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R Alwithenani and others

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Myopathic dysphagia caused by thyrotoxicosis: a case report and review of the literature

Raad Alwithenani[1,2,3,4], **Danielle M Andrade**[5], **Lingxin Zhang**[6,7] and **Karen E Gomez-Hernandez**[1]

[1]Department of Medicine, Division of Endocrinology and Metabolism, University of Toronto, Toronto, Ontario, Canada, [2]College of Medicine, King Saud Bin Abdulaziz University for Health Sciences, Jeddah, Saudi Arabia, [3]King Abdullah International Medical Research Center, Jeddah, Saudi Arabia, [4]Department of Medicine, King Abdulaziz Medical City, Ministry of National Guard Health Affairs, Jeddah, Saudi Arabia, [5]Department of Medicine, Division of Neurology, University of Toronto, Toronto, Ontario, Canada, [6]Department of Laboratory Medicine and Pathobiology, University of Toronto, Toronto, Canada, and [7]Department of Pathology and Laboratory Medicine, Mount Sinai Hospital, Toronto, Ontario, Canada

Correspondence should be addressed to R Alwithenani
Email
alwithenanira@mngha.med.sa

Summary

Myopathy caused by thyrotoxicosis is not uncommon. Skeletal muscles are commonly involved, but dysphagia is a rare manifestation of thyrotoxicosis. We aim to raise awareness of dysphagia caused by hyperthyroidism and review similar cases in the literature. We present a case of severe dysphagia caused by hyperthyroidism. We also summarize similar case reports in the literature. Our patient is a 77-year-old man who presented with thyrotoxicosis related to Graves' disease (GD), dysphagia to both liquid and solid food, and weight loss. Further investigations revealed severe esophageal dysphagia and a high risk for aspiration. He required the placement of a G-tube for feeding. After 8 weeks of methimazole treatment, his thyroid function normalized and his dysphagia improved significantly, leading to the removal of the feeding G-tube. We summarize 19 case reports published in the literature of hyperthyroidism leading to dysphagia. Patients with thyrotoxicosis and dysphagia are at higher risk for aspiration pneumonia and thyroid storm. Based on previous case reports, on average, approximately 3 weeks of treatment with anti-thyroidal drugs and beta-blockers is needed before patients can eat normally. We report a case of dysphagia associated with GD, which is rare and needs prompt recognition to restore euthyroid status. Dysphagia generally resolved with normalization of thyroid function.

Learning points

- Myopathy caused by thyrotoxicosis is not uncommon.
- Skeletal muscles are commonly involved, but dysphagia is a rare manifestation of thyrotoxicosis.
- Dysphagia due to hyperthyroidism resolves with normalization of thyroid function.
- Early recognition of dysphagia related to hyperthyroidism and early initiation of therapy may help reverse the dysphagia and prevent complications.

Background

Myopathy caused by thyrotoxicosis is not uncommon. Skeletal muscles are commonly involved, but dysphagia is a rare manifestation of thyrotoxicosis (1). We report a case of a patient with Graves' disease (GD) who presented with thyrotoxicosis and dysphagia. His dysphagia resolved once he became euthyroid a few weeks after commencing antithyroidal drugs. We also summarize 19 case reports of thyrotoxic dysphagia that different authors published over the past 50 years.

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Case presentation

A 77-year-old man with a history of hypertension, type 2 diabetes, chronic kidney disease, psoriasis, psoriatic arthritis, and ankylosing spondylitis presented to the hospital in 2017 with an 8-week history of typical symptoms of hyperthyroidism including weight loss of 15 kg, palpitations, excessive sweating, tremor, proximal muscle weakness, dysphonia, and severe progressive dysphagia. Two weeks prior to admission, he was diagnosed with hyperthyroidism, and methimazole had been commenced. On examination, he was alert, blood pressure: 111/76, heart rate: 101, temperature: 37°C, and Saturation: 97%. There were no signs of Graves' orbitopathy. Examination of the thyroid was complicated by the fact that the patient could not extend his neck due to cervical kyphosis, but a goiter was not easily palpable. The skin was warm and distal tremor was present. A complete neurological examination demonstrated no focal neurological deficit.

Investigations

Relevant initial laboratory investigations were consistent with hyperthyroidism due to GD (thyroid-stimulating hormone: <0.05 mIU/L (0.3–4.9), FT4: 44 pmol/L (9–22), FT3: 9.4 pmol/L (2.6–5.7), thyroid receptor antibodies: 38.4 IU/L (normal <1), white blood cell: 5.6, Hb: 125, platelet: 186, Na: 141 mmol/L, K: 4.0 mmol/L, Cr: 144 mmol/L, eGFR: 42, plasma glucose: 8 mmol/L).

