

Case Report

Neonatal Thyrotoxicosis Presenting with Heart Failure

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Abstract

Thyrotoxicosis in the newborn is usually the result of the passage of thyroid stimulating antibodies from the mother to the fetus. This is a case report of a ten-day-old male neonate who presented with signs of neonatal thyrotoxicosis and heart failure. He had a high T3 ($>7.7\text{nmol/l}$), high T4 ($>300\text{nmol/l}$), and low TSH (0.14 uIU/ml). He was hospitalized at the neonatal intensive care unit of Myungsung Comprehensive Specialized Hospital and was successfully treated with propylthiouracil, propranolol, furosemide, lugols iodine, hydrocortisone, and supportive care. Clinical features and management of neonatal thyrotoxicosis are discussed with a literature review.

Keywords: Thyrotoxicosis, newborn, heart failure

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Case report

This is a ten-day-old male neonate delivered at Myungsung Comprehensive Specialized Hospital to a 28-year-old primi para mother at a gestational age of 39 weeks and 2 days. The mother is known to have Graves' disease and has been on propylthiouracil (PTU) for the last two years.

It was a normal vaginal delivery with a birth weight of 3685 grams. Neonatal examination was normal except for the presence of anterior neck mass. A thyroid function test done at birth from venous blood revealed a normal T3 (1.68nmol/l), low T4 (24.2nmol/l), low free T4 (4.37 pmol/l), and high TSH ($>60\text{uIU/ml}$). He was appointed for follow-up in the outpatient clinic. On the tenth day of life, he presented with irritability and excessive crying. He had frequent watery stool. On examination, he was tachycardic (210 beats/min), tachypneic (100 breaths/min), Spo2 was low (71%) and restless. He had lost 550g from his birth weight. Laboratory tests showed; high T3 ($>7.7\text{nmol/l}$), high T4 ($>300\text{nmol/l}$), high free T4 ($>100\text{pmol/l}$), and low TSH (0.14 uIU/ml). CBC and CRP were in the normal range. Ultrasonography of the neck showed a diffusely enlarged thyroid gland with markedly increased flow on doppler study. Chest x-ray showed cardiomegaly with normal pulmonary vasculature (figure 1). He was provided intranasal oxygen and managed with propylthiouracil, propranolol, furosemide, lugols iodine, and hydrocortisone. On the 2nd day, his oxygen

demand progressively increased and could not maintain his saturation with a high liter of oxygen using face a mask. He was intubated and was on mechanical ventilation. He showed gradual improvement with normalization of heart rate and reduced oxygen demand and was successfully extubated after 48 hours. On the sixth day of hospitalization, the serum level of T3 and T4 had normalized but TSH was low. Patient was discharged after seven days of hospitalization in a stable condition with a follow-up arrangement at an outpatient clinic.

