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Hyperemesis gravidarum due to thyrotoxicosis

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Summary

A case of hyperemesis gravidarum at 9 weeks' gestation is described for which no cause could initially be found. Intravenous feeding was needed as a life preserving measure and following improvement on this regime the patient went into a thyrotoxic crisis which was successfully diagnosed and treated. The continuing pregnancy and its outcome is described. The need for practitioners to remember vomiting as a presenting sign of thyrotoxicosis is stressed.

Introduction

Hyperemesis gravidarum is accepted as an unfortunate complication of early pregnancy and its treatment is conservative by rehydration and the administration of anti-emetics. Occasionally this therapy fails to provide a lasting cure and relapse occurs. Such a case is now described, the cause of which was eventually elucidated as thyrotoxicosis, the emetic symptoms of which could only be controlled by propranolol during the pregnancy even when the patient was euthyroid; and yet control after delivery was effected solely with antithyroid drugs.

Case history

A 28-year-old, English-speaking Indian patient was admitted at 9 weeks' gestation with a 5-day history of vomiting following a 2-week history of upper abdominal pain. General examination was normal apart from gross dehydration which was corrected by i.v. fluids. The uterus was the correct size for gestation and an ultrasound scan confirmed a normal singleton fetus. Human chorionic gonadotrophin levels were normal for the pregnancy's gestation.

Although rapidly rehydrated, the patient was unable to take oral food for a week regardless of the diet offered—and she mentioned that she had had 4 similar episodes of nauseous anorexia over the preceding 2 years. Liver function tests showed raised enzyme levels on admission, but these settled on rehydration. Serum calcium concentrations were at the lower limit of normal. Malaria, tuberculosis, pancreatitis and renal disease were all excluded as the primary cause of her symptoms. After 10 days the patient was discharged only to be re-admitted 12 hr later with a recurrence of her vomiting.

The vomiting settled on conservative management, but she was more agitated and tremulous and complained of headaches and a stiff neck, peripheral paraesthesia and generalized weakness. The results of lumbar puncture were normal, so endocrine studies were initiated. However, the vomiting persisted regardless of therapy and the patient became emaciated and required parenteral feeding 14 days after her second admission. She slowly improved on this regime although difficulty was experienced in controlling her plasma sodium and calcium concentrations, and she required extra supplements. On the eighth day of intravenous feeding the patient spiked a pyrexia of 39°C with a tachycardia of 120/min and complained bitterly about her generalized myalgia, arthralgia and weakness that had become obvious following mobilisation. A pan-systolic murmur was heard at the apex for the first time, as was a thyroid bruit.

Initial worries about a septicaemia from the CVP line were confirmed with a culture of coagulase-negative *Staphylococcus,* sensitive to cephradine with which she was treated. Her other symptoms were explained by the timely return of her thyroid function tests, with a free thyroxine index of 279 (normal 48–134), this being the only reliable thyroid function test in pregnancy (Barnes, 1974; Ramsay, 1976). A diagnosis of thyrotoxic crisis was now made and successfully treated with carbimazole, propranalol and Lugol's solution. Hydrocortisone was given to cover the concomitant relative hypoadrenalism common in thyrotoxicosis. The patient's

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response was dramatic and she was discharged home 3 weeks later on maintenance doses of carbimazole and propranolol, the latter being given because her vomiting persisted without it.

Two weeks later (18 weeks' gestation) the patient was re-admitted, with vomiting which was controlled by re-introducing propranolol which she had discontinued. Although the remainder of the pregnancy was complicated by a small-for-dates fetus with falling human placental lactogen levels, necessitating Caesarean section at 38 weeks' gestation, there was no recurrence of her vomiting, so long as she had a maintenance dose of propranolol.

The patient's LATS* and LATS-P levels were low and the fetus had no problems with neonatal thyrotoxicosis (Dirmikis and Munro, 1975), although there were difficulties with feeding ascribed to the birth weight of 2·10 kg.

Postnatally the mother developed retrosternal pain and a choking sensation, but no retrosternal goitre or tracheal compression could be found. The patient remained euthyroid on anti-thyroid therapy alone, the propranolol being stopped after delivery, without adverse effect.

Discussion

Thyrotoxicosis is a rare disorder in pregnancy with an incidence of about 0·5/1000 pregnancies (Javert, 1940; Kibel, 1944; Becker and Sudduth, 1959; Hawe and Francis, 1962). Even more rarely it may present for the first time in pregnancy, although it is not certain whether pregnancy has a causal relationship (Barnes, 1974). The relevance of hyperthyroidism in pregnancy is obvious from the maternal standpoint, and from the fetal aspect one must remember the condition is associated with a high incidence of low birth weight fetus, fetal demise and an increased neonatal mortality rate (Ramsay, 1976).

Vomiting as a presenting symptom is rare and is usually seen as a presentation of thyrotoxic crisis (Cameron, 1945), the normal mode of presentation of thyrotoxicosis, even in pregnancy, being tachycardia, excessive sweating and weight loss (Barnes, 1974). Recent literature has suggested that vomiting may be more important as a cardinal symptom of thyrotoxicosis (Rosenthal, Jones and Lewis, 1976) and that it is not only seen in a crisis, but is part of a more prolonged chronic presentation with associated signs of sweating, exophthalmos and myopathy developing later. These symptoms may be related to hypercalcaemia or hypocalcaemia in equal proportions (Skrabanek, 1976; Gordon *et al.*, 1974; Rose and Boles, 1953; Twycross and Marks, 1969; Harper and Osborne Hughes, 1970; Guyer, 1965).

The mechanisms of vomiting in thyrotoxicosis are basically unknown, although it is thought to be due to a central effect on the medullary trigger centre as in hyperemesis gravidarum and other metabolic disorders causing vomiting (Rosenthal *et al.*, 1976).

This case has been described for 2 reasons, the first to remind medical practitioners yet again that common things are not the only things that occur, especially in pregnancy, and secondly to document the first case of proved pregnancy thyrotoxicosis in which the attainment of a euthyroid state, based on the free thyroxine index by administration of carbimazole, failed to control the vomiting—which, however, responded dramatically to treatment with propranolol which was necessary until delivery.

* Long-acting thyroid stimulator.

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