



Fetal Hyperthyroidism Secondary to Maternal Basedow–Graves' Disease

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Abstract

Fetal hyperthyroidism is a rare prenatal disease and can be life-threatening. The diagnosis is based on ultrasound in mothers with a history of Basedow–Graves' disease and elevation of thyrotropin receptor antibodies (TRAbs) levels. The treatment consists of antithyroid drugs. We present a mother with Basedow–Graves' disease, treated with radioactive iodine 16 years ago. She had an unplanned pregnancy at the age of 29 years, and an elevation of TRAbs (21 U/L) was found at the sixth week of pregnancy. At 22 weeks of gestation, fetal ultrasound displayed tachycardia, goiter, exophthalmos, and suspicion of craniosynostosis, hence methimazole was started. Concomitantly, suppressed maternal thyroid-stimulating hormone (TSH) was found. Her daughter was born at 33 + 6 weeks showing clinical and laboratory findings of hyperthyroidism. Consequently, treatment with methimazole was prescribed. Normal thyroid function was documented in the mother after giving birth. Clear explanation has not been found for the alteration of maternal TSH during pregnancy.

Keywords

- ▶ fetal hyperthyroidism
- ▶ Graves' disease
- ▶ hyperthyroidism
- ▶ fetal

Thyroid hormones are essential for fetal development during pregnancy, particularly for fetal neurogenesis.¹ At the 10th week of pregnancy, the synthesis of fetal thyroid hormones starts, prior to which the fetus depends exclusively on maternal hormones.² Around 20 weeks of gestation, fetal thyroid-stimulating hormone (TSH) receptors begin to respond to TSH and TSH receptor antibodies (thyrotropin

receptor antibody [TRAb]).³ TRAbs cross the fetoplacental barrier and act on fetal TSH receptors and fetuses of mothers who have a history of active Basedow–Graves' disease or treatment with thyroidectomy or radioiodine and currently being treated for hypothyroidism and who have high TRAb titers may develop hyperthyroidism after this period.^{2,3} Fetal hyperthyroidism is a rare and transient prenatal condition;

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however, it can have lethal consequences for the fetus if it is not investigated and treated immediately.⁴ Diagnosis is based on antenatal Doppler ultrasound from 20 weeks of pregnancy in at-risk mothers displaying signs of fetal thyroid dysfunction. The clinical and ultrasound manifestations are broad, ranging from tachycardia, goiter, growth retardation, and amniotic fluid volume alterations to more serious consequences such as microcephaly, craniosynostosis, hydrops fetalis, heart failure, premature birth, and stillbirth.^{4–7} Once the diagnosis of fetal or maternal hyperthyroidism is made, the primary treatment is antithyroid drugs.⁴

Case Report

A 29-year-old primigravid mother was diagnosed with Basedow–Graves’ disease at the age of 13 years and was treated with radioiodine at 14 years of age, and developed posttreatment hypothyroidism. She showed progressive dysthyroid orbitopathy and underwent decompressive surgery at 19 years of age. Lately, she was referred to endocrinology in the context of her thyroid history and unintended pregnancy. Previously, she had been treated with levothyroxine 150 µg/d. Laboratory workup showed elevated TRABs levels at the sixth week of pregnancy (21 U/L, normal value <0.1 U/L), with normal levels of thyroid function test during the first trimester of pregnancy (►Table 1). Repeated TRABs levels at 18 weeks remained positive. At 22 weeks of gestation, a fetal ultrasound was performed, which showed suggestive signs of fetal hyperthyroidism, including tachycardia, goiter with increased central vascular flow, exophthalmos, and suspected craniosynostosis. Concomitantly, a mild suppression in maternal TSH level was detected. TRAB typing was requested and the presence of stimulating antibody (thyroid-stimulating immunoglobulin) was confirmed. Methimazole 10 mg was started which was later increased to 20 mg according to ultrasound findings (►Table 1). Interruption of pregnancy by cesarean section at 34 weeks due to progressive manifestations of fetal hyperthyroidism was decided; however, the pregnant woman went into labor at 33 + 6 weeks.

