

Congenital Hypothyroidism Presenting with Postpartum Bradycardia

Semra Kara¹, Cüneyt Tayman¹, Alparslan Tonbul¹, Nesibe Andiran², Mansur Tatli¹ and Sadi Türkay³

ABSTRACT

Congenital hypothyroidism is a clinical condition characterized by lack of thyroid hormone because of thyroid gland developmental and thyroid hormone biosynthesis disorders. The most common cause of permanent hypothyroidism is congenital factors. Prompt diagnosis is critical. However, overt signs of hypothyroidism are rarely present at birth, and 95% of affected babies are asymptomatic. Hypoxemia, apnea, acidosis, increased intracranial pressure, vagal stimulus and central nerve system abnormalities represent the most common causes of bradycardia in the neonate. Bradycardia associated with congenital hypothyroidism is very rare. In this paper, a case of severe congenital hypothyroidism, induced by maternal blocker antibodies, who presented with bradycardia, is reported.

Key words: Congenital hypothyroidism. Maternal antibodies. Bradycardia. Neonate.

INTRODUCTION

Congenital hypothyroidism (CH) is a deficiency in thyroid hormone present at or before birth.¹ Newborn screening for hypothyroidism was first performed and consequently established in 1972 in Canada to permit early identification of infants at risk and allow prompt institution of thyroid hormone replacement therapy.^{1,2} It is the most prevalent endocrinological problem in the neonatal period and affects 1 in 3,000 – 4,000 newborns.³ Defective embryogenesis of the thyroid accounts for 80 – 90% of non-endemic congenital hypothyroidism.⁴ Female to male ratio is about 2:1, as detected by neonatal screening programs.² Prompt diagnosis is critical because a delay in treatment can lead to irreversible brain damage or cretinism.¹

Overt signs of hypothyroidism are rarely present at birth, and 95% of the affected babies are asymptomatic.⁵ Early manifestations include lethargy, inactivity, hypotonia, periorbital oedema, large anterior and posterior fontanelles, feeding difficulty, respiratory distress, pallor, prolonged icterus, perioral cyanosis, mottled skin, poor or hoarse crying, constipation, and hypothermia. Classic features of congenital hypothyroidism in the infant usually become apparent after about 6 weeks of age. These include typical facies, thick, dry, cold skin, coarse hair, abdominal distention, umbilical hernia, bradycardia. Hypoxemia, apnea, acidosis, increased intracranial pressure, vagal stimulus and central nerve system abnormalities represent the most common causes of

bradycardia in the neonatal period.⁶ Bradycardia associated with congenital hypothyroidism is very rare.

CASE REPORT

A male infant born to a 27-year-old mother (G1P1) with caesarean section at week 38, day 3 with a birth weight of 3260 g was referred to our hospital with bradycardia with an onset at postnatal 2 hours. His Apgar score was 9/10 (1 and 5 minutes). Prenatal history involved IVF pregnancy and the mother was on L-Thyroxine 4 therapy for hypothyroidism for the past two years and a half. The physical examination did not yield any relevant findings except for a posterior fontanelle gap of 3 x 2 cm and peak cardiac output of 72/minute. All biochemical, infection and blood gas parameters as per laboratory analyses were well within normal ranges. The ECG demonstrated a sinus bradycardia, cQT of 0.42 seconds and PCA of 74/minute with no blockade. Cranial ultrasound (USG) yielded normal results. No major abnormalities were noted with echo.

Thyroid function tests (TFT) yielded the following values; TSH: > 502 µIU/ml (0.5-20), FT4:5.2 pmol/L (18 – 40), TT4: 3 µg/dL (11.8 – 22.6). USG of the thyroid showed right lobe size to be 14 x 10.5 x 28 mm and left lobe to be 14 x 11 x 27 mm, with homogenous parenchyma.

Patient's antithyroglobulin was 1+, AntiTPO 2+, (0-58), TSH receptor blocker antibody (TRAB): 19 U/L (0-10) and thyroglobulin 926 ng/ml. Femur distal epiphysis was not present in the radiography of the knee. TFT analysis of the mother yielded the following; TSH: 3.4 µIU/ml, TT4:12µg/dL, FT4:14 pmol/L. Anti-thyroglobulin: 3+, AntiTPO: 3+, TRAB: < 2.4 U/L. treatment with oral L-thyroxine 10 µg/kg/day was started. On the second day of treatment, breath per minute (BPM) increased upto 120/minute. The follow-up of the patient a week later showed that BPM of 140/minute and TSH: 150 µIU/ml,

Division of Neonatology¹/ Endocrinology²/ Cardiology³,
Fatih University, Medical Faculty, Yenimahalle Ankara, Turkey.

Correspondence: Dr. Semra Kara, Neonatology Unit, Fatih
University Medical Faculty, Yenimahalle Ankara, Turkey.
E-mail: dr.semprakara@hotmail.com

Received January 21, 2012; accepted June 05, 2012.

