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Neonatal Thyroid Disease in Children of Hyperthyroid Mothers: A Case Series

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ABSTRACT

Introduction: Maternal Graves' disease (GD) can cause hyper or hypothyroidism in the newborn and its approach remains a challenge for physicians.

Case series: Two of the mothers had a late diagnosis of hyperthyroidism and treatment was initiated in the third trimester, without achieving thyroid eufunction during pregnancy. The third mother remained with normal thyroid function. Regarding neonates, neonate 1 presented neonatal thyrotoxicosis and received treatment, with complete resolution at six weeks old. Neonate 2, premature due to emergency cesarean section after severe maternal pre-eclampsia, presented central hypothyroidism. He remained asymptomatic and did not need replacement treatment, regaining normal thyroid function at one month old. The newborn of the mother with GD treated and controlled during pregnancy did not present alterations in thyroid function.

Discussion: Thyroid dysfunction in children of mothers with GD depends on the balance between stimulating and inhibitory antibodies to the TSH receptor, as well as on the effect of antithyroid drugs. Neonatal thyrotoxicosis is a rare but potentially severe disease. The main risk factor for its development is the presence of stimulating antibodies to the TSH receptor (TSI) in late pregnancy.

Conclusion: Early diagnosis and adequate treatment of GD in pregnancy are essential. If available, TSI measurement in the third trimester is crucial for decision-making, since it predicts a higher risk of developing neonatal GD.

INTRODUCTION

The prevalence of maternal hyperthyroidism due to Graves' disease (GD) ranges between 0.1 and 0.4% [1]. Maternal GD can cause hyper or hypothyroidism in the newborn and its approach remain a challenge for physicians. Neonatal thyrotoxicosis (NT) is a rare but potentially severe disease. We present 3 newborns of mothers with GD with different thyroid manifestations (neonates 1, 2 and 3).

CASE SERIES

Two of the mothers had poorly controlled pregnancies, with late diagnosis of hyperthyroidism and initiation of treatment in the third trimester, without achieving thyroid eufunction during pregnancy. The third mother is a patient with known GD, treated with propylthiouracil, who remained with normal thyroid function during pregnancy.

Regarding the neonates of mothers with uncompensated hyperthyroidism during pregnancy, we highlight relevant aspects below:

- Neonate 1 presented NT, clinically evidenced by tachycardia at the age of six days with a heart rate of 210 bpm, without hemodynamic repercussions. Biochemically, she presented a suppressed thyrotropin (TSH) value in cord blood, confirmed in peripheral blood with elevated thyroid hormone concentrations (fT4 > 7.7 ng/dl and fT3 > 32.55 pg/ml, [normal values by chemiluminescent immunoassay of microparticles fT4 0.70-1.48 ng/dl and fT3 1.72-3.54

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pg/ml, respectively]). She was treated with 0.5 mg/kg/day methimazole and 1 mg/kg/day propanolol with good response and complete resolution at six weeks.

- Neonate 2, 32 weeks premature due to emergency cesarean section for severe maternal preeclampsia, presented suppression of cord TSH and normal thyroid hormones, which progressed two weeks later to central hypothyroidism, with suppressed TSH and decreased fT4 (0.61 ng/dl, lower limit of normality 0.7 ng/dl). He remained asymptomatic without treatment and recovered normal thyroid function at one month old. TSH receptor stimulating inmunoglobulin (TSI) levels were high (4.40 IU/L, normal value by electrochemiluminescence < 1.75 IU/L).

The newborn of the mother treated and controlled during pregnancy did not present thyroid dysfunction.

DISCUSSION

Thyroid dysfunction in children of mothers with GD depends on the balance between stimulating and inhibitory antibodies to the TSH receptor, as well as the effect of antithyroid drugs, all of which have transplacental passage.

Fetal hyperthyroidism can cause abortion, death, and intrauterine growth restriction in fetuses of uncompensated mothers. Other manifestations include tachycardia, heart failure, hydrops, advanced bone age, craniosynostosis and microcephaly [2].

Regarding NT, its prevalence ranges from 1 to 5% [3]. The main risk factor is the presence of TSI antibodies in late pregnancy, even if the mother achieves normal thyroid function. Its elevation three times above the upper limit of normality in the last trimester predicts neonatal hyperthyroidism with a sensitivity and specificity of 100% and 43%, respectively [4]. TN is self-limited and its duration depends on the persistence of TSI in the neonate's circulation. It manifests clinically within days of birth, when the effect of antithyroid drugs received by the mother disappears, usually with a duration of one to two months and complete resolution at six months. Excess thyroid hormones in the neonatal period can generate acute complications (heart failure) or long-term sequelae (neurodevelopmental disorders), which confers high morbidity and mortality [5].

Occasionally, children of hyperthyroid mothers can develop hypothyroidism, both central and primary. Central, due to suppression of the hypothalamic-pituitary-thyroid axis as consequence of severe maternal hyperthyroidism. Primary, transient (passage of inhibitory antibodies to the TSH receptor or effect of intrauterine antithyroid drugs) or permanent (thyroid dysgenesis due to fetal exposure to high concentrations of maternal fT4) [1].

CONCLUSION

Early diagnosis and adequate treatment of GD in pregnancy are essential, reducing the risk of adverse outcomes. Close fetal and neonatal monitoring are mandatory and all possible thyroid dysfunctions should be taken into account. If TSI antibodies are available, their measurement in the third trimester is crucial for decision-making, since it predicts an increased risk of developing fetal and neonatal GD.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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