At birth, her daughter had a weight of 2,660 g (–0.47 standard deviation [SD]), height of 47.5 cm (1.2 SD), head circumference of 31.5 cm (–0.714 SD), and Apgar 8–9–9. Her physical examination revealed tachycardia, exophthalmos, bilateral palpebral retraction, and goiter. Treatment with methimazole 0.38 mg/kg/d and propranolol 1.5 mg/kg/d was immediately started. Thyroid function tests showed suppressed TSH, elevated free T4 (FT4) and T3. TRABs levels were 9.187 IU/L. In the first hour of life, she presented respiratory distress syndrome, requiring continuous positive airway pressure for 15 hours, without hemodynamic instability. At 7 days of life, the dose of methimazole was increased to 0.68 mg/kg/d, progressing with slow normalization of thyroid tests. At 4 days of life, she presented hyperbilirubinemia with a cholestatic pattern, and urso-deoxycholic acid and vitamin E were prescribed. Her abdominal and brain ultrasound were normal. Thyroid ultrasound showed slightly heterogeneous hypoechoic diffuse goiter

Table 1 Maternal–fetal evaluations since the beginning of the pregnancy

	6 wk	12 + 4 wk	18 + 3 W	22 + 4 wk	24 + 3 wk	27 + 4 wk	30 + 2 wk	33 wk	33 + 6 wk	Normal reference ranges
GA										
TSH	0.291	0.524	1.2	0.22	0.015	0.02	0.0011			0.3–4.2 µU/ml
FT4	1.3	1.31	1.3	1.09	1.33	0.9	1.26			0.7–1.48 ng/dL
T4	10.3	9.81	10	11.4	12.9	11.2	10			<16 wk: 5.5–11.5 µg/dL >16 wk: 8.25–15.25 µg/dL
TRAB s	21.3		16.4		18.7 (TSl)					< 0.1 U/L
Treatment	LT4 150 µg	LT4 150 µg	LT4 150 µg	LT4 150 µg Methimazole 10mg	LT4 150 µg Methimazole 15 mg	LT4 150 µg Thiamazole 20 mg	LT4 150 µg Methimazole 20 mg		Preterm labor	
Fetal US		HR: 153 × min AF normal		Fetal tachycardia, goiter with central hypervascularization. Long bones > p90	Sinus tachycardia, goiter improvement	Fetal tachycardia, goiter, exophthalmos	Similar goiter, normocardia, exophthalmos	Suspected craniosynostosis, HR: 160 × min		
Mother’s physical examination						Exophthalmos, HR: 95 × min tremor (+)				

Abbreviations: AF, amniotic fluid; FT4, free thyroxine; GA, gestational age; HR, heart rate; LT4, levothyroxine; T4, thyroxine; TRABs, thyrotropin receptor antibodies; TSH, thyroid-stimulating hormone; TSl, thyroid-stimulating immunoglobulin.

without focal lesions. Skull X-ray ruled out craniosynostosis. At 17 days of life due to persistent elevation of FT4, methimazole dose was increased (0.83 mg/kg/d). She was discharged at 22 days of life.

In her follow-up at the age of 2 months 5 days, the infant showed resolution of cholestasis, normalization of thyroid function with an adequate growth and weight gain; therefore, methimazole, ursodeoxycholic acid, and vitamin E were suspended, and levothyroxine was prescribed due to transient hypothyroidism secondary to methimazole (►Table 2). Ophthalmology evaluation reported regression of palpebral retraction with persistence of mild exophthalmos and her fundoscopic examination was normal. Nonetheless, dysphagia was suspected due to history of choking with feeding and saliva. She was evaluated by neurology and central hypotonia and an increased in her head circumference were noted (►Fig. 1). Magnetic resonance imaging displayed triventricular hydrocephalus and type 1 Chiari malformation and she underwent endoscopic ventricular cisternostomy at 12 months of age. She progressed with psychomotor developmental retardation; speech being predominantly affected. Brain stem evoked response audiometry was normal. At the age of 15 months, she underwent a new neurologic procedure owing to pseudomeningocele and a plasty of a cerebrospinal fluid noncommunicating fistula in the subgaleal space. She has been receiving speech therapy and has shown improvement in this area. Her growth has been adequate during follow-up (►Fig. 1).

