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

TP ENEVOLDSON,
JA BALL,
JM MCGREGOR,
Department of Neurology,
St Thomas' Hospital,
London SE1 7EH, United Kingdom

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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 difficulty 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 orophanynx 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 neuritides[1] 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.

SYLVAIN S JUNGER, ROBERT B WRIGHT,
Rush-Presbyterian-St Luke's Medical Center,
1653 West Congress Parkway, Chicago,
Illinois 60612, United States

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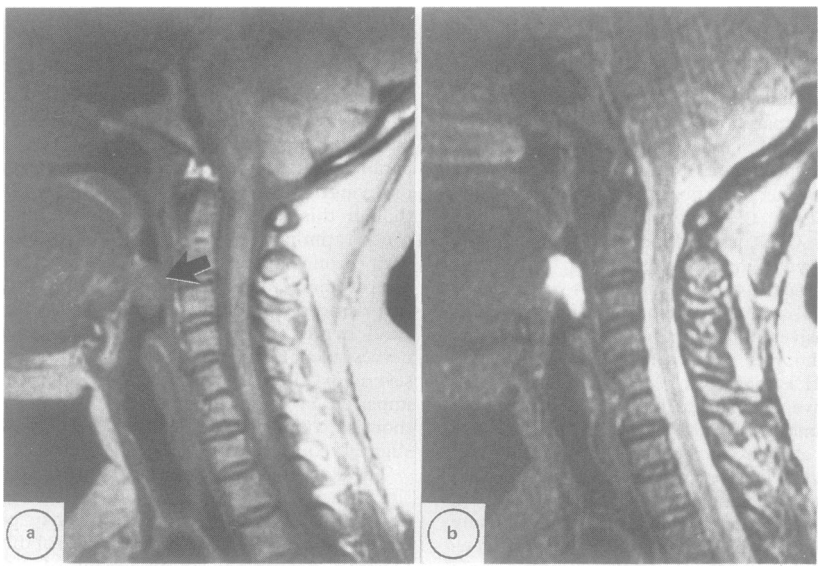

Fig Magnetic resonance image of the head. A T1 weighted image reveals a mass in the hypopharynx (arrow). B The mass demonstrates increased signal intensity on a T2 weighted image.

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 rhythmical 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 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;

performance rating 3·2/3·0. Reported side effects on trazodone were tiredness (4 patients), constipation (1 patient) and drowsiness for the placebo (1 patient).

Due to the high drop out rate, the final number of patients in the study group was smaller than the minimum number we have recommended for clinical trials in essential tremor.[7] However, our data were highly consistent for all patients and are similar to those of Koller[8] who found there were no effects of trazodone in a similar group of 10 patients studied in the United States.

Despite an earlier optimistic report,[5] these findings indicate that trazodone is ineffective in the control of ET. It seems unlikely therefore that serotonergic transmission is directly involved in the genesis of ET. Drugs increasing dopamine, acetylcholine and GABA transmission have been similarly ineffective.[9] Further neuropharmacological probes may be helpful in exploring the origin of ET. However, it would seem unlikely that a deficiency in a single neurotransmitter system represents the primary pathology in ET.

LYNN CLEEVES.
LESLIE J FINDLEY
MRC Neuro-Otology Unit,
National Hospital for Nervous Diseases,
Queen Square,
London WC1E 3BG.

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Low prevalence of HTLV-1 antibodies in the serum of patients with tropical spastic paraplegia from the Ivory Coast

A high prevalence of human T—lymphotropic virus type I (HTLV-1) antibodies has been reported in the serum of patients with tropical spastic paraplegia (TSP), from various parts of the world. This disease is also present in West Africa where, as in other tropical regions, no obvious aetiology was discovered.

To discover the possible link between TSP and HTLV-1 antibodies in West Africa, the serum of 20 patients from the Ivory Coast was collected. All patients fulfilled the clinical diagnostic criteria previously described for TSP.[1] Other causes of paraparesis were excluded by clinical and paraclinical investigations. The presence of HTLV-1, HIV-1 and HIV-II antibodies was determined by ELISA and western-blot methods. The results are summarised in the table. One patient was positive only for HTLV-1, two patients were positive for HTLV-1 and HIV-II, one was positive for HIV-II and one was positive for HIV-I and HIV-II. High HTLV-1 antibody titres were found ranging from 1/5000 to 1/10 000.

