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Neonatal Graves` Disease-A Case Report

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Case Report

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Abstract: Neonatal Graves' disease is a rare disorder seen in 1% of the offspring of mothers with either established or cured Graves' disease (GD). It results from the transplacental transfer of thyroid stimulating immunoglobulins (TSIs) from mother to fetus, in women with GD. A higher TSIs antibody in maternal serum makes hyperthyroidism more likely in the fetus or newborn. In spite of its rarity, its serious nature and its association with multisystem abnormalities justifies careful clinical screening and management. We report a term neonate with neonatal Graves` disease secondary to maternal Graves' disease. The patient was treated accordingly with a good response.
Keywords: Graves` disease, Hyperthyroidism, Thyroid stimulating immunoglobulins, Exophthalmos, Failure to thrive.

INTRODUCTION

Fetal and/or neonatal hyperthyroidism is a rare condition, and its incidence has been estimated around 1:4000–40000 [1]. The prevalence of maternal hyperthyroidism due to GD in pregnancy varies from 0.1% to 2.7% [2]. The prevalence of transient neonatal GD born to these mothers is varying from 1.5% to 20.0% in observational cohort studies [3,4]. In most cases, it results from the transfer of thyroid stimulating immunoglobulins from mother with GD to fetus through the placenta [5]. In 1–5% of the babies born to these mothers, these antibodies will stimulate the thyroid by binding with thyrotropin receptor, causing a clinical hyperthyroidism that may present at birth [6].

It is well known that maternal thyroid antibodies freely cross the placenta and can act in the fetal thyroid gland during the second half of pregnancy. The diagnosis of neonatal hyperthyroidism can be overlooked, resulting in morbidity and mortality with mortality rates up to 20% [7]. Further, recent reports suggest that delayed diagnosis and treatment can have adverse impact on neurocognitive outcomes [8]. Neonatal complication rates are higher in women who remain hyperthyroid during the second half of pregnancy [9].

CASE REPORT

A forty five day old male term baby admitted with prominent eyeballs, irritability, less sleep, weight loss and loose motion since birth. (Figure 1) Her mother was diagnosed as Graves' disease two years and six months back and treated with tab. Carbimazole followed by radio-iodine therapy. Since then she became hypothyroid and treated with tab thyroxine. On examination, the baby was alert, irritable, temperature was 100° F, pulse was 160/m, and respiratory rate was 38/m and had bilateral exophthalmos. (Fig 1) His weight was 2.5 kg, WA was – 3SD, length was 49 cm and OFC was 34 cm, both were on 3rd centile, and WL was -2.75SD. Investigation of the patient showed FT4 was 8.3 ng/dl (N;4.5-12.5 ng/dl), FT3 was 5.98 pg/ml (N;1.4-4.2 pg/ml) and TSH was <0.001 µIU/ml (N;0.8-9 µIU/ml). Baby`s anti TPO ab was 11.6IU/ml (N;<5.6 IU/ml), anti TG ab was <20 IU/ml (N; upto 40 IU/ml) and mother anti TPO ab was 68.6IU/ml (N;<5.6 IU/ml), anti TG ab was <20 IU/ml (N; upto 40 IU/ml ). USG of thyroid gland of patient was normal. We diagnosed the case as a neonatal Graves` disease and treated with tab carbimazole and tab propranolol. On follow up he became euthyroid and stopped drug after 3 months.

Fig-1: shows bilateral exophthalmos

DISCUSSION

The first case of fetal hyperthyroidism probably due to maternal antibodies was described by White in1912 [10]. The infant develops clinical symptoms within the first postnatal month, but usually is apparent by 10 days of age[8]. Late-onset neonatal hyperthyroidism occurring at about 45 days of age and persisting for more than 6 months also has been reported. The clinical manifestations of neonatal hyperthyroidism may be mild or severe (thyrotoxicosis) and include: intrauterine growth restriction, goiter, central nervous signs: irritability, jitteriness, and restlessness, ophthalmologic signs: periorbital edema, lid retraction, and exophthalmos, cardiovascular signs: tachycardia, arrhythmias, cardiac failure, and pulmonary hypertension, signs of hypermetabolism: voracious appetite, weight loss, diarrhea, sweating, and flushing, advanced bone age, craniosynostosis, and microcephaly [11]. Neonatal thyrotoxicosis usually remits after 8 to 20 weeks. Virtually all infants are euthyroid by 48 weeks' of postnatal age and rarely thyrotoxicosis persists [12]. Our patient presented with features of hyperthyroidism since birth. His clinical features were irritability, less sleep, loose motion, weight loss, exophthalmos and failure to thrive. The patient becomes euthyroid by 5th month of age with antithyroid drug.

It is well known that maternal thyroid antibodies freely cross the placenta and can act in the fetal thyroid gland during the second half of pregnancy [1]. In the absence of maternal treatment with anti-thyroid drugs, hyperthyroidism develops in the fetus during the second half of pregnancy [13]. Hyperthyroidism can develop even in the presence of anti-thyroid drug therapy if the titer of TRAbs is high. Mother of our patient was hyperthyroid before pregnancy and treated with antithyriod drug and radioactive iodine but during pregnancy mother was hypothyroid and anti-TPO antibody was positive therefore maternal antibody crossed placenta as evident by positive anti-TPO antibody in child, which was responsible for features of hyperthyroidism in infant. In our patient ultrasonography revealed normal finding, Ultrasonography that reveals fetal goiter is a valuable finding, but goiter can be difficult to detect and measure.[11] An-TPO antibody was positive in 80% and anti-TG antibody was positive in 85% untreated Graves` disease and significantly decreased with treatment [14]. An-TPO antibody was positive and anti-TG antibody was negative in our patient as well as in mother.

Symptomatic neonatal hyperthyroidism should consider an emergency and treated quickly. Medical treatment includes the antithyroid drugs (propylthiouracil, carbimazole or methimazole), which block thyroid hormone synthesis and inhibit the peripheral conversion from T4 to more active hormone T3 [15]. Beta blockers, which are effective in controlling symptoms and inhibit the peripheral conversion from T4 to T3, and iodine solution, which suppresses thyroid hormone synthesis, are also useful [10]. Our patient was treated with tab carbimazole and propranolol, and became euthyroid by 5th month of age. Babies at high risk for congenital hyperthyroidism due to maternal Graves` disease may require hospital monitoring for first few days, measuring FT4, FT3 and TSH from cord blood, and serial physical examination. FT4 and TSH should be measured between10 and 14 postnatal days also [8]. Neonatal Graves' disease tends to resolve spontaneously within 3–12 weeks as maternal thyrotropin receptor (TSHR) stimulating or blocking antibodies are cleared from the circulation.

CONCLUSION

Symptomatic neonatal hyperthyroidism should consider as a medical emergency. Prompt and adequate institution of treatment may prevent severe damage in the newborn. Graves` disease during pregnancy needs

to be monitored closely to avoid complications due to the free passage of antibodies through the placenta and the development of clinical manifestations of Graves` disease.

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