

migraine⁸⁻¹⁰ and one with atypical cluster headache.⁷ These changes are compatible with arterial spasm of vessel wall oedema and were found to be transient whenever angiography was repeated. In our case, however, at least some of the arterial constrictions persisted. It is possible that they were not related to the patient's symptoms. Nevertheless, it is also known that the spasm following subarachnoid haemorrhage can leave the vascular lumen permanently narrowed because of ensuing mural fibrosis.¹⁶ The possibility arises that similar structural changes occurred in our patient, and the spasm described in vascular headaches could occasionally lead to permanent narrowings of the affected vessels.

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Resolution of a severe sensorimotor neuropathy following resection of an associated asymptomatic gastric lymphoma

Lymphoma may cause a peripheral neuropathy by nerve infiltration^{1,2} or by non-metastatic effects³⁻⁵ and occasionally this

may be the presenting feature.¹⁻⁴ We describe a patient presenting with a severe sensorimotor axonal neuropathy which resolved following the resection of an asymptomatic localised gastric lymphoma.

Over a period of two months a 75 year old woman developed difficulty in walking followed by distal paraesthesiae and increasing weakness in all four limbs. She had lost 6 kg (15% of body weight) but had no other symptoms. General and cranial nerve examinations were normal. There was generalised muscle wasting in the limbs, particularly marked distally, with fasciculations in the hand muscles and quadriceps. There was hypotonia and symmetrical proximal and distal weaknesses of all limbs (MRC grade 3 or 4-). All tendon reflexes were absent with no post-exercise potentiation. Plantar responses were flexor. Sensation was intact except for minor distal impairment of light touch and pinprick. There was no ataxia. During the next two months weakness and distal sensory loss progressed considerably. Power became grade 2 or less in all limb muscles and light touch and pin prick loss ascending to mid-limb level with complete loss of proprioception to wrists and ankles. Cranial nerves (except eleventh), autonomic nerves and sphincters were spared clinically. There was no response clinically or myometrically to an eight week trial of prednisolone (up to 1.5 mg/kg/day).

Electromyography on admission showed fibrillation potentials, positive sharp waves and a reduced interference pattern in upper and lower limb muscles. Sensory action potentials (SAP) were normal in the upper limbs but sural SAPs were not obtainable. Motor conduction velocities were slightly reduced: right median 47 ms, ulnar 43 ms and left lateral popliteal 41 ms.

Routine blood investigations were normal including serum electrophoresis, heavy metals and Borrelia serology. Urinary porphyrins were negative. Cerebrospinal fluid (CSF) cell count, protein and cytology, chest radiographs and CT, abdominal and pelvic ultrasound, sigmoidoscopy and rectal biopsy, bone marrow aspirate and trephine were all normal.

A partial thickness sural nerve biopsy was performed. Paraffin sections revealed severe axon loss and demyelination with no inflammatory cells or amyloid. Plastic sections confirmed extensive loss of myelinated fibres and all remaining ones were thinly myelinated. There were numerous Schwann cell processes but very few axon sprouts. Teased fibre preparations showed most fibres to be columns of Schwann cells with no debris. The small number of myelinated fibres were all regenerated fibres. On immunohistochemistry, there were no deposits of IgG, IgM, C1q or C3d or cells showing the leucocyte common antigen.

At gastroscopy a suspicious area in the gastric fundus was biopsied. This revealed extensive infiltration by B-cell monoclonal lymphocytes (markers MT1 negative, UCHL1 negative, MB1 positive, MB2 positive, Kappa negative, lambda positive). A proximal gastrectomy was performed, resecting all the macroscopically abnormal stomach. The left gastric nodes were enlarged but all other nodes, the liver and spleen and the rest of the bowel were macroscopically normal. Histology confirmed a well differentiated low grade B-cell lymphocytic lymphoma, extending to the serosa and involving four lymph nodes. It stained with monoclonal

antibodies to lambda light chains and IgM heavy chains. Her neuropathy improved dramatically over the next three months. She could stand unaided after four weeks, walk with a Rollator six weeks later and manage stairs and live independently by three months. Improvement was confirmed myometrically. Sensation became normal except for minimal proprioceptive impairment in the fingers and toes.

