



Case Report:

Recurrent severe vomiting due to hyperthyroidism

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Abstract: Thyrotoxicosis may present in many ways; severe vomiting as a prominent symptom of thyrotoxicosis is uncommon. In this paper, we report a 24-year-old Chinese male with hyperthyroidism who presented with recurrent severe vomiting. The patient had had intermittent vomiting for seven years and had lost approximately 15 kg of weight. Gastroscopic examinations revealed chronic gastritis and one occasion peptic ulcer. He was treated with antacid and proton pump inhibitors, but his symptoms had no relief. His presenting symptoms suggested hyperthyroidism and were confirmed by laboratory data. After a month of propylthiouracil therapy, his symptoms were relieved. It should be noted that hyperthyroidism patients can have unexplained vomiting, and that hyperthyroidism may coexist with peptic ulcer in rare cases. Awareness of such atypical presentations of hyperthyroidism may help to make a correct diagnosis.

Key words: Vomiting, Hyperthyroidism, Peptic ulcer

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1 Introduction

Severe vomiting is not characteristic of typical hyperthyroidism, and it is rarely mentioned in standard textbooks of medicine. Rosenthal *et al.* (1976) reported that vomiting can be a presenting symptom of thyrotoxicosis. In this paper, we report a young Chinese male patient with hyperthyroidism who had experienced recurrent vomiting for seven years. The patient underwent several times of gastroscopy examinations before the thyrotoxicosis was diagnosed.

2 Case presentation

A 24-year-old Chinese man came to our outpatient clinic with chief complaints of intermittent severe vomiting and weight loss of seven years. The vomiting occurred after meals 2 to 3 times a day. Nausea and abdominal distention also occurred and relieved

by bed rest. The patient had no abdominal pain, diarrhea, or melena, and no heat intolerance, palpitation, excessive sweating, or nervousness. In the first year of the breakout of the symptoms, he was referred to a local hospital where gastroscopy revealed chronic gastritis, and he did not respond to antacid treatment. He lost appetite, had mild epigastric pain, and dropped 10 kg of weight. The patient took gastroscopy again, and an ampullary duodenal ulcer was found, so he was treated with proton pump inhibitors as well as antacid. Over the next six years, the patient continued to have intermittent nausea and vomiting despite the above therapy; however, his nausea and vomiting were eased a bit only by some Chinese herbal medicine.

One month before this admission, he again developed symptoms of nausea and vomiting, weight loss, and poor appetite. On admission, physical examination revealed that he weighed 42 kg, was 1.74 m in height, had decreased muscle mass, and was calm without any toxic symptoms. He presented with no sweating, tremor, or palpable goiter. His resting tachycardia was 102 beats per minute (bpm), blood

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pressure 135/78 mmHg, and body temperature 37.6 °C. The patient had no stare, proptosis, lidlag, or convergence lack, the thyroid gland was not palpable, and no tenderness or rebound tenderness was found in the epigastrium. The electrolytes, glucose, creatinine, and liver enzymes were all normal. Electrocardiograph (ECG) showed sinus tachycardia at 102 bpm. Abdominal ultrasonography was normal, while gastroscopy revealed chronic superficial gastritis, with no sign of *Helicobacter pylori* infection by staining. His gastrin was 43.07 pg/ml (ref. 25–100 pg/ml). Barium meal study of the stomach showed hypomotility and gastroptosis, while computed tomography (CT) scan of the abdomen result was normal. The patient was treated with prokinetic agents, but his vomiting was not relieved.

Because the patient had prominent weight loss and ECG showed tachycardia, we suspected that he might be a hyperthyroid. Free thyroxine (FT4) and free triiodothyronine (FT3) were 2.57 ng/dl (ref. 0.7–1.482 ng/dl) and 5.24 pg/ml (ref. 1.71–3.71 pg/ml), respectively, and thyroid stimulating hormone (TSH) was 0 mIU/L (ref. 0.35–4.94 mIU/L). Thyroglobulin antibody was 80.47 IU/ml (ref. 0–4.11 IU/ml). Thyroid ultrasonography showed that there was a small node (3.3 mm i.d.) in the right thyroid.

He was given propylthiouracil 100 mg three times a day, and his vomiting ceased one month later. His poor appetite improved, and he grew more active instead of lying in the bed all day long. His serum FT4 and FT3 became normal, and serum TSH was 0.0019 mIU/L one month later.

