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Case Report

Thyrotoxicosis with primary presentation as dysphagia: a rare manifestation

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ABSTRACT

Thyrotoxicosis refers to the clinical manifestations associated with elevated serum levels of T_4 or T_3 in an individual. Dysphagia is a common problem but a rare manifestation of hyperthyroidism. Dysphagia is an uncommon manifestation of thyrotoxic myopathy. Reports have been sparse and its incidence is not clear. In our case report patient presented who with dysphagia and weight loss and investigation revealed hyperthyroidism. Patient dysphagia subsequently resolved after receiving treatment with methimazole and beta-blockers. This case report the need to include thyrotoxicosis in the differential diagnosis of an otherwise unexplained case of dysphagia.

Keywords: Thyrotoxicosis, Myopathy, Dysphagia, Methimazole, Beta-blockers

INTRODUCTION

In thyrotoxicosis there is excess of thyroid hormone but it is not synonymous with hyperthyroidism. Hyperthyroidism exists when thyrotoxicosis is caused by thyroid overactivity. Major aetiologies of thyrotoxicosis are hyperthyroidism caused by Graves' disease, toxic MNG, and toxic adenomas. Prevalence of thyrotoxicosis is Caucasians is 2 to 3 per cent in women and 0.2 to 0.3 per cent in men.[1] Patients usually complain of restlessness, heat intolerance, increased sweating, pruritus, fatigue, weakness, muscle cramps, frequent bowel movements, or weight change. There may be palpitations or angina pectoris. Women frequently report menstrual irregularities.[2] However it is very rare that in thyrotoxicosis bulbar muscle wasting, weakness, and pharyngeal or esophageal dysmotility may occur, and the patient may presents with dysphagia.[3] In 1835 James Graves first described muscle weakness and atrophy during thyrotoxicosis. Up to 80% of thyrotoxicosis patients have neuromuscular symptoms and experience a wide range of muscular disorders: proximal myopathy, exophthalmic ophthalmoplegia, thyrotoxic periodic paralysis,myasthenia gravi.[4]

CASE REPORT

We report a case of 47-year-old male who came with a history of difficulty swallowing liquids and solid food for a period of around 1month of. He also gave history of hoarseness of voice, along with intermittent nasal regurgitation. He gave history of 15 pounds weight loss over the past 8 weeks, along with history of weakness in proximal girdle muscles despite of increased appetite. He denied any other history like, vomiting, diarrhea, diplopia, or dysarthria. He did not give any history alcohol or drug use.

On general physical examination patient was thin built, and he appeared cachectic. He was febrile, blood pressure

130/90 mmHg, pulse 110 beats per minute synchronous with regular rhythm. His respiratory rate was 16breaths per minute, and the oxygen saturation 98%. He had no lymphadenopathy. One examination there was diffuse enlargement of thyroid gland with palpable lower border and firm in consistency; however no bruit was heard over the gland. Ophthalmological examination showed lid lag but no proptosis (Figure 1).

Figure 1: Ophthalmological examination showed lid lag but no proptosis.

On cardiovascular examination heart sounds were heard and there was tachycardia. Respiratory system examination was normal. Nervous system examination there was mild weakness in the hip flexors and extensors bilaterally. The deep reflexes were brisk with normal plantar response. There was no sensory impairment and he had no cerebellar signs although s resting tremor of both the hands was present.

Investigation

Baseline investigation including blood count, E.S.R., blood sugar, urea, electrolytes, urine analysis, were normal. Liver function tests revealed a total bilirubin of 1.2 mg/dl, serum albumin 3g/l (normal 32-82 G/L) and mildly raised serum transferase levels 67 IU/l and serum aspartate 60 IU/l (Normal 0-35) and normal alkaline phosphatise. The corrected calcium was mildly raised at 2.8 mmol/L (normal levels 2.2 to 2.7 mmol/L) with a normal inorganic phosphate level. Chest X-ray and ultrasonography of whole abdomen were normal. Workup for dysphagia included a modified barium swallow test which revealed severe oropharyngeal dysphagia.