A CT scan of the head and neck showed a mildly enlarged and heterogeneous left thyroid lobe and no evidence of an acute vascular event. An MRI of the brain was unremarkable. A Video Fluoroscopic Swallow Study showed severe esophageal dysphagia to both liquid and solid food. An upper gastrointestinal endoscopy showed that extrinsic protrusion at the level of pharyngoesophageal region subsequently correlated with an osteophyte at that level. Otherwise, the study was normal.

Treatment

Treatment with methimazole was continued and propranolol was started. Because he could no longer swallow, he required the placement of a G-tube for feeding. After about 8 weeks, his thyroid indices normalized. He was able to tolerate food by mouth and underwent a repeat Video Fluoroscopic Swallow Study, which showed significant improvement leading to the removal of the G-tube.

Further investigations showed a mildly enlarged thyroid gland on ultrasound, predominantly on the left side, with multiple nodules of up to 2.8 cm. A thyroid uptake and scan showed an increased 24-h iodine uptake at 53.1% with increased radiotracer uptake consistent with GD as well as a cold area corresponding to the largest nodule on the left lobe (Fig. 1). A fine needle aspiration of the cold nodule yielded benign cytology.

Outcome and follow-up

Hyperthyroidism was difficult to control with methimazole due to significant fluctuation of free thyroid hormone levels, even with minor adjustments in the methimazole dose. It was ultimately decided to treat him with radioactive iodine. An initial dose of 15 mCi was administered, but unfortunately he remained hyperthyroid. Therefore, he received a second dosage of 30 mCi 7 months later. Despite the second treatment, he remained hyperthyroid. Eventually, he underwent a total thyroidectomy which he tolerated well with no complications. The thyroidectomy specimen displayed histological evidence of residual diffuse hyperplasia of the follicular epithelium, consistent with GD (Fig. 2). Treatment effects were present, including increased colloid production, radiation-induced nuclear atypia, and focal fibrosis. There was no evidence of malignancy.

Discussion

Based on previous case reports (Table 1), the median age of patients who presented with dysphagia in the setting of hyperthyroidism was 65 years (mean: 60, S.D. ±16). The underlying cause of thyrotoxicosis was GD in 75% of the reported cases. Men account for 46% of the reported

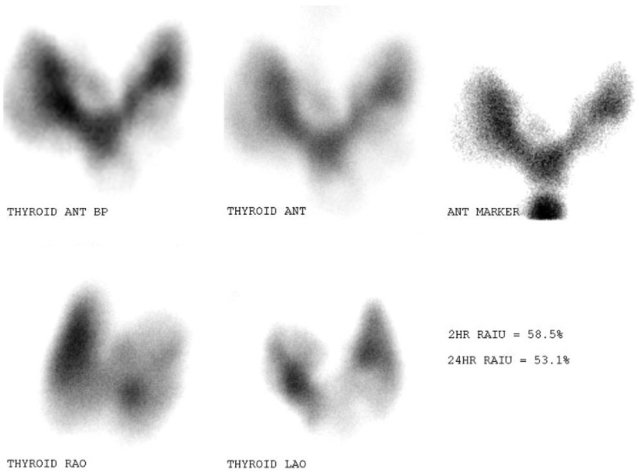

Figure 1
Thyroid uptake and scan show increased radiotracer uptake and inhomogeneous thyroid gland, consistent with history of Graves' disease with multiple nodules.

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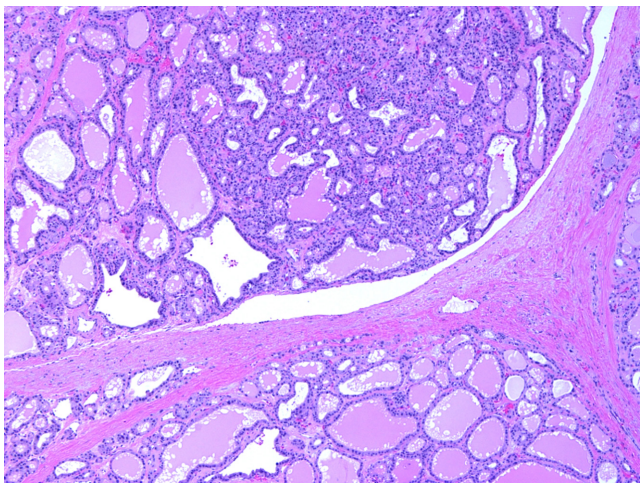

