CASE REPORT

Congestive cardiac failure as a presentation of neonatal Graves in twin

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ABSTRACT

Neonatal Graves is a rare entity and neonatal thyroid storm is even rarer. Graves disease is an autoimmune disorder with production of thyroid stimulating immunoglobulin (TSI) resulting in diffuse toxic goiter. Neonatal thyrotoxicosis presents with hyper-excitability, poor weight gain, tachycardia. We report a case of congestive cardiac failure with paroxysmal supraventricular tachycardia as a presentation of neonatal Graves in twin babies.

Key Words: Congestive cardiac failure, Graves’s disease, paroxysmal supraventricular tachycardia, thyroid storm

Introduction

Neonatal Graves occurs only 1 in 70 cases of thyrotoxic pregnancies (1). In most cases neonatal disease is due to transplacental passage of thyrotropin receptor stimulating antibody (TRSAb). Neonatal thyrotoxicosis usually present with hyper excitability, poor weight gain, tachycardia (2). Rarely it may manifest with cholestatic jaundice, hyperammonemia, thrombocytopenia, hepatosplenomegaly (3,4). Congestive cardiac failure following paroxysmal supraventricular tachycardia in neonatal Graves is hitherto unreported.

Case report

Twin babies were born by vaginal delivery to a 25 year-old primipara woman. Mother was diagnosed to have Grave’s disease antenatal. It was a preterm labour at 34 week of gestation. Mother was diagnosed to have Graves disease at 5 month of pregnancy and was on carbimazole (5mg twice daily) and propranolol 10 mg tablet daily since then. Mother had a goiter and exophthalmos as the disease was poorly controlled because of poor compliance (Figure 1). Mother had no other complications. On examination, 1st twin, girl weighing 2.1 kg, appropriate for gestation and did not have birth asphyxia. During admission after birth this baby appeared to be extremely restless, irritable, hyperactive and unusually alert with wide opened eye (Figure 2). Baby had heart rate of 160-170/min, respiratory rate of 70/min, mean arterial pressure of 51mm of Hg. She had no visible thyromegaly. Within 8 hours of life, baby became more tachypneic and had heart rate was 230-250 /min with capillary refilling time of < 3 seconds. Liver was palpable 2.5 cm. During stay at neonatal intensive care unit (NICU) baby developed unconjugated hyperbilirubinemia on 3rd day. Investigation of 1st twin revealed negative sepsis screen with blood sugar of 80 mg/dL, sodium 140 mEq/dL, potassium 3.6 mEq/dL and calcium 9.5 mg/dL. Echocardiography (ECG) showed paroxysmal supraventricular tachycardia. ECG showed no structural heart defect. On third day of life her bilirubin level was 12.5mg/dl (unconjugated-12mg/dl). Hormonal estimation measurements were as follows; TSH-0.09 micro unit /ml (normal cord blood and neonatal value -<25), T4-24.86 ng/dL (normal upto 3 days of life 8.2-19.9), T3-2.39 ng/dL (normal in neonate-0.75-2.6), TRSAb -160 5U/L (>14U/L-Positive). 2nd twin, girl, weighing 1.7 kg, small for gestation was hyperactive and had alert look with wide opened eyes. At 8hrs of life baby had heart rate 220/min and respiratory rate 80/min, mean arterial pressure of 50 mm of Hg with capillary refilling time of <3 seconds. She also had no goiter. Investigation revealed negative sepsis screen, blood sugar of 98 mg/dL, sodium 140 mEq/dL, potassium 4 mEq/dL and calcium 9.3 mg /dL. ECG showed paroxysmal supraventricular tachycardia. There was no structural defect in echocardiography. On 3rd day investigations revealed TSH-0.04 microunit/mL, T4-21.98 ng/dL, T3-2.25 ng/dL, TRSAb-156.5 U/L.

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Mother had TSH-0.001 microunit/mL, T4-15 ng/dL, T3 2.9 ng/dL at 5th month of gestation. TRSAb was very high-286 U/L. USG of thyroid gland showed-diffuse goiter, radionuclide thyroid scan revealed-increased homogenously distributed radioiodine uptake. Hence, our diagnosis of neonatal Graves disease with thyroid storm was confirmed in twin babies. In these cases babies had congestive cardiac failure at birth following neonatal Graves. Paroxysmal supraventricular tachycardia resolved with 2 doses of rapid bolus adenosine (0.1 mg/kg/dose) in both. But they still had tachypnoea with heart rate of 170 180/min with signs of heart failure. During first few days we introduced frusemide intravenously (1mg/kg/dose twice a day) for controlling cardiac failure symptoms. We started carbimazole to both babies (0.5 mg/kg/day in 3 divided doses) and propranolol 1mg/kg/day along with close monitoring of vital parameters. 2nd twin received antibiotic in addition. Within 48 hours of starting antithyroid therapy heart rate and respiratory rate became normalized. We discharged the babies with carbimazole therapy and asked for follow up after 2 weeks for monitoring weight gain.

Discussion

Causes of congestive cardiac failure at birth usually occurs due to congenital heart disease like hypoplastic left heart syndrome (HLHS), severe tricuspid and pulmonary insufficiency and critical aortic stenosis etc (5). Perinatal asphyxia with myocardial injury, and neonatal arrhythmia may also present with congestive cardiac failure. Estimated incidence of SVT in neonate is approximately 1 in 25000 (6). The common causes are sepsis, hyperthermia, hypoglycemia, myocarditis etc, congenital heart disease. PSVT due to neonatal thyrotoxicosis is extremely rare (6). Neonatal hyperthyroidism occurs in only 2% of newborns born to mothers with a history of Graves’s disease (7). The finding of very high level of TRSAb in these mothers usually predicts the occurrence of affected newborns. Very high level of TRSAb usually results in classical neonatal hyperthyroidism as in our cases. Most babies are premature as well as small for date as in our cases. Classical neonatal Graves also have exophthalmos, tachypnoea, tachycardia, hypertension alert look etc. as in our case. Only one case series of 32 pregnancies with maternal thyrotoxicosis done in AIIMS showed one affected newborn with thyrotoxicosis and goiter (8). As per report by Zakarija et al, (9) chance of neonatal thyrotoxicosis is 100% when mother have TRSAb titre >500% of normal. In a newborn with congestive cardiac failure and hyper alert look possibility of neonatal thyrotoxicosis should be ruled out.

References

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