

Dysphagia as a primary manifestation of thyrotoxicosis: A case report

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Summary

This report deals with a middle aged man in whom the presenting symptom of the disorder was dysphagia. The clinical approach to the final diagnosis of thyrotoxic myopathy causing dysphagia is outlined and the pathophysiology of dysphagia is then discussed. The need to include thyrotoxicosis in the differential diagnosis of an otherwise unexplained case of dysphagia is stressed.

Key words: Thyrotoxicosis, myopathy, dysphagia.

Case Report

A 51-year old man was admitted to the hospital in January, 1988 for evaluation of dysphagia. The patient, a rubber estate supervisor, complained of progressive loss of appetite and difficulty in swallowing during the preceding three months. He complained of food getting stuck whenever he ate. The sensation was felt with both liquids and solids in the lower midline portion of his neck and was frequently associated with vomiting. He had lost 23 kg. in weight over the preceding six months. Later, he admitted having had difficulty in getting up from squatting position without support, climbing stairs and combing his hair.

The pertinent past history included one of recurrent episodes of epigastric pain with occasional vomiting and diarrhoea. He was admitted for these complaints several times to different hospitals and underwent upper endoscopic examinations on four occasions; no causative lesion was observed.

Physical examination revealed an ill looking, cachexic and an apathetic man with no interest in his surroundings. (Fig. 1). The pulse rate was 136 per minute and regular. His blood pressure was 160/100 mm of hg. He had no lymphadenopathy and the thyroid gland was normal to palpation with no bruit. There was bilateral symmetrical weakness of neck and proximal muscle weakness in both upper and lower limbs. The deep reflexes were brisk with normal plantar response. There was no sensory impairment and he had no cerebellar signs.

Full blood count, E.S.R., blood sugar, urea, electrolytes, urinalysis, chest x-ray and plain x-ray of the abdomen were normal. Liver function tests revealed a total bilirubin of 34 micromol/L (normal 3–18.0 micromol/L), serum albumin 26 G/L (normal 32–82 G/L) and a mildly raised alkaline phosphatase at 16.5 K.A. units/100 ml. (normal 3–13 K.A. units/100 ml.) The corrected calcium was mildly raised at 2.8 mmol/L (normal 2.2–2.6 mmol/L) with a normal inorganic phosphate level at 1.28 mmol/L.

Meanwhile his serum thyroxine (T₄) and tri-iodothyroxine (T₃) levels were reported in the



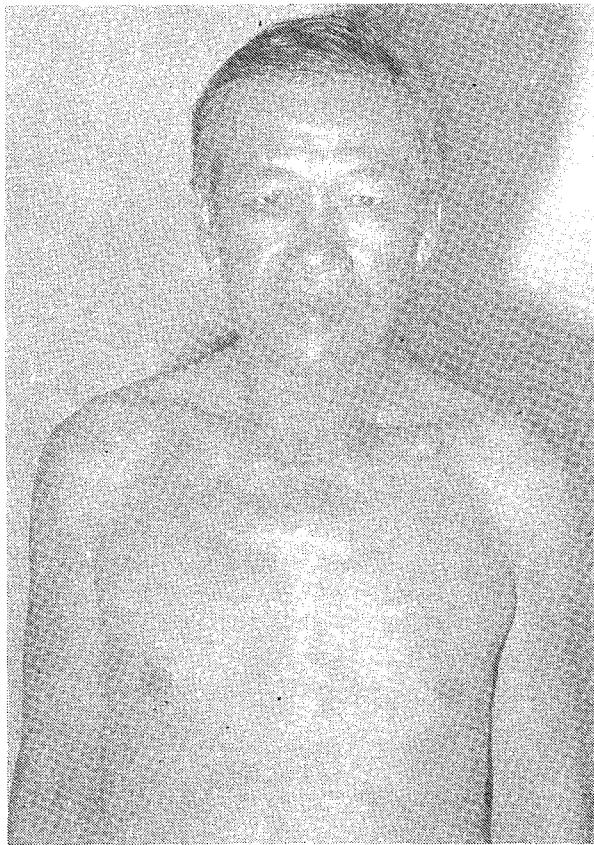
hyperthyroid range at 277 nanomol/L (normal 64.4 to 148 nanomol/L) and 4.6 nmol/L (normal: 1.2–3.4 nmol/L) respectively.