This elderly woman presented with a sensorimotor neuropathy which was of subacute onset, progressive and eventually very severe. The clinical suspicion of an underlying malignancy was eventually confirmed by the finding of a B-cell gastric lymphoma. The lack of demonstrable spread despite extensive investigation, the normal CSF findings and the lack of infiltration on sural nerve biopsy suggests that this neuropathy is a paraneoplastic phenomenon rather than due to direct nerve or root infiltration. Moreover, in our case the subsequent improvement of the neuropathy following partial gastrectomy argues against infiltration.

Five types of peripheral neuropathy have been described in association with lymphoma, distinguishable by their differing degrees of sensory and motor involvement and by their time course:¹ sensory neuropathy, acute sensorimotor neuropathy, subacute and chronic sensorimotor neuropathies, relapsing and remitting sensorimotor neuropathies, and subacute motor neuropathies.⁵ We believe this case to be of the subacute sensorimotor type. In previous reports such cases have always been relentlessly progressive and usually axonal in type and are thus distinguishable both from the relapsing neuropathies which are demyelinating in type and from the subacute motor neuropathies described by Schold *et al.*⁵ This last group was characterised by frequent spontaneous stabilisation and minor sensory involvement both clinically and at necropsy.

Although the early electrophysiological studies demonstrated a predominantly motor neuropathy, our case is distinguished from those of Schold *et al* by the severity of the eventual clinical sensory loss and by the very severe axonal loss in the biopsy of the sural nerve. Furthermore, in this case the neuropathy did not merely stabilise but resolved dramatically. This occurred after the removal of the lymphoma and although proof of a causal relationship is lacking, the timing between improvement of the neuropathy and the resection of the lymphoma is compelling. Furthermore, the patient has not relapsed in over eleven months of observation. There have been no previously reported cases of a paraneoplastic lymphomatous neuropathy resolving with treatment of the tumour, although this has been noted without such precise timing in neuropathies associated with bronchial carcinomas.³ The marked improvement in our case was all the more surprising in view of the severe axonal loss on sural nerve biopsy. Presumably, in other nerves axonal loss could not have been so severe or alternatively their function was impaired by an element of demyelination, though conduction velocities were only slightly reduced.

The stomach is the commonest extranodal site for non-Hodgkin's lymphoma and resection with or without radiotherapy may be curative. Given the gratifying response of this patient's neuropathy to treatment of her localised lymphoma we suggest that investigation in such cases be extended to the

gastrointestinal tract even in the absence of localising symptoms or signs.

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Dysphagia due to a pharyngeal mucocoele mimicking myasthenia

Paranasal mucocoeles on occasion may cause neurological deficits.^{1,2} By contrast, mucocoeles of the oropharynx and trachea rarely produce neurological complications.³ We report a case of progressive dysphagia in a myasthenic patient caused by an aryepiglottic mucocoele.

A 29 year old right handed black woman with congenital myasthenia gravis, and a history of depression, presented to the emergency department two days after developing suicidal thoughts and abruptly discontinuing her medication which included pyridostigmine. In addition to her psychiatric symptoms, she complained of headaches, fatigue, diplopia, dysarthria and dysphagia. The dif-

ficulty she had in swallowing was progressive over several weeks, equal for solids and liquids, and accompanied by the sensation of a painless "lump in the throat". Although she had experienced swallowing difficulties in the past related to myasthenia, her current dysphagia was qualitatively different, although she could not elaborate further. Her history was significant with a thymectomy at the age of nine and numerous admissions for chronic upper respiratory infections and myasthenia. She had a long history of depression and a personality disorder.

The head and neck examination was normal without evidence of masses. Neurological examination revealed bilateral ptosis and dysarthria. The oropharynx was clear, the gag reflex was intact and she swallowed water without difficulty. Volitional motor testing was unreliable. The rest of the neurological examination was normal. An MRI of the head demonstrated a mass in the hypopharynx (fig). A left ary-epiglottic cyst was removed by direct laryngoscopy which proved to be a mucocoele on microscopic sections. Post operatively, the dysphagia resolved and the myasthenia was controlled with pyridostigmine.

