Thyrotoxicosis Presenting as Acute Bulbar Palsy

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Abstract

Myopathy chiefly affecting the proximal muscles of the limbs is frequently seen in hyperthyroidism. But isolated acute bulbar palsy without skeletal muscle involvement is rare in thyrotoxicosis. We report the case of a 52 year old man who presented with severe dysphagia, dysphonia and bouts of aspiration. Laboratory testing revealed an underlying Graves’ thyrotoxicosis. His symptoms recovered dramatically within 6 weeks with treatment of hyperthyroidism. This case is reported to emphasize that thyrotoxicosis should be considered in the differential diagnosis of dysphagia of obscure etiology.

Introduction

Myopathy is frequently associated with thyrotoxicosis. Proximal skeletal muscles are predominantly involved but dysphagia and dysphonia are extremely rare.1 Bulbar involvement is considered a rare manifestation of thyrotoxicosis.2 The following case report is of a middle aged man with Graves’ disease presenting as acute bulbar palsy.

Case Report

A 52-year-old man presented with abrupt onset of dysphagia and dysphonia 6 weeks prior to admission. He had paroxysms of coughing and choking on swallowing both solids and liquids. His voice was barely a whisper. There was no diurnal variation in his symptoms. In addition, he had lost 10 kg weight over the last 3 months.

On examination, he was emaciated and irritable. He had no goiter. General examination was otherwise normal. On CNS examination, he had bilateral absent gag reflex and reduced movements of the palate. His deep tendon reflexes were brisk but plantar reflexes were flexor. Other systems were normal. ENT examination revealed pooling of secretions in the valleculae and pyriform fossae. Possibilities considered were (a) malignancy of the hypopharynx (b) brain stem demyelination.

On investigation complete blood counts, blood sugar, liver function tests, renal function tests, chest x-ray, MRI brain and upper gastrointestinal endoscopy were normal. In view of significant weight loss, thyroid function tests were done and the reports were free T4 9.78 (Normal range = 2.5 – 3.9 pg/ml), free T3 3.43 (Normal range = 0.6 – 1.12 ng/ml), TSH < 0.01 (Normal range = 0.34 – 5.6 Iu/ml), and Anti TPO 120.6 (Normal upto 50 IU/ml). Thyroid scan was suggestive of Graves’ thyrotoxicosis. Electromyography and repetitive nerve stimulation tests were normal.

Patient required nasogastric feeds. Beta blocker (propranolol hydrochloride 120mg in divided doses) and anti-thyroid drug (Neomercazole) were started. Patient also underwent radioiodine ablation. His voice improved significantly within one week. His dysphagia to solids improved in 2 weeks and his nasogastric tube could be removed in 6 weeks. During follow up, patient had complete recovery of dysphonia and dysphagia and gained 8 kg weight.

Discussion

Myopathy to some extent is virtually ubiquitous among thyrotoxic patients1,2 and 80% of thyrotoxicosis patients have neuromuscular symptoms.3 Weakness is primarily of the proximal limb muscles and associated bulbar involvement is rare and found only in 21% of patients with thyrotoxic myopathy.2 Earlier it was thought that isolated bulbar palsy did not occur without associated chronic thyrotoxic myopathy. But in the past 30 yrs., several authors have reported the existence of acute bulbar palsy in the absence of obvious muscle wasting.1 There are only isolated reports of dysphagia occurring as the sole myopathic feature of thyrotoxicosis.2,4,5 All these case reports emphasize the need for considering thyrotoxicosis in the differential diagnosis of dysphagia of obscure etiology. Once recognized and treated, the outcome is eminently satisfying. The dysphagia is known to resolve rapidly with the treatment of thyrotoxicosis.1 The mechanism of disordered esophageal motility in thyrotoxicosis is unknown. The postulates include hypercalcemia and hypomagnesemia. Hypercalcemia is known to cause dysphagia by its neuromuscular effects at neuromuscular junction. Left untreated, the complication of acute bulbar paresis is aspiration pneumonia, thyroid storm, cardiac failure, psychosis, coma and death. Beta-blockade alone can rapidly reverse a substantial part of the muscle weakness in hyperthyroidism. This response indicates that reversible changes in calcium handling and cyclic-AMP moderated contractile function contributes to thyroid myopathy.1 Beta blockers competitively inhibit the action of catecholamines at tissue receptor sites. Although no anatomic or biochemical studies of thyrotoxic bulbar myopathy have been reported, proximal skeletal muscles from such patients have revealed non-specific structural alterations. Possible neuromuscular causes of dysphagia in thyrotoxicosis include bulbar or esophageal myopathy, concomitant myasthenia gravis, and hypokalemic periodic paralysis. Mechanical compression by an enlarged goiter also can cause dysphagia. According to one report 16% of patients with thyrotoxicosis were found to develop bulbar muscle dysfunction. It is usually associated with chronic myopathy or thyroid crisis. An acute bulbar palsy without chronic thyrotoxic myopathy is extremely rare. The pathophysiological mechanism of impaired esophageal motility remains obscure.

Conclusion

Although acute thyrotoxic bulbar palsy as a sole presenting manifestation of thyrotoxicosis is rare yet it is well documented. Hence, hyperthyroidism must be considered in unexplained dysphagia.

Acute bulbar palsy is fraught with complications. Hence, early diagnosis and treatment can be life saving. The bulbar palsy evolves rapidly and is severe, but improves dramatically with treatment. Treatment is early administration of antithyroid drugs and concomitant administration of beta-blocker. The bulbar symptoms recover in as short as 1-10 weeks.
References