Discussion

The most common management scenario in children of mothers with maternal hyperthyroidism due to Graves' disease is neonatal hyperthyroidism with several cases being described in the literature. Due to the severe consequences of fetal hyperthyroidism,⁸ screening during pregnancy is mandatory in mothers with a history of active Graves' disease or those who have been treated with radioiodine and/or thyroidectomy.⁴ Laboratory workup with TRAbs and thyroid function levels immediately after pregnancy diagnosis is suggested in the literature.^{4,9} If TRAbs are negative, no further follow-up is required. Conversely, if TRAbs are positive, repeated levels should be requested at 18 to 22 weeks of pregnancy. If TRAbs remains positive at that time, new levels should be determined at 30 to 34 weeks of pregnancy. In addition, an obstetric ultrasound should be performed starting at 20 to 24 weeks of gestation every 4 to 6 weeks.⁹

To the best of our knowledge, 15 cases of fetal hyperthyroidism have been reported in the literature (►Table 3). Six of those cases had a history of maternal radioiodine treatment for Graves' disease several years prior to pregnancy. This is consistent with data showing that high levels of TRAbs have been observed 5 years after treatment with I-131, in contrast to thyroidectomy.¹⁰ In our case, TRAbs remained positive after 16 years of radioiodine therapy.

Regarding maternal thyroid function at the time of pregnancy diagnosis, our patient was receiving levothyroxine showing normal thyroid function, which was similar to another seven case reports (►Table 3).

Table 2 Clinical and laboratory follow-up of the newborn

Age	0	4 d	7 d	10 d	13 d	17 d	20 d	22 d	1 mo 5 d	1 mo 17 d	2 mo 5 d	2 mo 11 d	2 mo 25 d
GA						36+3						25 d	1 mo 9 d
Weight (g)	2,660	2,130	2,090	2,205	2,270	2,345	2,390	2,380	3,100	3,510	4,040		
Height (cm)	47.5									53.5	55		
TSH (mIU/mL)	0.027		<0.015					15	<0.01	<0.01	0.33	629	<0.01
FT4 (ng/dL)	5.59	6.37	5.07	3.63	3.22	3.48	3.03	2.27	1.0	0.7	0.4	0.54	1.7
T3 (ng/mL)	3.54	2.88	3.32	3.89	4.1		3.6		2.0	1.1	0.9	129 ng/dL	
TRabs (U/L)		9.187					6.88					0.19	
Therapy	Methimazole 0.5–0.5 mg (0.38 mg/kg/d) Propranolol 2–2 mg (1.5 mg/kg/d)	↓ Propranolol 1–1 mg due to bradycardia, start of ursodiol and vitamin E	↑ Methimazole 1–0.5 mg (0.7 mg/kg/d) Propranolol 2–2 mg	↓ Methimazole 1–1 mg (0.83 mg/kg/d) Propranolol 2–2 mg (1.67 mg/kg/d)				Propranolol suspension, Ursodiol 25 mg c/8 h (20 mg/kg/d)	↓ Methimazole 1–0.5 mg (0.48 mg/kg/d), ursodiol, vitamin E suspension	↓ Methimazole 0.5–0.5 mg (0.28 mg/kg/d)	Methimazole suspension	LVT 25 µg (6 µg/kg/d)	LVT suspension

Abbreviations: FT4, free thyroxine; GA, gestational age; LVT, levothyroxine; T3, triiodothyronine; TRAbs, thyrotropin receptor antibodies; TSH, thyroid-stimulating hormone. Note: References values: TRAbs (NR <1.5), LVT (0.78–2.19), T3 (0.97–1.7), and TSH (0.9–7.7).