After discharge, the patient was on propylthiouracil 50 mg two times a day. Ten months later, he came back to our clinic for a follow-up, presenting no nausea or vomiting. The repeated gastroscopy revealed chronic gastritis that is similar to the previously found one, and the thyroid function was in a normal range.

3 Discussion

Thyrotoxicosis may present in many ways, including nausea, abdominal pain, palpitation, and vomiting. However, vomiting as a prominent presenting symptom of thyrotoxicosis is uncommon. After reviewing literature, 34 cases of thyrotoxic

vomiting were reported in the English literature. Of those patients, the ages ranged from 19 to 74 years old, and only 5 were male (Chen *et al.*, 2003; Al Sayed and Al Awadhi, 2007). We found similar data in the Chinese literature. Xu *et al.* (2007) reported that the ages of those 132 Chinese patients ranged from 20 to 76 years old, among which 26 were male, and most of them were misdiagnosed with digestive disease. The course of disease was quite different, with the shortest being 4 d and the longest 270 d. In our case, the young man had recurrent severe vomiting for seven years, the longest history of thyrotoxic vomiting in a patient. What is more, the young man presented with severe vomiting that affected his life, which is quite different from other reported cases.

The patients reported in both English and Chinese literatures all improved their vomiting symptoms after the diagnosis and treatment of hyperthyroidism. In our case, the young man also stopped vomiting after treatment even though he still had chronic gastritis. The vomiting in hyperthyroidism may be caused by thyrotoxicosis, which can be the prominent presenting symptom for hyperthyroidism.

The mechanisms causing vomiting in thyrotoxicosis are yet to be determined. Possible mechanisms for this symptom include altered gastric motility, which can occur from thyroid hormone excess (Sellin and Vassiopoulou-Sellin, 2000). Hyperthyroidism may cause hyperperistalsis resulting in outlet dysfunction leading to vomiting. Kisakol *et al.* (2003) suggested that in subclinical hyperthyroidism, gastric emptying of solids was similar to that in age-matched healthy control subjects. Gastrointestinal dysfunction is dependent on the severity of the hyperthyroid state and might be a result of impaired neurohormonal regulation (Kisakol *et al.*, 2003). The increase in β -adrenergic activity in hyperthyroidism is the most likely mechanism of thyrotoxic vomiting (Bilezikian and Loeb, 1983).

Lying in bed can relieve symptom of vomiting, as it was observed in our patient. Indeed, his vomiting was worse after meals or during walking. We regarded gastroptosis as a result of severe emaciation. This symptom would be aggravated by standing, especially after meals, which was supported by the barium meal study of the stomach.

The patient suffered from chronic gastritis and one occasion peptic ulcer. The coexistence of

hyperthyroidism and peptic ulcer is extremely rare in literature. Crile (1934) reported that the incidence of peptic ulcer in hyperthyroid patients was 48 cases out of 9618 examined (about 0.5%). However, it was also reported that the disease is often accompanied by chronic gastritis (Garbat, 1951). Peptic ulcer is now known to be caused by *H. pylori* infection and excessive gastric acid secretion. With regard to the correlation between hyperthyroidism and peptic ulcer, Aoyagi *et al.* (1982) found that the mean value of gastric acid output was higher in hyperthyroid patients than in controls, with extremely high gastric acid output being noted in 8 of the 24 hyperthyroid patients. They regarded the coexistence of hyperthyroidism and peptic ulcer to be infrequent but not rare (Aoyagi *et al.*, 1982). In our case, we found the *H. pylori* staining negative by biopsy of the gastric mucosa. Although the patient was only partially relieved after treatment with proton pump inhibitors, he still suffered from gastritis in the long term, which was supported by the endoscopy. Since his symptoms of nausea and vomiting disappeared after taking propylthiouracil, so we suspect that hyperthyroidism may be the primary cause for vomiting in our case.

4 Conclusion

Although weight loss is always an important clue to make diagnosis of hyperthyroidism, it should be noted that hyperthyroidism patients can have unexplained vomiting, and that hyperthyroidism may coexist with peptic ulcer in rare cases. Therefore,

awareness of such atypical presentations of hyperthyroidism may help to make a correct diagnosis.

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