Table Results of HTLV-1, HIV-I and HIV-II antibodies in the serum of 20 TSP patients from the Ivory Coast (ELISA and western-blot methods)

	HTLV-1	HIV-I	HIV-II
Patients 1 to 15	−	−	−
Patient 16	+	−	−
Patient 17	+	−	+
Patient 18	+	−	+
Patient 19	−	+	+
Patient 20	−	−	+

The total prevalence of HTLV-1 positivity among TSP patients was 15%. This observation contrasts with the high prevalence of HTLV-1 positivity reported in the other tropical regions, particularly in the Seychelles (85%)[1] and in Martinique (59%).[2]

The seroprevalence of HTLV-1 among healthy controls in the Ivory Coast (1·6%)[3] is similar to that observed in the French West Indies (2%).[4]

These findings confirm that in West Africa antibodies against retroviruses other than HTLV-1 are present in the serum of patients with TSP, as we reported previously.[5] The lack of HTLV-1, HIV-I and HIV-II antibodies in 75% of patients with TSP indicate that different aetiologies may be linked to TSP in West Africa, such as other viruses, toxins or malnutrition.

Our results underline the need for a larger study of TSP in Africa to evaluate the role of various aetiological factors with the geographical distribution of the disease throughout the continent.

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J HUGON,*
JM VALLAT,*
M DUMAS,*
M VERDIER,
F DENIS,†
F AKANI,‡
YF BOA,‡
C GIORDANO‡
*Institute of Tropical Neurology and Department of Neurology,** *Faculté de Médecine, Limoges, Department of Bacteriology and Virology,† Hôpital Universitaire Dupuytren, Limoges, France*
Department of Neurology,‡ CHU d'Abidjan, Ivory Coast

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Wegener's granulomatosis presenting as peripheral neuropathy: diagnosis confirmed by serum anti-neutrophil antibodies

The case described by Kirker, Keane and Hutchinson[1] illustrates that neuropathy may occur early in Wegener's granulomatosis in the absence of more classical pulmonary and renal findings, leading to delay in diagnosis. Even if the diagnosis is suspected, histopathological confirmation may be difficult. We describe a patient presenting with rapidly progressive peripheral neuropathy and inconclusive biopsy findings for whom the demonstration of serum autoantibodies to neutrophil cytoplasm antigen (ANCA) permitted early diagnosis and therapy.

A 57 year old man developed rapidly progressive numbness and weakness of both hands and the right leg over a period of 10 weeks. He had right wrist and foot drop and marked wasting of both hands. Right elbow, wrist, hand, ankle and foot movements were MRC grade 2–3; limb power elsewhere was grade 4. Fine touch and pinprick sensation were diminished over both hands, right foot and the lateral aspect of the right leg; other sensation was unimpaired. He had a vesiculopapular rash on his elbows and hands but no other abnormality, and urinalysis was negative.

Urea, electrolytes, haemoglobin and chest radiograph were normal. White cell count was 19·9 × 10^9/l, 74% neutrophils, ESR 38 mm/l hour, and serum C-reactive protein 45 mg/l. Serum alkaline phosphatase was 1031 U/l, alanine transaminase 211 U/l, and aspartate transaminase 73 U/l. Urinary creatinine clearance was moderately reduced at 58 ml/min. Rose-Waaler was positive (1 : 1280). Serum B12, blood lead, abdominal ultrasound scan and CSF were normal. Screening for porphyria, hepatitis viruses and antinuclear and antimitochondrial antibodies were negative, digital renal arteriography showed no aneurysms, and the only abnormality on CT scanning of the skull, thorax and abdomen was fluid in both maxillary sinuses. ENT assessment revealed a granular mass on the right postnasal space. Biopsies of this area and of the skin lesions showed non-specific chronic inflammation. Despite unhelpful histopathological findings, Wegener's granulomatosis was suspected and the presence of serum ANCA was confirmed on indirect immunofluorescence (titre 1 : 80).

The patient was started on cyclophosphamide 2 mg/kg and a reducing dose of prednisolone. Six months later right wrist extension and finger movements were grade 3; limb power was otherwise normal. Sensory impairment was limited to the fingertips and the right sole. ESR, C-reactive protein, white cell count and creatinine clearance were normal, Rose-Waaler negative, and ANCA not detectable.

Histopathological confirmation of Wegener's granulomatosis may be difficult. Parlevliet *et al*[2] obtained a firm histological diagnosis in only 2 of 11 patients with typical symptoms and signs of the disease. While a presumptive diagnosis is acceptable when the presentation is typical, the decision to start cytotoxic therapy with its attendant morbidity is difficult in atypical cases.

Savage *et al*[3] showed that ANCA was not present in a small series of patients with polyarteritis nodosa and Churg-Strauss syndrome, but was highly sensitive and specific for Wegener's granulomatosis and micros-