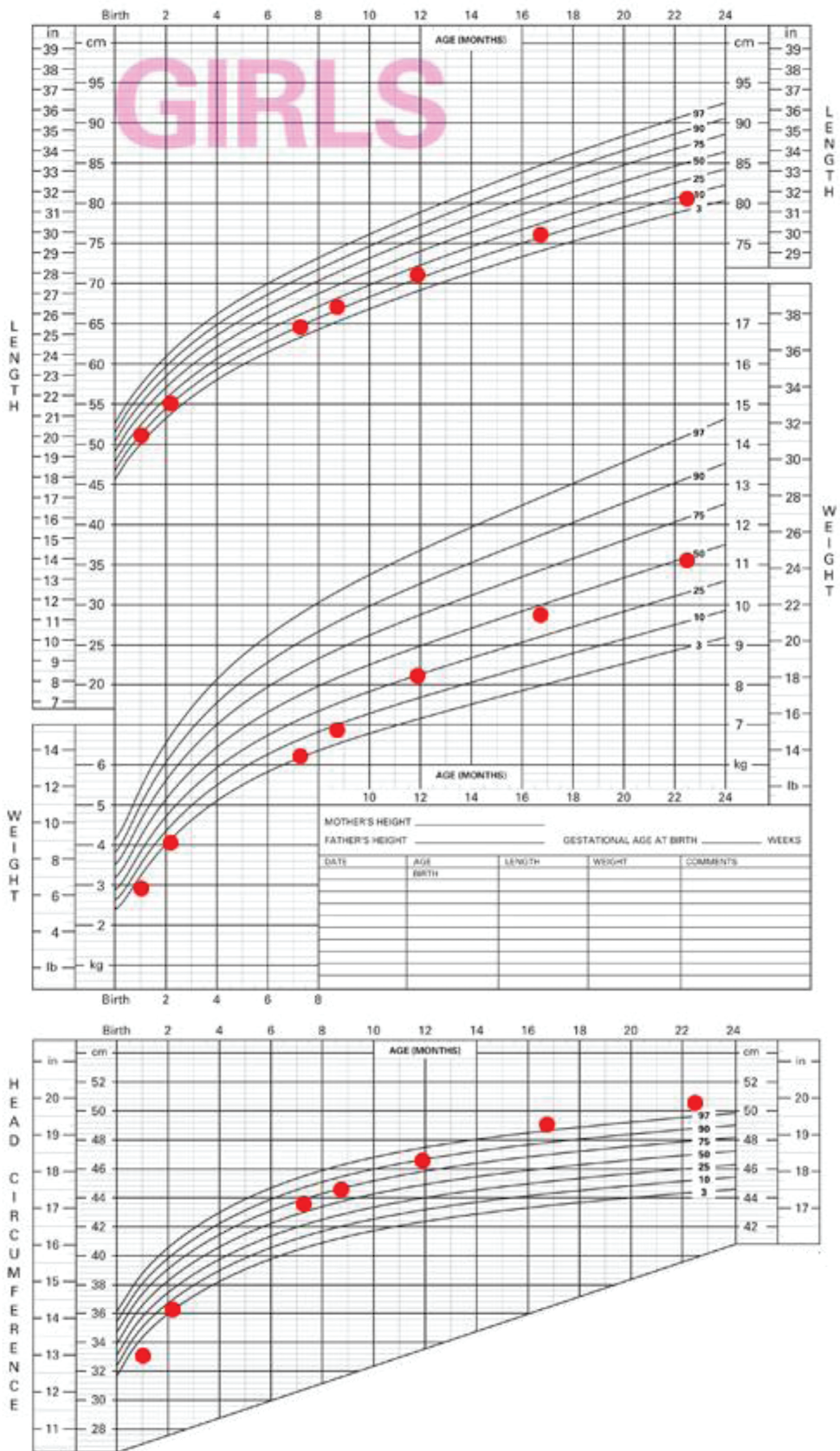


Fig. 1 Height, weight, and head circumference growth charts.

