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

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Dysphagia due to a pharyngeal mucocoele mimicking myasthenia

Paranasal mucocœles on occasion may cause neurological deficits.1 By contrast, mucocœles of the oropharynx and trachea rarely produce neurological complications.2 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 pyridostig- 

mine. In addition to her psychiatric symp- 

toms, 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 dys-

phagia 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 nor- 

mal 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. Vocal 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 aryepiglottic 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 pyridostig- 

mine.

A mucocoele is a mucus gland retention cyst which typically arises in the cranial sinuses, although other head and neck struc- 

tures may be involved. Predisposing factors for the development of a mucocoele include trauma, structural abnormalities, and chronic infections.3 Neurological complications may arise depending on the location of the mucocoele. Paranasal mucocœles may cause a variety of cranial neuritides and suprasellar extension may mimic a pituitary adenoma.4 By contrast, upper respiratory tract mucocœles may cause airway compromise, but neurological manifestations are distinctly rare.5

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 anti- 

cholinesterase medications. This case illus- 

trates the difficulty inherent in the psy- 

chiatric 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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Trizadone 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 for ET,1 so it is presumed that it results from an abnormality in some aspect of neuronal func- 

tion, for example, changes in transmitter levels. In a single study using positron emission tomography, increased glucose metabo- 

lism was found in the inferior olive in patients with ET.2 It has been suggested that rhythm- 

ical activity of neurons in the inferior olive may play a role in the generation of symp- 

tomatic tremor in humans.3 Olivary neurons receive a serotonergic input,4 and a recent paper reported a good result from the serotonin agonist trazadone hydrochloride (Molipaxin) 150 mg/day, in two patients with ET.1 The study was not, however, placebo controlled and no objective quantita- 

tion of tremor was used. We therefore carried out a double-blind, placebo controlled trial of trazadone 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, trazadone 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 pron- 

ated posture) was evaluated by 1) acceler- 

ometry, according to a method previously described,2 2) clinical rating on a scale from 0 (no visible tremor) to 5 (gross, self-injuring tremor), 3) patients’ self-rating from 0+ (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 trazadone and placebo on any of the measures used. Median tremor magnitude (in millig, where g = 981 cm^2/s^2) was 23-4 (placebo), 29-7 (trazadone); mean scores for clinical rating (placebo/trazadone 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.