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

Lymphoma may cause a peripheral neuropathy by nerve infiltration or by non-metastatic effects and occasionally this may be the presenting feature. 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 (13 lb), but had no other symptoms. General and cranial nerve examinations were normal. There was generalised muscle wasting in the limbs, particularly the distal calf muscles, and there was hypotonia and symmetrical proximal and distal weakness of all limbs (MRC grade 3 or 4). All tendon reflexes were absent with plantar responses flexor.

At this stage the patient's condition was unaided. She was having no difficulty walking and, as a result of her improved mobility, she was able to return to her own home. She was able to carry out all her daily activities without assistance and was in general very well. She maintained the weight that she had regained following the initial period of weakness.

She was observed over a period of six months and during this time she made a full recovery. She remained asymptomatic and no further examination revealed any signs of neuropathy.

The possibility arises that similar structural changes occurred in our patient, and that the symptoms described in this patient emerged as a consequence of a localised gastric lymphoma, which was of the B-cell type.

The confirmation of a localised gastric lymphoma, which was of the B-cell type, in our patient is important as it suggests that the development of a peripheral neuropathy may be related to the presence of a localised gastric lymphoma. The importance of this finding is supported by the fact that the lymphoma was confirmed by histological examination of the resected tissue.
Dysphagia due to a pharyngeal mucocoele mimicking myasthenia

Paranasal mucocoeles on occasion may cause neurological deficits.1 By contrast, mucocoeles of the oropharynx and trachea rarely produce neurological complications.1 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 difficulty she had in swallowing was progressive over several weeks, equal for solids and liquids, and accompanied by the sensation of a "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 for 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.1 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.3 By contrast, upper respiratory tract mucocoeles may cause airway compromise, but neurological manifestations are distinctly rare.4

A clinician may not be overly 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.

**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.