An upper endoscopic examination and a barium meal follow through study did not show any structural abnormality to account for his symptoms. An ultrasound examination of the abdomen too was normal. The tensilon test was negative.

The patient was started on propylthiouracil 150 mg. t.d.s. and propranolol 40 mg. b.d. He responded dramatically to the medications and became fully alert within a week. His symptoms cleared rapidly as he returned to euthyroid status. He gained 32 kg. in six months (Fig. II). His serum calcium as well as T_3 and T_4 levels too fell within normal limits. He remains well nine months later.

Discussion

The causes of dysphagia broadly fall into two categories: obstructive and motility disorders.² The normal endoscopic and barium meal examinations virtually excluded an obstructive lesion in this patient. This made a motility disorder a strong possibility in him. Moreover, the 'liquid dysphagia' in him also favoured a neuromuscular basis for his complaints.² Unfortunately, motility studies could not be undertaken for want of facilities.



The common motility disorders causing dysphagia are shown in Table I. The patient did not exhibit any of the peripheral manifestations of the disorders shown in the table, to warrant them to be included in the differential diagnoses. The negative tensilon test with the paucity of other symptoms made myasthenia gravis unlikely. The normal blood sugar and the absence of long standing diabetes excluded diabetic gastroparesis as a possible explanation in him.

Thus, thyrotoxic myopathy presenting with dysphagia was thought to be the most likely diagnosis in him. This was subsequently confirmed by the T_3 and T_4 levels. Thyrotoxicosis presenting with dysphagia alone is rare. In retrospect, he appears to have had a number of clues to point towards hyperthyroidism in him. These features include: sinus tachycardia, wide pulse pressure, proximal muscle weakness and brisk reflexes. Loss of appetite experienced by this patient is a noted feature of the apathetic variant of the disorder.^{3,4} His recurrent episodes of epigastric pain simulating a peptic ulcer syndrome is also a recognised feature of thyrotoxicosis.³

The mild hypercalcaemia, raised alkaline phosphatase level and hyperbilirubinemia seen in this patient, are also consistent with thyrotoxicosis.⁴ They reverted to normal levels when the patient became euthyroid with treatment. The acid test in the patient was however the therapeutic challenge with antithyroid medications to which he responded promptly.

The mechanism of disordered oesophageal motility in thyrotoxicosis is not known. The postulates include hypercalcaemia and hypomagnesaemia. Hypercalcaemia is thought to cause dysphagia by its effect on neuromuscular release at the neuromuscular junction.¹

Table I²*

Motility disorders causing dysphagia

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- 1.) **Oropharyngeal.**
Cerebrovascular accident.
Myasthenia gravis.
Polymyositis.
Dermatomyositis.
Myopathies.
Bulbar.
Poliomyelitis.
Myotonic.
Dystrophica.

 - 2.) **Oesophageal.**
Achalasia.
Diffuse oesophageal.
Spasm.
Collagen vascular disease.
Alcoholism.
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* Modified.

Low levels of magnesium which increase with treatment has been found in thyrotoxicosis. It is felt hypomagnesaemia interferes with upper gastrointestinal motility by its direct action on its autonomic innervation. It has also been suggested that vomiting in thyrotoxicosis is possibly due to the stimulation of the chemoreceptor trigger zone by excess thyroxine hormones.⁵

Whatever the pathophysiology, it is necessary to recognise the possibility of thyrotoxicosis causing dysphagia as it is easily amenable to treatment. If left unrecognised, it might lead to recurrent episodes of aspiration pneumonia⁶ in addition to the usual sequelae of untreated hyperthyroidism.

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