Vomiting due to gastric stasis as the presenting feature in thyrotoxicosis

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Summary

A patient with severe thyrotoxicosis of the apathetic variant in whom vomiting was the prominent presenting symptom is described. An alternative mechanism for thyrotoxic vomiting is postulated.

Introduction

Vomiting in thyrotoxicosis is a well recognized, but unusual phenomenon (Rosenthal, Jones and Lewis, 1976), and it has been postulated that this is due to direct action of thyroid hormone on the chemoreceptor trigger zone.

Case report

The patient was a 43-year-old bricklayer who presented with a 4-week history of vomiting in the form of effortless regurgitation directly after meals. He also complained of weight loss of 19 kg over the previous 4 weeks. During this time he had had generalized abdominal pain, constipation, lethargy and weakness.

On examination he was dehydrated, pyrexial and there was evidence of proximal upper limb muscle wasting. He had a staring gaze, but no lid lag and an extremely small goitre. The only other abnormality was a sinus tachycardia of 120/min.

Investigations

Full blood count, urea and electrolytes, serum calcium normal. Thyroid function tests: T_4 250 nmol/l (normal range 50–150 nmol/l). Free thyroxine index 125 (normal range 11–51). Autoantibodies: thyroid microsomal ++++; gastric parietal cells +++++. Serum magnesium 0.50 mmol/l (normal range 0.78–1.03 mmol/l). Barium meal with ciné screening showed markedly diminished peristalsis in oesophagus, stomach and duodenum.

Treatment was commenced with carbimazole 15 mg 4 times/day, and within 48 hr his vomiting had ceased. Over the next 4 weeks his other symptoms resolved. Repeat barium meal one month after commencing treatment showed normal peristalsis, and the serum magnesium was within normal limits.

Discussion

Rosenthal *et al.* (1976) described 7 patients in

whom vomiting was a prominent symptom of thyrotoxicosis, postulating that it was due to stimulation of the chemoreceptor trigger zone by thyroid hormone. In the present patient the authors have observed a marked diminution in upper gastrointestinal motility, which reverted with treatment, and they postulate that hypomagnesaemia was responsible for the aperistalsis, which in turn resulted in vomiting.

This is in keeping with the findings of Hamed and Lindeman (1978) who described aperistalsis of the pharynx and oesophagus in a female patient with hypomagnesaemia secondary to parenteral nutrition. Full peristalsis was restored following i.v. magnesium supplements. Magnesium deficiency has been causally implicated in apathetic thyrotoxicosis, a variant of the disease usually occurring in the older patients in whom symptoms are mild or veiled although the intoxication is severe. The major features of this variant include fatigue, apathy, listlessness, dull eyes, weakness, congestive heart failure and low grade fever.

In experimentally induced hypomagnesaemia (Shils, 1969) apathy, weakness, nausea and vomiting were present in the majority of subjects. These features were due to an increase in both neuronal excitability and transmission across the neuromuscular junction.

It is postulated that the hypomagnesaemia in the patient described was responsible for his apathetic presentation and contributed to his vomiting by a direct action of the autonomic innervation of the upper gastrointestinal smooth muscle.

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