THYROTOXIC HYPEREMESIS: A CASE REPORT

K W Wang, K S Mui

ABSTRACT

A male patient with hyperemesis as a result of hyperthyroidism was presented. Investigations for causes were negative except for hyperthyroxinaemia. Treatment with antithyroid drug relieved the symptom. Hyperemesis associated with hyperthyroidism occurs predominantly in females. A likely emetic factor oestrogen. Hyperthyroidism could have potentiated this effect. Levels of oestrogens are raised in thyrotoxicosis. The hyperthyroidism, the raised oestrogens and a low emetic threshold conspired to hyperemesis in this patient, a situation not unlike hyperemesis gravidarum. Thyrotoxic vomiting once recognised is readily relieved by antithyroid treatment.

Keywords: Graves' disease, hyperemesis, causative factors

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INTRODUCTION

Thyrotoxicosis may present with a variety of disturbances. Gastrointestinal manifestation is sometimes pronounced with polyphagia and diarrhoea. Intractable hyperemesis is an uncommon symptom in thyrotoxicosis. We report here an unusual but interesting patient with thyrotoxic hyperemesis and discuss a possible pathogenetic mechanism.

CASE REPORT

The patient, a 28-year old male carpenter was admitted on 28 August 1986 for the problem of epigastric pain, abdominal distension and vomiting on and off since 1980. His symptoms were getting worse one week prior to admission. One day before admission, he visited the Accident & Emergency Department for severe vomiting. He was treated as for gastritis. However the symptoms persisted and he was admitted the next day.

He had no recent weight loss but had slight loss of appetite. Examination revealed a fit looking young man, afebrile with a blood pressure of 130/80 and a pulse rate of 120/min. There was no clubbing or oedema and the heart and lungs were normal. The thyroid gland was not enlarged. Slight tenderness was detected in the epigastrium but no other abdominal signs were elicited. There were fine tremors of the hands, the palms were warm but not sweaty. There was a suspicion of lid lag but no exophthalmos was noted. No obvious pallor or jaundice was detected.

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A provisional diagnosis of exacerbation of peptic ulcer was made. The vomiting was intractable in the ward and abdominal discomfort exacerbated. Consequently, he was put on nasogastric suction and intravenous regime for 2 days. During this time, he was monitored for an acute abdomen. His haemoglobin, total white count, urea and electrolytes were normal. Serum amylase was not raised. Chest and abdominal x-rays were normal. ECG showed a sinus tachycardia and left ventricular hypertrophy by voltage criteria. Gastroscopy and oral cholecystogram did not show any abnormality. Serum thyroxine level was sent for.

He improved after a few days and was discharged. On follow- up, about 2 weeks later, he still had nausea and occasional vomiting. The serum thyroxine level was noted to be high, 20.8 ug/dl. A full thyroid function panel was ordered for confirmation. The results were T_4 15.5 ug/dl, T_3 uptake 125%, free thyroxine index 19.4 (normal range: 4.6-11.6). Thyroid antibodies to microsomal and thyroglobulin were negative. He was started on Carbimazole 20 mg bd and Propranolol 20 mg tds.

A month later, he had no more vomiting or nausea. His weight increased from 52 kg to 59 kg. T_4 was 64 ug/dl and FTI 6.1 eight weeks later. He was maintained on Carbimazole for a year. During this time he remained well with no further complaint of nausea and vomiting.

DISCUSSION

Thyrotoxicosis has an effect on all systems of the body. The gastrointestinal manifestation is commonly that of polyphagia, hyperdefaecation or diarrhoea. Vomiting as a presenting symptom is distinctly uncommon. It was not listed in a standard text on endocrinology (1) nor featured in a local study (2). Its occurrence has been mentioned mostly in relation to gestational hyperthyroidism. Thyrotoxic hyperemesis that requires institutional management is even more unusual apart from hyperemesis gravidarum. Thyrotoxic emesishyperemesis appears to have a female preponderance even outside of the gravid state. The series of Rosenthal (3) has only one male out of seven patients with such a presentation. It is likely that a sex related pathogenic factor is involved (vide infra). The absence of other gastrointestinal symptoms refutes hypermotility as a causative mechanism.

Hyperemesis associated with hyperthyroidism has been studied in patients with hyperemesis gravidarum. That the thyrotoxicosis caused the hyperemesis was shown by an improvement in this complaint with antithyroid treatment (4-7). Transient hyperthyroidism was found in 43.6% of patients in a study (8) and in 73% in another study (9) on hyperemesis gravidarum. The symptoms and hyperthyroidism subsided spontaneously in some patients but others required antithyroid treatment. At present the role of hyperthyroidism in patients with hyperemesis gravidarum remains unclear. Is it a causative factor or a concomitant phenomenon?

Thyroxine per se has no demonstrable emetic property. Most thyrotoxic patients do not have vomiting and patients with thyrotoxic vomiting do not have more severe disease clinically or biochemically. Human chorionic gonadotrophin (HCG) was claimed by some (10, 11) but not all researchers (12) to be the cause in hyperemesis gravidarum. Among the sex related factors, the oestrogens may fulfil the role of an emetic agent. Some females developed nausea and vomiting (12, 13) when given oestrogenic preparations and they would develop similar complaints in pregnancy. Likewise women who had similar complaints in pregnancy were intolerant of diethylstilbestrol (12). Studies of pregnant women, however, have not shown a significant difference in the levels of oestrogens between those with and without symptom of emesis (11-13). There is, therefore, a variable susceptibility to the emetic effect of the oestrogens. It is also pertinent to note that oestrogens rise progressively in pregnancy but the vomiting occurs mainly in the first trimester. It is possible that gradual desensitisation has attenuated the effect. Indeed some authors claimed therapeutic successes with oestrogen treatment of hyperemesis gravidarum (12). Other workers had been able to overcome oestrogen intolerance with graduated doses. If an indirect role for hyperthyroidism is involved in emesis-hyperemesis, it could be the potentiation of this stimulatory effect.

We postulate that a similar play of factors occurred in our patient. Researchers have documented increased levels of oestrogens in patients of both sexes with thyrotoxicosis (14-16). Indeed male patients with gynaecomastia had oestrogen concentration comparable to that of a normal female (16). A rise in oestrogens could result in nausea and vomiting in the susceptible subject. Our patient would have been subject to such changes. That he manifested the symptoms is most likely a result of his intrinsic hypersensitivity as evidenced by the preceding history. He had recurrent nausea and vomiting for six years which pointed to a low emetic threshold. The onset of hyperthyroidism tipped the scale precipitating a hyperemetic state not unlike that of hyperemesis gravidarum. As such, antithyroid treatment abolished the problem. It will be educational to observe his condition should he relapse because hyperemesis will be predicted by this postulate.

CONCLUSION

We report here a patient with an unusual presentation of hyperthyroidism. Hyperemesis in this setting is simple to manage once diagnosis is made, missing this will lead to a great deal of anxiety and unnecessary investigations. A similar situation may occur in pregnancy and timely treatment will alleviate suffering. We postulate a common mechanism in both situations. It will be interesting to examine the effect of antioestrogen on hyperemetic state when the opportunity next arises in this patient.

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