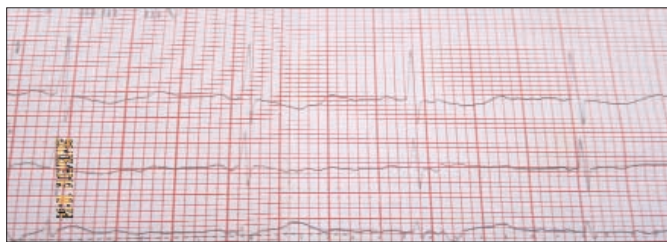


Figure 1: ECG of patient before treatment.



Figure 2: ECG of patient after treatment.

FT4: 0.9 ng/dL (0.85 – 1.98), TT4: 8.6 µg/ml (3.2-12.6) with no abnormal findings on examinations. Patients is still being followed.

DISCUSSION

Hypothyroidism is a clinical condition characterized by thyroid hormone deficiency which may be associated with developmental disorders of the thyroid gland or disorders of thyroid hormone bio-synthesis and iodine deficiency as well as with some certain drugs (e.g., anti-thyroid preparations) used by the mother.⁶ The most common cause of permanent hypothyroidism is congenital factors.⁶ Severity of the symptoms and physical manifestations are correlated with the degree of hypothyroidism.⁶ CH is the most common preventable cause of mental retardation. Right after delivery, TSH levels increase due to hypothermia and stress, and then decrease gradually over the next few days. Increases in TSH levels is followed by increases in T4 and T3 levels and hormone levels generally increase above the upper limit of normal during the first 24 hours. This is called physiologic hyperthyroidism. This should be considered when interpreting the thyroid hormone results during the neonatal period.⁶ TSH or T4 monitoring should, therefore, be performed on the postnatal 3-5th days. The incidence of permanent congenital hypothyroidism in Turkey was reported as 1/3.344 based on a comprehensive screening of 187.728 neonates.⁷ The most common cause of congenital hypothyroidism is iodine deficiency.⁸ In regions with low prevalence of iodine deficiency, the most common cause is thyroid dysgenesis. Less frequently, blocker antibodies which pass from the mother may also result in congenital

hypothyroidism.⁹ Heart rates below 80/minute in a term infant is considered bradycardia. Hypoxemia, apnea, acidosis, increased intracranial pressure, vagal stimulus and central nerve system abnormalities represent the most common causes of bradycardia in the neonatal. More rarely, hypothermia, digoxin intoxication, sinus node dysfunction and hypothyroidism lead to bradycardia in this population.⁶

Total T4 is reduced and TSH serum concentration is increased in congenital hypothyroidism. Congenital hypothyroidism secondary to maternal blocker antibodies and presence of bradycardia as the evidence of congenital hypothyroidism are observed in very rare cases.⁹ This case presented here is a severe congenital hypothyroidism which developed as a consequence of maternal blocker antibodies that, most likely, have also affected the intrauterine period of the infant, with bradycardia as the initial finding. Therefore, in the differential diagnosis of neonates who present with bradycardia, hypothyroidism should be considered, and it should be borne in mind that the etiology of established hypothyroidism may involve blocker antibodies.

REFERENCES

1. LaFranchi SH, Austin J. How should we be treating children with congenital hypothyroidism? *J Pediatr Endocrinol Metab* 2007; **20**:559-78.
2. Rose SR, Brown RS, Foley T. Update of newborn screening and therapy for congenital hypothyroidism. *Pediatrics* 2006; **117**: 2290-303.
3. Grüters A, Biebermann H, Krude H. Neonatal thyroid disorders. *Horm Res* 2003; **59**:24-9.
4. Knobel M, Medeiros-Neto G. An outline of inherited disorders of the thyroid hormone generating system. *Thyroid* 2003; **13**: 771-801.
5. Kappy MS, Blizzard RM, Migeon CJ, editors. Wilkins the diagnosis and treatment of endocrine disorders in childhood and adolescence. 4th ed. Springfield: *Charles C Thomas Pub* 1994.
6. Susan RR. Thyroid disorders. In: Martin RJ, Fanaroff AA, Walsh MC, editors. *Fanaroff and Martin's text book of neonatal - perinatal medicine*. 9th ed. Philadelphia: *Mosby* 2011; p. 1556-84.
7. Yordam N, Ozon A. Neonatal thyroid screening: methods-efficiency-failures. *Pediatr Endocrinol Rev* 2003; **1**:177-84.
8. LaFranchi S. Hypothyroidism. In: Kliegman RM, Stanton BF, ST. Geme JW, Schor NF, Behrman RE, editors. *Nelson textbook of pediatrics*. 19th ed. Philadelphia PA, *WB Saunders* 2011; p. 1895-03.
9. Brown RS, Bellasario RL, Botero D. Incidence of transient congenital hypothyroidism due to maternal thyrotropin antibodies in over one million babies. *J Clin Endocrinol Metab* 1996; **81**:1147-51.

....☆....