Upper GI endoscopy was done and it did not show any structural abnormality as shown in Figure 2. MRI brain was normal. Electromyography and creatinephosphokinase level 92 U/L (normal range <171U/L) were normal. Our work up included antibodies to the acetylcholinesterase receptor and Anti-Nuclear Antibodies (ANA) which were negative. Thyroid function tests revealed TSH 0.005 µIU/mL (0.25-4.5), free T_4 4.61 ng/dL (0.90-1.80) & total T4 17.2 ug/dL (4.6-12), total T_3 4.8 ng/mL (0.40-2.0), antithyroid peroxidase antibody (TPO) 298 IU/mL (<4 IU/mL). Thyroid ultrasound revealed diffuse heterogeneous multiple ill-defined nodules with largest measuring 0.5 x 0.8 cm suggesting of multinodular goitre. On the basis of history, clinical findings, and laboratory data, we considered hyperthyroidism secondary to multinodular goitre as the most likely cause for dysphagia. Patient was initially put on methimazole with dose of 10 mg three times daily which later increased to 50 mg in three divided doses and propranolol 40 mg twice daily. After around one week treatment patient was tolerating liquids and later on, after one month of treatment his symptoms resolved completely. Thyroid function tests repeated showed TSH 0.01 µIU/mL (0.270-4.0) and free T_4 1.94 ng/dL (0.90-1.80).

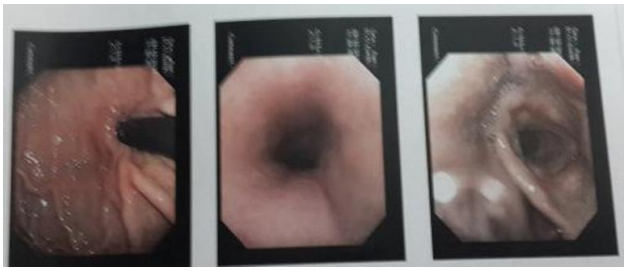

Figure 2: Upper GI endoscopy: it did not show any structural abnormality.

DISCUSSION

The most common presentations of thyroid disease are thyrotoxicosis (i.e. hyperthyroidism), hypothyroidism and goitre. Excess of thyroid hormone circulating in blood results in state called as thyrotoxicosis.[2] Thyroid hormones regulate the basal metabolism .and cat on in on almost all organs of body including gut and viscera, resulting in numerous gastrointestinal manifestations.[5] Review literature shows that levels of liver enzymes aspartate aminotransferase and alanine aminotransferase increases by 27% and 37%, respectively in hyperthyroids pateints.[6] Dysphagia as primary manifestation of hyperthyroidism is very rare and it can have an acute or chronic pattern.[7] As it is unusual for thyrotoxicosis to present dysphagia as primary symptom, it is important to rule out other causes of dysphagia. Work up in such a case will include investigation like modified barium swallow test, upper GI endoscopy, MRI of the brain, EMG & ACH antibodies. The causes of dysphagia broadly fall into two categories: obstructive and motility disorders. Common causes for dysphagia include are Zenker's diverticulum, myasthenia gravis, stroke,

Parkinson's disease & esophageal carcinoma.[8] Oropharyngeal dysphagia may typically present with symptoms like nasal regurgitation, chocking and aspiration especially in those with a bulbar palsy.[9] In our patient normal endoscopic and barium meal examinations virtually excluded an obstructive lesion. This made a motility disorder a strong possibility in him. Dysphagia in thyrotoxicosis may because of direct impingement of oesophagus by enlarging cervical or retrosternal goiter or, and possible neurologic causes may include eosphageal myopathy or dysmotility, concomitant myasthenia gravis and hypokalemia periodic paralyisis.[10] Myasthenia gravis occurs in 0.35% cases of hyperthyroidism while as 1-5% of patients of with myasthenia gravis develop hyperthyroidism.[11] In our case serum potassium levels were normal and anti ACH antibodies were not present so largely excluding possibility of associated myasthenia gravis and hypokalemia periodic paralyisis. Thyrotoxic myopathy usually appears after 1-3 months of thyrotoxicosis. It is more common in men, and can be the presenting feature. In review of literature most common musculoskeletal findings of thyrotoxicosis are painless skeletal muscle wasting, with normal muscle enzymes and nonspecific electromyographic abnormalities as was seen in our case.[12] In more than 50% of patients complains of muscle weakness may be present and 63% may show evidence of proximal muscle weakness or wasting.[13] The mechanism of disordered oesophageal motility in thyrotoxicosis is not known. The postulates include hypercalcemia and hypomagnesemia. Hypercalcemia is thought to cause dysphagia by its effect on neuromuscular release at the neuromuscular junction.[14] 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. The outcome of treatment is mostly good & dysphagia is known to resolve rapidly with the treatment of thyrotoxicosis.[12] However if left untreated, the complication of acute bulbar paresis is aspiration pneumonia, thyroid storm, cardiac failure, psychosis and even death. Beta-blockade alone can rapidly reverse a substantial part of the muscle weakness in hyperthyroidism.[15]

CONCLUSION

Although dysphagia may be a common symptom in middle or old age but in thyrotoxicosis its very rare manifestation. Our case tries to highlight the need to include thyrotoxicosis in the differential diagnosis of an otherwise unexplained case of dysphagia.

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