CASE REPORT

Hyperthyroidism Presenting as Dysphagia

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Abstract

A 65-year-old man presented with hyperthyroidism associated with thyrotoxic dysphagia. Treatment with thiamazole improved his symptoms promptly. Although dysphagia is a rare manifestation of thyrotoxicosis, it should be emphasized that the possibility of hyperthyroidism must be discussed in unexplained dysphagia because it is readily treatable.

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Key words: Graves’ disease, thyrotoxicosis, myopathy

Introduction

Dysphagia is an uncommon manifestation of thyrotoxic myopathy. Reports have been sparse and its incidence is not clear. We present a case of hyperthyroidism with dysphagia, which improved with the treatment for hyperthyroidism. We would like to emphasize that hyperthyroidism should be considered as a possible cause of unexplained dysphagia.

Case Report

A 65-year-old man with a history of hypertension presented with progressive dysphagia. He initially had difficulty swallowing liquids for one month, followed by dysphagia for both solids and liquids. He also complained of generalized weakness and weight loss of 3 kg over the three months prior to admission. He was not taking any medication.

On admission, he was alert and oriented, and appeared pale, cachectic, and diaphoretic. His vital signs were blood pressure 210/90 mmHg with no orthostatic change, pulse rate 130/min which was irregular, respiratory rate 18/min, and body temperature 37.2°C. Physical examination revealed diaphoresis, slightly enlarged soft thyroid, irregular heart rhythm, and fine intention tremor of the hands. Cranial nerves were intact. Gag reflex was present. There was generalized muscle weakness of 4/5 strength throughout. No exophthalmos, dysarthria, leg edema, or abnormal reflexes were noted and the remainder of the examination was within normal limits.

Laboratory data included a white cell count of 13,700/mm³ with normal differentiation, hemoglobin 12.5 g/dl, hematocrit 37.4%, platelet 158,000/mm³, potassium 5.2 mEq/l, calcium 10.2 mg/dl (normal: 8.4–9.7 mg/dl), albumin 3.3 g/dl (normal: 3.7–4.9 g/dl), alkali phosphatase 232 IU/l (normal: 60–201 IU/l), total cholesterol 110 mg/dl (normal: 129–232 mg/dl), creatine kinase 16 IU/l (normal: 55–210 IU/l), glucose 104 mg/dl, free thyroxine (T4) 6.99 ng/dl (normal: 0.91–1.82 ng/dl), free triiodothyronine (T3) 19.4 pg/ml (normal: 2.7–5.5 pg/ml), thyroid-stimulating hormone (TSH) 0.01 µU/ml (normal: 0.57–4.00 µU/ml), antithyroid stimulating antibody 419% (normal: <180%), antithyroid peroxidase antibody 7.8 U/ml (normal: <0.3 U/ml), and negative antithyroglobulin antibody. Electrocardiogram showed atrial fibrillation. The results of cardiac echo were within normal limits.

In the further work-up, computed tomography (CT) of the neck demonstrated no goiter and no mass compressing the esophagus, and upper endoscopy revealed no organic lesion or obstruction. The patient could not tolerate upper gastrointestinal series. Pharyngolaryngoscopically study showed diminished movements of the posterior pharynx, saliva pooling at the entry of the esophagus, and normal vocal movements. CT scan of the head was negative. Magnetic resonance imaging (MRI) study of the brain showed probable lacunar infarctions in the bilateral basal ganglia and periventricular area, but it did not reveal any lesion in the brain stem. The result of electromyogram was within normal limits. Acetylcholine receptor antibody was within the normal range and Tensilon test was negative.

Based on the above results, the patient was diagnosed as having Graves’ disease complicated with thyrotoxic myopathy and dysphagia, and he was started on thiamazole (30 mg per day) and propranolol (20 mg per day). He then developed aspiration pneumonia, for which intravenous antibiotics and hyperalimentation were started. Subsequently, dysphagia and muscle weakness began to ameliorate significantly as the thyroid function responded to the treatment (Table 1). Propranolol was discontinued when his blood pressure returned to normal.
and atrial fibrillation disappeared. Four weeks later, the patient became able to tolerate food and fluid intake, and intravenous thiamazole was tapered and switched to tablets (30 mg per day). Repeated upper gastrointestinal series and pharyngolaryngoscopic study showed normal pharyngeal and esophageal movements. The patient became ambulatory and was discharged on thiamazole (5 mg per day) after two months in the hospital.

**Discussion**

Muscle weakness and wasting secondary to myopathy are recognized complications of thyrotoxicosis. Skeletal muscles are predominantly affected, and dysphagia is rare (1–5). Possible neuromuscular causes of dysphagia in thyrotoxicosis include bulbar or esophageal myopathy, concomitant myasthenia gravis, and hypokalemic periodic paralysis. Mechanical compression by enlarged goiter also can cause dysphagia (6).

According to one report (7), 16% of the patients with thyrotoxicosis were found to develop bulbar muscle dysfunction. It is usually associated with chronic myopathy or thyroid crises and acute bulbar palsy without chronic thyrotoxic myopathy is extremely rare (7–9). The incidence of esophageal dysfunction in thyrotoxicosis is not clear (1, 6). Myasthenia gravis occurs in 0.35% of patients with hyperthyroidism, while 1–5% of the patients with myasthenia gravis develop hyperthyroidism (9, 10).

The present patient presented with hyperthyroidism of new onset, associated with dysphagia and generalized myopathy. Thyrotoxic bulbar myopathy was unlikely in light of the clinical course. Myasthenia gravis was ruled out based on the clinical manifestations and negative laboratory tests. Hypokalemic periodic paralysis was not likely because there had been no significant electrolyte disturbances, no episode of paralysis earlier in his life, and no family history. CT scan did not demonstrate an enlarged goiter. Therefore, it is most likely that this patient developed esophageal myopathy as part of generalized thyrotoxic myopathy. Prompt improvement in swallowing function after treatment with thiamazole for underlying thyrotoxicosis provides further support for this possibility. However, the pathophysiological mechanism of the impaired esophageal motility remained elusive.

On the other hand, he had multiple risk factors for stroke including age, hypertension, atrial fibrillation and probable lacunar infarctions in the past. Although MRI study of the brain stem was negative, this fact points to the possibility that he might have had the complication of a small bulbar infarction.

In summary, a case of hyperthyroidism presenting as dysphagia is described, which responded well to medical treatment. We conclude that this patient developed severe esophageal motility dysfunction secondary to thyrotoxicosis. It should be emphasized that possible hyperthyroidism must be discussed in a patient with unexplained dysphagia because it is readily treatable and it could result in a clinical outcome of serious importance if not treated.

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**References**


