Thyrotoxic myopathy presenting as dysphagia

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Summary

A case of aspiration pneumonia due to inco-ordination of swallowing is described. Its aetiology lies in the myopathy of thyrotoxicosis and a tentative hypothesis as to its causation is suggested.

Case report

The patient was a 56-year-old woman who worked as a housewife. For 3 years she had noticed hyperhidrosis, weight loss and tremor but had not complained of this to anyone. She had suffered from many episodes of pneumonia which had been treated with antibiotics. Two episodes had been treated in hospital and the chest radiograph revealed that the localization of the pneumonia was in the apex of the lower lobe of the right lung. Bronchoscopy on the last admission had revealed no lesion and on both occasions the pneumonias had cleared up with physiotherapy and antibiotics.

She was admitted to hospital with a further episode of pneumonia. Again, this was in the apex of the left lower lobe of the right lung. It was on this occasion that her hypermetabolic state suggested thyrotoxicosis and a serum thyroxine revealed a level of 226 nmol/l. Again she was treated with antibiotics and physiotherapy and she made an excellent recovery.

Further questioning revealed that she slept on her back at night and she also had episodes of coughing when 'food went down the wrong way'. A barium swallow showed no physical obstruction. There were repeated swallowing attempts and there were filling defects found in both pyriform fossae, which were probably food residue. There was a lack of co-ordination in swallowing and barium stayed in the pyriform fossae for several swallows, which is a contra-indication to the normal state of affairs where it would be expected to clear after just one swallow. There was also spill of barium through the larynx into the trachea (Fig. 1).

She was treated with carbimazole 15 mg 3 times/

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FIG. 1. X-ray of oesophagus showing spill of barium through larynx into trachea.

day and after one month, barium swallow showed no reflux of barium into the trachea.

Follow-up for 2 years has revealed no further episodes of pneumonia.

Discussion

Severe muscular weakness and wasting was first reported by Graves (1835) and von Basedow (1840) in their descriptions of hyperthyroidism. Bathurst in 1895 and Sattler in 1952 specifically described patients who presented with severe muscular atrophy and weakness as the first indications of their thyrotoxicosis. Bathurst noticed the predominantly proximal involvement of the arms and legs and described how his patient found it difficult to rise from the lying or kneeling positions without using his arms.

Pipberger, Kälin and Wegmann (1955) considered that if thyrotoxic patients are carefully examined clinically and electromyographically, a majority will be found to have muscle involvement. Biochemically, Kobayashi and Takeuchi (1967) found low levels of serum magnesium which increased after treatment and Neguib (1963) claimed that thyrotoxic patients show an increase in muscular strength after treatment with magnesium chloride. Thyroxine increases the rate of respiration of liver mitochondria in the absence of ADP and causes mitochondrial swelling

and subsequent rupture. Thus, this would provide a possible mechanism for muscle dysfunction in thyrotoxicosis. In the case described here, magnesium was 0.43 mmol/l at the time of dysphagia but had risen to 0.82 at the time of disappearance of symptoms and achievement of the euthyroid state.

In summary, a case of thyrotoxic myopathy is described which resulted in inco-ordination of the swallowing process with resultant aspiration pneumonia. Treatment of the underlying thyrotoxicosis resulted in disappearance of symptoms and coincided with a rise in serum magnesium to normal levels.

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