Congenital Thyrotoxicosis: a Case Report.

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Abstract

A case of a preterm neonate with congenital thyrotoxicosis is reported. Her mother had thyrotoxicosis which was diagnosed at 27 weeks gestation, and was treated with propranolol and carbimazole. The diagnosis of thyrotoxicosis in the neonate was confirmed by an elevated serum tri-iodo-thyronine (T3) level of 6.7 nmol/L, a thyroid stimulating hormone (TSH) level of less than 0.3 mu/L, and advanced bone age of between 3-6 months, at birth. The baby was treated with propranolol and Lugol's iodine, with a favourable outcome.

Keywords: Thyrotoxicosis, Maternal, Congenital, Neonate [Trop J Obstet Gynaecol, 2002, 19: 51-53].

Introduction

Congenital or neonatal thyrotoxicosis is a rare condition. Since the first report in 1912 [1], about seventy-nine cases have been reported in the medical literature [2,3,4], with only one report involving a Nigerian neonate [4].

It occurs in about 1-5% of infants of mothers with either a past or present history of thyrotoxicosis (Grave's Disease) [2,5]. The majority of affected newborns have a transient, self-limited clinical course, which may or may not require active management. In a very small percentage, however, the condition may be life threatening and the disorder may persist for months or years. The prevalence of this condition in Nigeria is unknown. This second report in a Nigerian neonate is being made to further draw attention to the condition, the associated complications, and the problems of management.

Case Report

Baby I. O., female, Hospital No. 490896, was delivered at the University of Nigeria Teaching Hospital (UNTH), Enugu by emergency lower segment caesarian section at a gestational age of 34 weeks, because of uterine contractions with drainage of liquor and decreased fetal movement of 24 hours duration in a thyrotoxic pregnant woman.

The mother, a 31 year old para3+0 Nigerian Ibo who resides in Lagos, presented at the Ante-natal clinic at a gestational age of 27 weeks with an eight month history of progressive weight loss despite a good appetite, palpitations, diarrhea, easy fatigability, tremors, hot sweaty palms, heat intolerance and breathlessness on exertion. There was exopthalmos and a neck swelling of two weeks duration. She had been seen at several private clinics in Lagos without a definite diagnosis.

A clinical diagnosis of thyrotoxicosis in pregnancy was made. She was admitted and commenced on carbimazole 20mg twice daily and propranolol 40mg eight hourly after investigation and review by the Physicians. The diagnosis was confirmed by a serum T3 level > 10mmol/L (1.2-2.8), T4 > 250mmol/L (50 – 150) and a low TSH of 0.3mu/L (0.5-6.5). Three weeks into admission, she was discharged against medical advice. Four weeks later, she represented with uterine contractions, drainage of liquor and decreased fetal movement. A viable fetus in breech presentation was confirmed on sonography and an emergency C/S was performed, resulting in the delivery of a live female baby with Apgar 5/8. She was immediately transferred to the Newborn Special Care Unit (NBSCU). On admission, we saw a small for age, irritable, unusually alert female neonate with increased spontaneous activity. She had an EGA of 34 $\pm$ 2 weeks, a length of 44cm, weight 1800gm and head circumference of 29.5cm. The anterior fontanelle was patent, admitting the tip of the index finger. The was a tachycardia and tachypnoea of 180 and 100/minute respectively. The Central Nervous System was normal, no goiter evident or palpable and there was no clinical evidence of congestive cardiac failure. The blood pressure was not measured.

Diagnoses of Low Birth Weight, moderate prematurity and probable congenital thyrotoxicosis were made and she was considered to be at risk for cardiac failure and septicaemia. She was managed appropriately and commenced on naso-gastric propranolol 1 mg 8 hourly, after blood was collected for thyroid function tests and CBC. Lugol's iodine, one drop 8 hourly, was added as soon as it became available.

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X-ray of the left wrist for bone age showed two epiphyseal centers and the bone age was reported by the Radiologist as "advanced to 3-6 months." Thyroid function levels of T3 – 8.7mmol/L and TSH of 0.3mu/L, confirmed the diagnosis of congenital thyrotoxicosis. There was progressive clinical improvement on the above regimen during the seven weeks of hospitalization. On discharge, the baby weighed 2.75kg with a length of 48cm and head circumference of 34cm. The sleeping pulse and the respiratory rate had dropped to 140 and 68 per minute respectively, while cardiovascular and neurologic examination remained normal. The patient, unfortunately, was lost to follow-up. Repeat thyroid studies and X-ray were not done before discharge due to financial constraints.

Discussion

This case describes the classical and more common clinical, biochemical and radiologic features of congenital thyrotoxicosis, a very rare condition whose aetiopatogenesis remains unclear. The commonly accepted postulation that it results from transplacental passage of thyroid stimulating antibodies/immuno-globin (TSAB) (previously called LATS), resulting in stimulation of the fetal thyroid gland, does not satisfactorily explain the condition. Some reports[6,7,8] have indicated that congenital thyrotoxicosis can occur without detection of TSAB in the mother while a mother with TSAB can give birth to a normal neonate. Untreated, thyrotoxicosis in a pregnant woman has a high incidence of maternal and fetal complications, the time of onset of symptoms and the severity of manifestation being modified by maternal thyroid function biochemisty. The course of the condition is variable, being 'transient' in the majority of cases and life-threatening in a few, through vascular complications[6]. Fetal complications include intra-uterine death, premature labour and delivery, intra-uterine growth retardation and congestive cardiac failure. Maternal death could occur[9,10]. The classical disease runs a self limited course, resolving spontaneously by 3-6months. Intra-uterine exposure to anti-thyroid drugs may delay onset of symptoms by as long as 12 days, a latent period believed to allow for degradation of maternally acquired anti-thyroid drugs[5]. Both variants may be complicated by craniosynostosis, intellectual impairment ranging from mild perceptual handicaps to severe psychomotor retardation on long term follow-up[6]. The explanation for this variation remains unclear, making follow-up mandatory. Our patient was symptomatic at birth while the first case reported in a Nigerian neonate[4] from LUTH became symptomatic on day 12.

The cardiac decompensation commonly seen in thyrotoxic neonates has been attributed to the hypermetabolic state and sympathetic overstimulation of a cardiac muscles by excess thyroxin. Adelman, however, found that neonatal hypertension may also be contributory[11], emphasizing the need for systematic measurement of blood pressure in an infant at risk.

Modalities of treatment of the thyrotoxic neonate have been derived from the principles of treatment of older patients. Combinations of propylthiouracil, carbimazole, propranolol, digoxine and Lugol's iodine, have been used[12,13]. Out patient received propranolol and Lugol's iodine. Propranolol causes a rapid reduction in cardiac/respiratory rates, which iodine paralyses the 1st step in the release of thyroid hormone from storage colloids, resulting in an almost immediate shut-down of hormone secretion[5]. Transue et al[14], recently reported the management of an infant using monotherapy with sodium iopanoate, and maintaining a euthyroid status with minimal effort.

Although neonatal thyrotoxicosis is uncommon, early diagnosis in pregnancy and treatment of a symptomatic neonate are mandatory to achieve good perinatal outcome. Close surveillance of maternal thyroid status in the affected woman, intensive fetal monitoring and the early involvement of the Paediatrician, all have a synergistic beneficial effect on outcome. The transient self-limiting course of the disease may be misleading because of the identified sequelae[6]. Long-term follow-up of treated patients is strongly advocated.

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