Table 3 Diagnosis and management of previous cases of fetal hyperthyroidism reported in the literature

Case report	Cases	Prenatal maternal treatment	Maternal thyroid function and/or TRAbs/TSI	Gestational age at diagnosis (wk)	Diagnosis	US diagnosis	Treatment
Srisupundit et al (2008) ¹¹	1	Thyroidectomy	Euthyroidism	28	US + FBS	Tachycardia	PTU
Lembet et al (2004) ¹²	1	PTU + propranolol	Hypothyroidism TRAb 46.7 U/I/L	26	FBS	Fetal goiter, IUGR, and oligohydramnios	PTU
Spoke and Martin (2020) ⁸	1	Radioiodine TT + desiccated thyroid extract and LVT	Hypothyroidism TRAb >40 U/I/L	17	US	Fetal goiter, fetal tachycardia, and pericardial effusion	MMI at week 23. PTU since week 25
Luton et al (1996) ¹³	2	Partial thyroidectomy	TRAb 297 U/I/L	24	FBS	Fetal goiter, pericardial effusion, and advance in bone maturation	PTU
Donnelly et al (2015) ¹⁴	1	Thyroidectomy + LVT	Euthyroidism TRAb 469 U/I/L	25	FBS	Fetal goiter, fetal tachycardia	PTU
Kazakou et al (2018) ¹⁵	1	TT + LVT	(-)	24 + 3	US	Fetal goiter, tachycardia, bone maturation acceleration in proximal and distal tibial epiphysis, and dilated cardiomegaly	PTU
Wallace et al (1995) ¹⁶	1	Radioiodine TT >3 y	Euthyroidism TSI > 500%	32	US + FBS	Fetal goiter, tachycardia, facial edema, pleural and pericardial effusion, IUGR, hydrops, oligohydramnios	MMI
Wenstrom et al (1990) ¹⁷	2	Radioiodine TT	Hypothyroidism TSI >500%	29	US	Tachycardia, IUGR, oligohydramnios	PTU
Hadi and Strickland (1995) ¹⁸	1	Radioiodine TT + PTU	Hyperthyroidism TSI: 500%	23	FBS	(-)	MMI
Porreco and Bloch (1990) ¹⁹	1	Thyroidectomy	Hyperthyroidism TSI: 470%	30	US + FBS	Fetal goiter, tachycardia	PTU
Vali et al (1993) ²⁰	1	Thyroidectomy	Euthyroidism TSI > 500%	25	FBS	Tachycardia	PTU
Belfar et al (1991) ²¹	1	Thyroidectomy	Hyperthyroidism TSI > 100%	22	US	Fetal goiter	Carbimazole
	1	PTU + propranolol	Euthyroidism	26	US	Tachycardia	PTU

Table 3 (Continued)

Case report	Cases	Prenatal maternal treatment	Maternal thyroid function and/or TRAbs/TSI	Gestational age at diagnosis (wk)	Diagnosis	US diagnosis	Treatment
Pekonen et al (1994) ²²	1	Thyroidectomy	Euthyroidism	31	US	Thyroid cyst, tachycardia	Carbimazole
Hatjis (1993) ²³	1	Radioiodine TT	Hypothyroidism TSI: 770%	26	US	Fetal goiter, tachycardia, IUGR, oligohydramnios	PTU
Heckel et al (1997) ²⁴	1	(-)	Hyperthyroidism TSI > 500%	28	US + FBS	Fetal goiter, tachycardia	Carbimazole

Abbreviations: FBS, fasting blood sugar; IUGR, intrauterine growth restriction; I/T, levothyroxine; MMI, methimazole; PTU, propylthiouracil; TRAbs, thyrotropin receptor antibodies; TSI, thyroid-stimulating immunoglobulin; TT, total thyroidectomy; US, ultrasound.

