Thyrotoxic crisis following eclampsia and induction of labour

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Summary

A patient was diagnosed to be thyrotoxic and commenced on medications, but it was not known that she was 13 weeks pregnant at the time. She failed to take the medications and presented at 25 weeks with eclampsia and thyrotoxic crisis. Her management is discussed.

Introduction

Hyperthyroidism has been estimated to complicate approximately 2 in 1000 pregnancies in the United Kingdom (Niswander and Gordon, 1972; Burrow, 1977; Montgomery and Harley, 1977). Thyrotoxic crisis is now rare. A case is described in which pregnancy was complicated by both eclampsia and a subsequent thyrotoxic crisis.

Case report

A 28-year-old Indian woman presented to the surgical out-patient clinic with complaints of weight-loss and a neck swelling. On examination the surgeon found that the patient had a smooth goitre, lid lag and a tachycardia of 120 per min. The serum thyroxine (T4) level was 215 nmol/l (normal 60-135). She was started on carbimazole 10 mg three times daily. It was however, not known that she was 13 weeks pregnant at this time. She did not turn up for review 4 weeks later.

Nine weeks after initial presentation to the surgical out-patient department, she attended the antenatal booking clinic in her 5th pregnancy at 22 weeks gestation. She had had four previous uneventful pregnancies. The uterine size corresponded with dates and the blood pressure was 120/80 mmHg and the pulse rate 110 per min. She had a smooth goitre and lid lag was present. She claimed to be taking carbimazole regularly (though this later proved to

be false). Thyroid function tests were not done.

Three weeks after booking she had two fits and was admitted as an obstetric emergency. On arrival blood pressure was 190/105 mmHg, pulse rate was 140 per min, and there was heavy proteinuria with ankle oedema. Eclampsia was diagnosed and treatment started with a constant infusion of 0.8% chlormethiazole and hydralazine 40 mg in 50 ml of 5% dextrose intravenously. After an epidural anaesthetic block, labour was induced with extra-amniotic prostaglandin. Eight hours later a female fetus was aborted.

Following delivery her condition was stable for approximately 7 hr, when she developed extreme restlessness despite the chlormethiazole. She was tachypnoeic with a tachycardia of 200 per min, blood pressure was 170/95 mmHg, and there were bilateral basal lung crepitations. She became unrouseable and chlormethiazole was stopped. There were no localizing signs in the central nervous system. Cardiac failure was treated initially with digoxin 0.5 mg and frusemide 40 mg intravenously, but her condition did not improve. Thyrotoxic crisis was suspected and treatment started with propranolol 40 mg four times daily, carbimazole 15 mg three times daily, digoxin 0.25 mg (two doses), and 5 drops of Lugol's iodine, all given through a Ryle's tube. She gradually improved and 40 hr postpartum was fully conscious with a normal blood pressure and no signs of cardiac failure, although a tachycardia of 100 per min persisted. She had passed 12.5 litres of urine in the preceding 24 hr.

Carbimazole and propranolol were continued during the puerperium and she was discharged 7 days after admission. Thyroid function tests on admission confirmed thyrotoxicosis—T4 146 nmol/l (60-135), T3 uptake 66% (93-117), free thyroxine index (FTI) 17 (3.4-13).

Discussion

This patient was found to be thyrotoxic in early pregnancy before booking but did not take her medication. Thyrotoxic crisis usually results from inadequate treatment (Ingbar, 1966), and can be precipitated by infection, trauma, surgical emergencies, eclampsia or labour (Kamm *et al.*, 1963). In this patient, the thyrotoxic crisis appears to have been precipitated by both eclampsia and inadequate antithyroid therapy. The latter was mainly due to the patient's failure to take the prescribed medication. This fact was not appreciated by the obstetric staff at booking as she appeared controlled clinically and when, for the same reason, thyroid function tests were not done. If these had been done, her thyrotoxic state would have been discovered and the subsequent thyrotoxic crisis might have been prevented.

Thyrotoxic crisis can be life-threatening, and is characterized by extreme irritability, delirium, coma, fever, tachycardia, hypotension, vomiting and diarrhoea (Komins, Snyder and Schwarz, 1975). High output cardiac failure frequently results. Urgent treatment is required and should include oxygenation and rehydration as well as general supportive measures. Specific treatment is designed to prevent secretion of thyroid hormones and to modify their effects on endorgans (Harrison, 1977). Anti-thyroid drugs will prevent hormone synthesis and Lugol's iodine will inhibit secretion of already synthesized hormones. Propranolol counteracts the peripheral effects mainly by its effect as a beta-

adrenergic blocker. With combined treatment the hypermetabolic state can be controlled, and its effectiveness was dramatically illustrated by the excellent response in our patient.

This case illustrates the dangers of untreated thyrotoxicosis in pregnancy and it also emphasizes the utmost importance of controlling thyrotoxicosis rapidly during pregnancy. This should preferably be done by a physician experienced in the handling of thyroid problems.

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