HYPERTHYROIDISM IN AN INFANT OF A MOTHER WITH AUTOIMMUNE HYPOTHYROIDISM WITH POSITIVE TSH RECEPTOR ANTIBODIES

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Background:

- Neonatal hyperthyroidism is a rare condition seen in infants born to mothers with Graves disease, with transplacental transfer of TSH receptor antibodies (TRAb) to the baby.
- Some patients have been shown to swing from hypothyroidism to hyperthyroidism as TSH receptor blocking and stimulating antibodies may coexist.
- Graves disease following Hashimotos's hypothyroidism has previously been reported with one individual continuing to be hypothyroid even in the presence of thyroid stimulating antibodies.



Objective:

To describe a rare clinical event of a neonate with severe Graves hyperthyroidism, born to a mother with autoimmune hypothyroidism .

Patients and Methods:

A baby boy born preterm at 35 weeks gestation had significant irritability, tachycardia and a suspicion of proptosis 36 hours after birth.

The mother was known to have autoimmune hypothyroidism, diagnosed at age ten and was taking levo-thyroxine replacement with normal thyroid function throughout this pregnancy. She had never been thyrotoxic. She also had pernicious anaemia and extensive vitiligo. The maternal grandmother had Graves disease and maternal aunt and great grandmother had a history of Hashimoto's thyroiditis.

Results:

The baby's thyroid function on day 3 demonstrated gross thyrotoxicosis, TSH<0.01mU/I (NR day 3 <10 mU/I), FT4 >77pmol/L (20-35) and FT3 15.4 pmol/L. TRab was elevated at 18.4 IU/L (<1.8).

The mother's TRab was high at 24.7 IU/L.

The baby was commenced on propranolol on day 7, with some symptomatic improvement, however thyroid hormones continued to rise. After endocrine consultation, on day 17 carbimazole(CBZ) was commenced, at 0.3mg/kg/day. Thyroid function normalized within ten days, CBZ was gradually tapered and medication was weaned by 7 weeks. He has remained euthyroid. His mother continues to require

replacement thyroxine.

The baby's mother therefore was tested for TRab.

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Table 1: Hormonal profile of patient

Age	TSH (mIU/L)	FT4 (pmol/L)	FT3 (pmol/L)	TRab	Medication	Rare of antibolist and means of the second sec
D3	< 0.01	>77	15.4	184	nil	Howe thurse
D7	<0.01		18		Propanolol started 1mg 6 hourly	thyrot mater
D17	<0.01	63	27.1		CBZ started @0.3mg/kg/d	Our fi follow
4 weeks	0.01	24	7.4		CBZ tapered to 0.2mg/kg/d	pregn
7 weeks	0.01	13	5.6		CBZ stopped	References: 1. Polak M, Legac I, V patient. Hormone res al. Graves' disease fo 1990;33(6):687-98.
10 weeks	0.86	11	5.2		nil	
3 months	1.52	10.1	6.7		nil	
7 months	1.52	15.3	7.6		nil	
1 year	3.01			<0.3	nil	
CE	BZ: Carbimazo	ole. FT4:10-25	omol/I, FT3- 4.2	2-83.pmol	Ι/L	
935P2	Thyroid					

Conclusion:

- We hypothesize that the mother initially had Hashimoto's thyroiditis which damaged the thyroid to the extent that later Trab positive Graves disease was unable to elicit thyrotoxicosis
- Rare cases of de novo development of TSH stimulating antibodies are described in patients on levothyroxine and might be an alternate explanation for our case.
- However, almost all infants reported with neonatal thyrotoxicosis were either de novo or associated with maternal history of active or treated Graves disease.
- Our findings have important implications for future follow up of this family and for management of future pregnancies.

.. Polak M, Legac I, Vuillard E, Guibourdenche J, Castanet M, Luton D. Congenital hyperthyroidism: the fetus as a patient. Hormone research. 2006;65(5):235-42. 2. Takasu N, Yamada T, Sato A, Nakagawa M, Komiya I, Nagasawa Y, et II. Graves' disease following hypothyroidism due to Hashimoto's disease: studies of eight cases. Clinical endocrinology. .990;33(6):687-98.





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