Figure 2
Histologically, the thyroid gland in Graves' disease has a lobulated architecture with accentuated fibrous septa. In areas with less prominent treatment effect, the pale, the follicles are irregularly shaped due to papillary infolding of the epithelium ('scalloping'), with formation of vacuoles along the pale, watery colloid.

cases of GD and dysphagia, which is more than expected in a disease that predominantly affects women. Thyroid storm was diagnosed in 21% and aspiration pneumonia in 31% of reported cases (Table 1). Both oropharyngeal and esophageal dysphagia were reported with thyrotoxicosis. Oropharyngeal dysphagia was more common and was associated with more aspiration pneumonia (3).

Different pathophysiological mechanisms have been suggested to explain the underlying etiology of dysphagia in patients with thyrotoxicosis. These include bulbar or esophageal myopathy, electrolyte abnormalities like hypokalemia, and mechanical cause by enlarged goiter (2). During the initial assessment of dysphagia in patients with thyrotoxicosis, the potential causes of dysphagia should be ruled out, such as a vascular etiology like stroke, neuromuscular causes like myasthenia gravis, electrolyte abnormalities like periodic hypokalemic paralysis, and mechanical causes. Indeed, myasthenia gravis was reported in 3–10 % of patients with thyrotoxicosis and dysphagia (1).

Patients with thyrotoxicosis and dysphagia are at higher risk for aspiration pneumonia and thyroid storm (3). Additionally, some patients with thyrotoxicosis and dysphagia will need temporary nutritional support through enteral feeding (1, 2, 3, 4). Based on previous case reports, on average, about 3 weeks of treatment with antithyroidal drugs and beta-blockers is needed before patients can eat normally (Table 1). Therefore, prompt recognition and treatment of thyrotoxicosis are vital. Indeed, some experts suggest treating thyrotoxicosis-related dysphagia as a thyroid storm to restore euthyroidism quickly and prevent the complications mentioned above (3).

In summary, we report a case of dysphagia associated with GD which is rare and need prompt recognition

Table 1 Summary of case reports of dysphagia and thyrotoxicosis.

Study	Patient's age, sex	Diagnosis	Treatment	Time to dysphagia resolution
Kammer *et al.* (4)	40, M;	GD	ATD+BB	4 weeks
	65, F;	GD	ATD+BB	3 weeks
	71, F;	GD	ATD	8 weeks
	28, F;	GD	ATD	5 days
Marks *et al.* (5)	56, F	Thyrotoxicosis*	ATD	4 weeks
Branski *et al.* (6)	69, F	Thyrotoxicosis*	ATD+BB	2 weeks
Sweatman *et al.* (7)	71, F	GD	ATD	Rapid recovery
Lleo *et al.* (8)	48, F	TMNG	ATD+BB	1 week
Noto *et al.* (2)	65, M	GD	ATD and BB	4 weeks
Garzon *et al.* (9)	82, M	TMNG	ATD	12 weeks
Chiu *et al.* (3)	50, M;	GD	ATD+BB+lugol's solution	8 weeks
	32, M;	GD	ATD+BB	2 weeks
	45, F	GD	ATD+BB+lugol's solution	3 days
Guldiken *et al.* (10)	70, F	GD	ATD	3 weeks
Okada *et al.* (1)	36, F	GD	ATD+BB+lugol's solution +steroid	8 weeks
Parperis *et al.* (11)	82, M	GD	ATD+BB	4 weeks
Baburaj *et al.* (12)	70, M	GD	ATD+BB	Not documented
Sukhdeo *et al.* (13)	73, F	GD	ATD+BB	3 days
Boddu *et al.* (14)	64, M	TMNG	ATD+BB+lugol's solution +steroid	4 days
Our case	77, M	GD	ATD+BB	8 weeks

*Authors did not specify the underlying cause of thyrotoxicosis.
ATD, antithyroidal drugs; BB, beta blockers; M, male; F, female; GD, Graves' disease; TMNG, toxic multinodular goiter.

to restore euthyroid status. Almost always dysphagia will resolve once thyroid functions normalized. The pathogenesis of thyrotoxic dysphagia is unknown, and more data are needed.

Declaration of interest
The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of the research reported.

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Patient consent
Written informed consent for publication of their clinical details and/or clinical images was obtained from the patient.

Author contribution statement
All authors were involved in the clinical care of the patient.

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