A mucocoele is a mucus gland retention cyst which typically arises in the cranial sinuses, although other head and neck structures may be involved. Predisposing factors for the development of a mucocoele include trauma, structural abnormalities, and chronic infections.⁴ Neurological complications may arise depending on the location of the mucocoele. Paranasal mucocoeles may cause a variety of cranial neuritides¹ and suprasellar extension may mimic a pituitary adenoma.² By contrast, upper respiratory tract mucocoeles may cause airway compromise, but neurological manifestations are distinctly rare.³

A clinician may not be overtly concerned with a depressed patient complaining of a "lump in the throat" or a myasthenic patient experiencing bulbar symptoms while off anticholinesterase medications. This case illustrates the difficulty inherent in the psychiatric patient with an organic complaint which may be further confounded by an established diagnosis. Mucocoele should be included in the differential diagnosis of the

predisposed patient who develops cranial neurological symptoms.

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Trazodone is ineffective in essential tremor

The pathophysiology of essential tremor (ET) is not known. No gross structural changes in the nervous system have been found in ET,¹ so it is presumed that it results from an abnormality in some aspect of neuronal function, for example, changes in transmitter levels. In a single study using positron emission tomography, increased glucose metabolism was found in the inferior olive in patients with ET.² It has been suggested that rhythmic activity of neurons in the inferior olive may play a role in the generation of symptomatic tremor in humans.³ Olivary neurons receive a serotonergic input,⁴ and a recent paper reported a good result from the serotonin agonist trazodone hydrochloride (Molipaxin), 150 mg/day, in two patients with ET.⁵ The study was not, however, placebo controlled and no objective quantitation of tremor was used. We therefore carried out a double-blind, placebo controlled trial of trazodone in ET.

Fourteen patients with ET, not taking any form of medication, were entered into the study. Nine patients (mean age 60 years, range 41-74 years) completed the study and five withdrew for personal reasons. Patients received, in random order, trazodone 50 mg twice daily for one week followed by 50 mg three times a day for three weeks or a matching placebo. At the end of each treatment period, tremor was assessed in the laboratory 1.5 hours after the morning dose. Postural tremor of the hands (arms supported to the wrists with hands held horizontally in pronated posture) was evaluated by 1) accelerometry, according to a method previously described,⁶ 2) clinical rating on a scale from 0 (no visible tremor) to 5 (gross, self-injuring tremor), 3) patients' self-rating from +5 (large increase in tremor), through 0 (no change), to -5 (large reduction in tremor), 4) manual performance test (spiral tracing) scored for accuracy by three assessors on a scale from 0 (smooth tracing, no errors) to 5 (very tremulous tracing, many errors).

Results showed no statistically significant differences between trazodone and placebo on any of the measures used. Median tremor magnitude (in milli-g, where $g = 981 \text{ cm/s}^2$) was 23.4 (placebo), 29.7 (trazodone); mean scores for clinical rating (placebo/trazodone respectively) were 2.3/2.3; self rating 0.2/0.0;

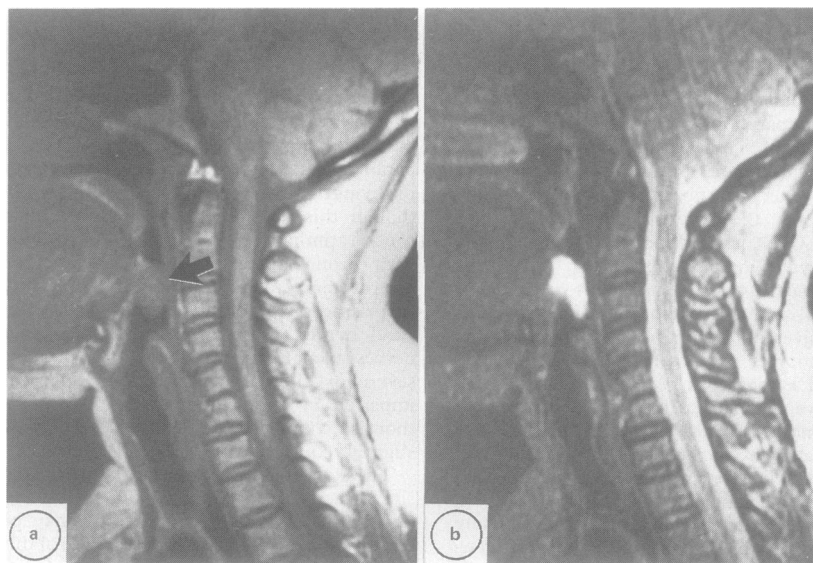


Fig Magnetic resonance image of the head. A T1 weighted image reveals a mass in the hypopharynx (arrow). B The mass demonstrates increased signal intensity on a T2 weighted image.