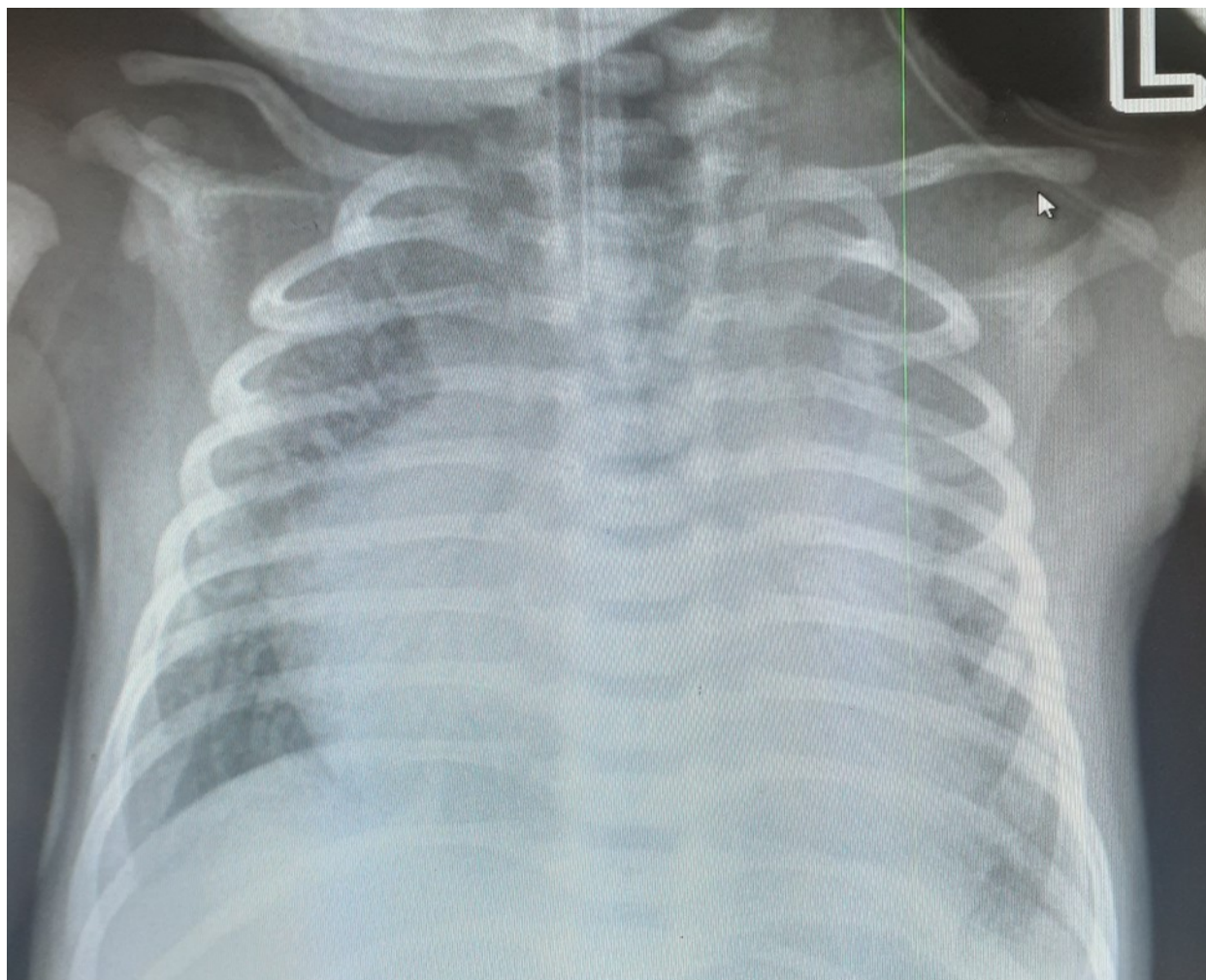


Figure 1 Chest x-ray of the infant taken on the 11th day of life showing cardiomegaly

Discussion

Thyrotoxicosis in the newborn is usually the result of the passage of thyroid-stimulating antibodies from the mother to the fetus towards the end of pregnancy. Women with Graves' disease have Thyroid Receptor Antibodies (TRSAbs) which are usually the cause of this disease. Approximately 0.2% of pregnant women have Graves' disease, and between 1% and 12.5% of their offspring are expected to have overt thyrotoxicosis. Because the mother's TRSAbs readily crosses the placenta, TRSAb transfer to the fetus may result in hyperthyroidism in utero and/or after birth (1). Manifestations of neonatal thyrotoxicosis persist in the newborn until the maternal antibodies disappear from its circulation (2, 3, 4).

The presence of thyrotropin receptor-blocking antibodies and the transplacental passage of antithyroid drugs taken by the mother may modify the onset, severity and course of the disease. Manifestation of

neonatal hyperthyroidism is delayed by 3-4 days if the infant has been exposed to antithyroid drugs, as the maternally derived antithyroid drug is metabolized (5). Manifestation in newborns typically begins during the first one to two weeks (6).

This newborn didn't have clinical manifestations and laboratory findings suggestive of thyrotoxicosis for the first 9 days which could be due to the effect of propylthiouracil which was taken by the mother. However, as the effect of PTU disappeared the newborn started to manifest signs of hyperthyroidism.

The main characteristic signs and symptoms of neonatal thyrotoxicosis include tachycardia, irritability, prominent eyes, and poor weight gain. If there is goiter it may be related to maternal antithyroid drug treatment as well as to the neonatal Graves' disease itself. If treatment is delayed or inadequate it may result in ar-

rhythmias and cardiac failure which may cause death. Without treatment it is associated with deleterious long-term consequences, including cranial synostosis, developmental delay and failure to thrive (6).

This newborn had typical manifestations including tachycardia, tachypnea, irritability, weight loss, diarrhea, goiter and heart failure with an enlarged heart and requiring management of heart failure.

The treatment of neonatal thyrotoxicosis is mainly by providing antithyroid medications like propylthiouracil or methimazole which inhibits the synthesis of thyroid hormone. The addition of Lugol or potassium iodide solution is recommended for severe cases as it avoids the entry of thyroid hormones into the circulation (7). Steroids, which prevent the production of thyroid hormone and peripheral transformation of T4 to T3, are reserved for severe cases (8). Propranolol effectively restores a normal heart rate (9).

This newborn was treated with antithyroid drugs, propylthiouracil, propranolol, and steroids in addition to the supportive care given for heart failure.

At presentation the patient's condition was grave; however urgent administration of medications, supportive

care, and meticulous follow-up saved his life.

Conclusion

Early identification of infants at risk of neonatal thyrotoxicosis and prompt initiation of treatment is life-saving.

Consent

Patient's parents gave verbal consent for the case study.

Competing interests

There was no funding for the study and no conflicts of interest to disclose.

Acknowledgement

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