological examination revealed the amplitude of the evoked CMAP recorded from the right upper trapezius to be 59% compared with the left. Six month follow-up showed the amplitudes of the evoked CMAPs to be symmetrical (9.0 mV). Motor latency on the left was again 1.8 ms whereas the right had decreased to 2.5 ms. No fibrillations were found in the right trapezius on needle electrode examination. MUPs were slightly decreased in number and several were definitely increased in amplitude, duration and complexity.

This woman’s accessory neuropathy is notable for its rapid initial improvement following ligature release. The onset of symptoms was also relatively slow over days rather than abrupt. The acute nerve compression model of Ochoa could not be the sole pathophysiological mechanism because of the prolonged time that the ligatures were present and by the lower amount of pressure that the ligatures produced. A more plausible explanation may derive from compression of the nerve microvasculature, producing ischaemic anoxia in the 1 cm segment isolated by the two ligatures. Oxidative metabolism occurs throughout the length of the axon and has been suggested to maintain not only axoplasmic flow, but also the Na⁺-K⁺-ATPase system of the axon membrane upon which excitability is dependent. In experimental models using compression to induce ischaemic anoxia, even markedly reduced axoplasmic flow may recover rapidly, with excitability soon following. Reduction of axoplasmic flow by compression alone has an uncertain effect on excitability.

The delayed improvement (after 48 hours) of our patient is more likely due to direct physical damage to the myelin sheath. The motor latency of the accessory nerve proximal to the lesion was prolonged, a feature suggesting myelin damage in those fibres conducting an impulse. The position of the injury precluded distal stimulation and conduction block could not be demonstrated. The amplitude of the evoked CMAP was already 59% of the normal side at 2 months after operation, an insufficient time for axonal regrowth. The MUPs recorded at 6 months were not so greatly enlarged or complex as would be seen if reinnervation was the prominent mechanism for recovery.

There are numerous causes for accessory neuropathy. This case demonstrated that prolonged compression from ligature directly around the accessory nerve may have a favourable outcome. Previous series emphasise the importance of electrophysiological investigation of iatrogenic accessory neuropathies but also show a relatively poor prognosis. None had a lesion similar to the present case. Ligature injury may be under-diagnosed and should be considered in persistent post-operative accessory neuropathy, particularly when the lesion is incomplete as the prognosis for recovery would appear favourable.

References

Letters


Accepted 26 March 1987

Smoking and dementia of Alzheimer type

Sir: Several surveys have reported a negative association between smoking habits and Parkinson's disease suggesting that smoking may decrease an individual's risk of developing the condition.1,2 An observation made by Appel3 that only six of 30 Alzheimer patients in his study had smoked at any time in their lives, led him to conclude that a similar effect may operate in dementia of Alzheimer type (DAT). This is of interest in view of the recently demonstrated loss of nicotinic receptors in the cerebral cortex of DAT patients.

Conversely, Shalat4 has presented data from a case control study, showing that Alzheimer patients were more than twice as likely to be smokers or ex-smokers than were controls. Furthermore, a decreased risk of the disease was positively correlated with level of cigarette consumption.

Our data refer to patients attending the Maudsley Memory Clinic and the psycho-geriatric unit of the Royal Bethlem and Maudsley Hospitals. The medical notes of 81 persons over the age of 65 with a diagnosis of “probable” dementia of Alzheimer type5 were reviewed to obtain information about smoking habits. Where such information was not recorded the next of kin were contacted by telephone. One hundred and twelve persons from a local luncheon club and care home were selected as age-matched controls and interviewed.

Seventy one per cent of male DAT patients and 32% of female DAT patients either smoked or gave a history of having smoked, as compared with 77% of male controls and 44% of female controls. Even allowing for sex differences (with the use of an appropriate log-linear model8) no significant difference in the lifetime prevalence of smoking between the two groups was found ($\chi^2 = 1.23$, p = 0.075, Odd's ratio = 1.58).

Our results do not support either study referred to above but are in accord with the findings of an epidemiological study of patients developing the disease before the age of 70.9 Clearly further studies are required to elucidate this question but can only be conducted if past and present smoking behaviour is perceived as an important variable and therefore accurately recorded in medical notes.

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References


6 Shalat S. Personal communication.


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Listeria monocytogenes infection with rhombencephalitis

Sir: Listeria monocytogenes meningitis is not uncommon,1,2 but rhombencephalitis as a complication is rare, only six cases being reported in the English literature.3-8 We describe a further case which draws attention to impaired central control of respiratory movement during the acute illness and to the development of new brain stem lesions long after the disappearance of active infection.

A previously healthy 43 year old Causcasian male presented with nausea, abdominal pain and headache of 4 days' duration. Increasing unsteadiness of gait, slurring of speech and difficulty swallowing had been present for two days. On the day of admission he developed numbness and left facial weakness. The temperature was 38°C, pulse 100 bpm and blood pressure 150/90 mm Hg. He was alert and orientated. The neck was supple. There was evidence for multifocal bilateral brainstem disease with dysarthria, right ptosis, pupillary asymmetry, left conjugate gaze paresis, left facial paralysis of lower motor neuron type, and weakness of the left palate and right side of the tongue. Pinprick and temperature sensation were impaired in a muddle distribution extending down the right side from the face to the T1 dermatome. There was dysmetry of the left arm and leg and gross truncal and gait ataxia.

A contrast enhanced CT brain scan and chest radiograph were normal. Cerebrospinal fluid (CSF) contained 125 cells/μl (56% polymorphonuclears, 38% lymphocytes, 6% monocytes), the protein was 0.76 g/l, and glucose 5.5 mmol/l. No organisms were present in the stained film or grew on culture.

He received intravenous fluids, ampicillin, clocaciillin and tobramycin. Five hours after admission, while awake, there was a rapid decrease in respiratory effort and he became cyanosed. He could take deep breaths on command but during a prolonged apnoeic episode a tonic-clonic seizure occurred. After recovery of consciousness automatic respiratory movement did not return and intermittent positive pressure ventilation (IPPV) was begun. On the following day Listeria monocytogenes was isolated from the blood cultures. Ampicillin and tobramycin were continued for four weeks.

The systemic evidence for infection settled over the first two days but there was further neurological deterioration with the development of complete right ptosis, right facial weakness, severe weakness of jaw opening and closure, paralysis of tongue protrusion...