

## Case Report

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## Thyrotoxicosis: A Rare Cause of Dysphagia

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## ABSTRACT

Excess and deficiency of thyroid hormones can have a variety of gastrointestinal manifestations ranging from heartburn, dysphagia, vomiting, diarrhea to constipation. We present a case of chronic thyrotoxic myopathy presenting as generalized weakness with bulbar involvement. He made a complete recovery with antithyroid therapy.

**Keywords:** *Thyroid disorders, dysphagia, esophageal dysmotility*

## INTRODUCTION

Thyroid hormone mediates metabolism in almost all organs of the body including gastrointestinal viscera. Thyroid disorders can have a variety of digestive manifestations ranging from heartburn, dysphagia, vomiting, diarrhea to constipation. Hypothyroidism related gastrointestinal manifestations are widely reported in literature. Pathophysiology has been postulated as accumulation of glycosaminoglycans; mainly hyaluronic acid; in soft tissues followed by interstitial edema (1). However, hyperthyroidism related intestinal motility disorders have not been well described in literature (2,3). Altered neurohumoral regulation and electrolyte imbalance are thought to be the pathology, but the complex phenomenon is not completely elucidated.

## CASE REPORT

We report a case of a 69-year-old previously healthy farmer, who was presented with a 5-month history of difficulty in swallowing liquids, followed by dysphagia to both solid and liquid food. He gave a history of 14kg weight loss despite increased appetite. He noticed intermittent nasal regurgitation of liquid, but denied any diplopia, dysarthria or limb weakness. He has noticed a generalized weakness with significant difficulty in combing hair and standing up from squatting position. There was no history of diarrhea, vomiting, fever, melaena, alcohol or drug abuse. He was not on any medications.

On general examination, the patient was cachectic with a BMI of 16kgm<sup>-2</sup>. He was pale. He had no proptosis or signs of dysthyroid eye disease. A fine tremor on outstretched hands was noted. He had no goiter or lymphadenopathy. He was tachycardic with a pulse rate of 110 / bpm and a blood pressure



of 130/80mmHg. The upper-limb and lower-limb girdle muscles were wasted with no tenderness. The muscle power of proximal muscle groups of both upper and lower limbs was 3/5 MRC grading. The knee and biceps reflexes were diminished with preserved ankle and supinator jerks. Palatal movements were normal bilaterally. There was no sensory involvement. The Cerebellar examination was normal.

His hemoglobin was 9.6mg/dl with a MCV of 82fl. Serum sodium was 134 mmol/l and serum potassium were 3.2 mmol/l. Renal and liver function tests were normal. ECG showed sinus tachycardia. Serum TSH was 0.05 u/L (0.35- 5), free T4 was 30 ng/dl (0.7-1.53), free T3 was 14 ng/dl (0.2-0.5). Serum calcium and magnesium levels were normal. Serum cortisol was normal. USS neck showed an ill-defined nodule suggestive of multinodular goiter without compression on oesophagus. Thyroid peroxidase antibody and thyroid stimulating hormone receptor antibody was negative. A barium swallow revealed oropharyngeal dysphagia. Upper gastrointestinal endoscopy failed to demonstrate any intra-luminal abnormality. There was no extra-luminal compression or thymoma in contrast enhanced CT imaging of the neck and chest. Repetitive nerve stimulation in bulbar muscles did not show a decrementing pattern. Acetylcholine antibodies were negative. Electromyography of right quadriceps revealed myopathic pattern. However, creatinine phosphokinase levels remained normal. Oesophageal manometry and motility studies were deferred due to unavailability.

The electrolyte imbalances were corrected by intravenous potassium replacement. Despite normalization of potassium the dysphagia and proximal muscle weakness persisted. A clinical diagnosis of hyperthyroidism associated oesophageal motility disorder was considered and he was started on carbimazole and beta blockers. One week into therapy the patient clinically improved and was tolerating liquid diet. One month into therapy, symptoms completely resolved, patient started to gain weight and returned to work.

## DISCUSSION

Dysphagia is a rare gastrointestinal manifestation of thyrotoxicosis. It can have an acute or chronic presentation, the latter being more common (4). It can be classified as mechanical or dysmotility related. Direct compression from an enlarged thyroid on oesophagus leads to progressive mechanical dysphagia. An imbalance of electrolytes and alteration of complex neurohumoral mechanisms are described as pathophysiology of dysmotility related dysphagia in thyrotoxicosis. Hypokalemic periodic paralysis, thyrotoxic periodic paralysis, thyrotoxicosis related hypercalcemia and hypomagnesemia, Graves's associated myasthenia gravis are a few accepted mechanisms of dysmotility.

A case series describes chronic thyrotoxic myopathy presenting as generalized weakness with bulbar involvement in 16.4% as in our case (5). Although both oropharyngeal and esophageal dysphagia were reported with thyrotoxicosis, oropharyngeal dysphagia was more common and was associated with more aspiration pneumonia (3). There was no significant difference between the sexes as to the groups of muscles involved in the myopathic process. Electromyography findings of thyrotoxic myopathy include a reduction in mean action potential duration, low voltage of the motor unit potentials, and an increased incidence of polyphasic potentials (6). Our patient had EMG finding compatible with chronic thyrotoxic myopathy.

The incidence of thyrotoxicosis preceding, accompanying or following the clinical onset of myasthenia gravis is 5% with female preponderance (5). Our patient did not fulfill clinical or laboratory criteria of myasthenia gravis. Okinaka et al described 1.9% incidence of periodic paralysis in their thyrotoxic group (7). Among them only 1 patient out of 199 had a family history of periodic paralysis. We failed to establish a family history or a past history suggestive of periodic paralysis in our patient.

Oesophageal manometry and motility studies are the gold standard for diagnosis for dysmotility disorders (3). However, due to unavailability we proceeded with treatment without motility studies.

Treatment with beta blockage alone and combination of beta blockage and carbimazole rapidly reverse neuromuscular manifestations (8). Failure to detect thyrotoxicosis as a cause for completely treatable form of bulbar weakness can lead to recurrent regurgitation, aspiration pneumonia and even death.

## CONCLUSION

Excess and deficiency of thyroid hormones can have a variety of gastrointestinal manifestations. This case highlights the importance of recognizing thyrotoxicosis as a cause of unexplained dysphagia.

### Author declaration

#### Authors' contributions:

Drafting of the manuscript: A.M.B.D.A; Study supervision: W.W.L.A.J.

#### Conflicts of interest:

The authors declare that there is no financial or non-financial conflict of interest.

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#### Statement on data availability:

All data generated during this study are included in this published article.

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