Ultrasound and measurement of maternal TRAbs were used as diagnostic methods for fetal hyperthyroidism in concordance with 12 others reports. However, cordocentesis has also been described as a diagnostic tool following ultrasound suspicion of fetal hyperthyroidism in most of the cases. In our case, cordocentesis was not performed due to maternal and fetal risks, fetal death being the most feared.²⁵

In our patient, diagnosis was based on ultrasound findings of fetal tachycardia, goiter with central hyperemia, exophthalmos, and suspicion of craniosynostosis, whereas in the cases previously reported (– Table 3), goiter was described in 6 fetuses and fetal tachycardia in 13. Furthermore, other signs observed were advanced bone maturation, oligohydramnios, intrauterine growth restriction, pleural effusion, pericardial effusion, among others.^{4–7}

Once diagnosis of fetal hyperthyroidism is confirmed, mothers should receive low-dose antithyroid drugs: propylthiouracil during the first trimester of pregnancy, transitioning to methimazole or carbimazole later. Fetal hypothyroidism is the most risky complication of this management.^{4,6,9}

In our case, due to suppressed maternal TSH without having modified levothyroxine dose concomitant with manifestation of fetal hyperthyroidism on the ultrasound, methimazole was started at 22 weeks of gestation. Abnormal levels of TSH were normalized postpartum. TSH suppression is rare in a pregnant woman with a history of hypothyroidism secondary to radioactive iodine ablation. Additionally, an improvement in clinical and laboratory manifestations during pregnancy has been reported in Th1-predominant autoimmune diseases in pregnancy, such as Graves' disease, which make the hypothesis of reactivation of maternal hyperthyroidism as a cause of this TSH level abnormality unlikely.²⁶

On the other hand, human chorionic gonadotropin-induced maternal TSH receptor activation could not explain the suppression of TSH in our patient since this phenomenon began in the second trimester of pregnancy.²⁷

Other less common causes of TSH suppression in pregnancy are those related to autonomous secretion of thyroid hormones, destruction of thyroid follicles, and extrathyroidal sources of thyroid hormones.²⁸ These were also ruled out, considering the absence of a history of nodular goiter, viral or bacterial infection, anterior cervical pain, among others.

We hypothesized that the suppression of maternal TSH was due to a transplacental transfer of thyroid hormones from the fetus to the mother. This was suspected since TSH values began to fall concomitantly with the onset of ultrasound manifestations of fetal hyperthyroidism at the time when fetal thyroid hormone secretion begins, especially considering that TSH suppression was corrected postpartum.

Monocarboxylate transporter 8 could play a fundamental role in the transport of thyroid hormones of fetal origin toward the maternal circulation. This transporter is present in the placenta in both apical and basal membranes of maternal and fetal endothelial cells, in addition to the villous stroma and a potent and specific affinity for T3 has been described.²⁹

Another mechanism that may support the theory of bidirectional transport is reverse T3 (T3r), which is

synthesized by the fetal thyroid and is transported from the fetus to the maternal circulation through the placenta,³⁰ showing 40 times greater affinity for placental nuclear binding sites than T4 and 63 times greater than T3.³¹ However, it has been observed that T3r is functionally inactive and does not generate serum TSH modifications,³² so if T3r played any role, it would be indirect and due to other mechanisms not yet established.

In summary, no clear explanation was found for the suppression of TSH during pregnancy in our patient. Further studies addressing transport or metabolism mechanisms of thyroid hormones are needed.

Conclusion

Health care providers must be alert to the eventual development of fetal hyperthyroidism in any mother with a history of GBD, even when she has received definitive treatment (surgery or radioiodine) several years before pregnancy, since the production of TRAbs can persist for a long time. Therefore, it is important that the entire care team for a pregnant woman inquire about maternal thyroid history and apply a protocol for monitoring TRAbs and ultrasound and, if necessary, treatment with antithyroid drugs.

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Conflict of Interest

None declared.

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