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Case report
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Unusual concomitance during the neonatal period: Tachyarrhythmia and hypothyroidism

Yenidoğan döneminde olağandışı birliktelik: Taşiaritmi ve hipotiroidizm

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Abstract
Congenital hypothyroidism is a significant issue in neonates. A variety of cardiac abnormalities have been described in cases of severe hypothyroidism. It is commonly associated with sinus bradycardia, low QRS complexes, prolonged QT interval and conduction blocks but may rarely cause arrhythmias. Although supraventricular arrhythmias are ordinary features of hyperthyroidism, the aim of this report is to underline the possible etiological link between supraventricular tachycardia and hypothyroidism. Although a few adult cases with hypothyroidism and tachyarrhythmia have been previously reported in the literature, to our knowledge, these are the only neonatal cases reported.
Keywords: Neonate, Hypothyroidism, Tachycardia

Öz
Konjenital hipotiroidizm yenidoğanlarda önemli bir sorundur. Şiddetli hipotiroidizm vakalarında çeşitli kardiyak anormallikler tanımlanmıştır. Hipotiroidizm yaygın olarak sinüs bradikardisi, düşük QRS kompleksleri, uzun QT aralığı ve iletim blokları ile ilişkilidir, ancak nadiren aritmilere neden olabilir. Supraventriküler aritmiler hipertiroidizmin sıradan özellikleri olmasına rağmen; Bu çalışmanın amacı supraventriküler taşikardi ve hipotiroidizm arasındaki olası etyolojik bağın altını çizmektir. Literatürde hipotiroidizm ve taşiaritmi birlikteliği olan birkaç erişkin olgu daha önce bildirilmiş olmasına rağmen, bilgimize göre yenidoğanlarda bildirilen vakalar sadece bunlardır.
Anahtar kelimeler: Yenidoğan, Hipotiroidizm, Taşikardi

Introduction

Thyroid hormones are important for cardiovascular function. In case of decreased thyroid function, neither the heart nor the blood vessels function normally [1].

A variety of cardiac abnormalities, consisting of functional, structural, and electrical conduction problems, have been described in cases of severe hypothyroidism [2]. Electrocardiographic changes are commonly recognized; however, sustained, or life-threatening ventricular ectopy is rarely seen [3].

Although a few adult cases with hypothyroidism and tachyarrhythmia have been previously reported in the literature, to our knowledge, these are the only neonatal cases reported.

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Case presentation

Parents gave written informed consent for their children.

Case 1

A female infant of non-consanguineous parents was born via vaginal delivery at 30 weeks of gestational age. Apgar scores were 7 and 9 at 1st and 5th minutes of life, respectively. She received ventilator support. On admission, she had supraventricular tachycardia. Electrocardiography revealed that the heart rate was 200 beats/min with narrow QRS complexes. Adenosine was effective in inducing a sinus rhythm, however as supraventricular tachycardia recurred (with 220 beats/min), the patient was digitalized. Echocardiography yielded no pathologic findings. On the 4th and 5th day of life she had recurrent supraventricular tachycardia attacks, which were treated with adenosine and digitalization. Propranolol treatment was added to digoxin on the 3rd day of life. As digoxin level was high, it was ceased on the 10th postnatal day.

Hematological parameters, BUN, creatinine, electrolytes were within normal limits. On 5th day of life, thyroid stimulating hormone level was 438.12 mIU/ml and free thyroxine was 0.73 pg/ml. Thyroid ultrasonography was normal. Levothyroxine treatment was started at a dose of 10 µg/kg. The mother was euthyroid and had no autoimmune diseases.

Supraventricular tachycardia attacks did not recur after the 10th postnatal day. She was discharged on the 42nd postnatal day with propranolol treatment. Hyperthyroidism was detected during follow-up, and levothyroxine treatment was stopped at 60 days of life. Supraventricular tachycardia did not continue, and propranolol was stopped at 90 days of life. She is now 17 months old and uneventfully observed in the outpatient clinic without any medication.

Case 2

The male infant of healthy consanguineous parents was born on the 33rd gestational week by caesarean section due to acute fetal distress. Bradycardia and tachyarrhythmia were detected during monitorization in the NICU. On the 7th postnatal day, he was referred to our hospital for arrhythmia. Body weight was 1,800 g (50%), length was 47 cm (75-90%). Echocardiography yielded no pathologic findings. He was monitored on Holter for 24 hours, which revealed atrial tachyarrhythmia with atrioventricular block. On electrocardiography, heart rate was 220-230 beats/min with narrow QRS complexes. Propafenone (10 mg/kg/d) was started on 10th day of life. WBC, BUN, creatinine, blood glucose and electrolytes were within normal limits. On the 8th day of life, thyroid stimulating hormone level was 58.8 mIU/ml and free thyroxine was 0.63 pg/ml. Thyroid ultrasonography was normal. Levothyroxine treatment was started at a dose of 10 µg/kg. The mother was euthyroid and had no autoimmune diseases.

He was discharged with propafenone and levothyroxine treatments on the 23rd postnatal day. At 7 months of age, propafenone was stopped during follow-up. He had no tachyarrhythmia on Holter monitoring. He is now 15 months old and still using levothyroxine.

Case 3

The male infant of non-consanguineous parents was born via C/S at 35 weeks of gestational age. Fetal arrhythmia was diagnosed at the 32nd gestational week. Apgar scores were 4 and 6 at 1st and 5th minutes of life, respectively. He was intubated in the delivery room due to resistant bradycardia. The mother had a cardiac pacemaker due to Brugada Syndrome. Body weight and length were 3,280 g (75-90%) and 49 cm (75-90%), respectively. Auscultatory findings of the lungs were normal. On admission, he had supraventricular tachycardia. On electrocardiography, heart rate was 260 beats/min with narrow QRS complexes. Adenosine was not effective in inducing a sinus rhythm. Atrial flutter was observed, after which amiodarone was administered on the first postnatal day. Propranolol was added on the 4th postnatal day to amiodarone. On the 7th postnatal day, he was extubated. Echocardiography yielded no pathological findings. On the 8th postnatal day, thyroid stimulating hormone level was 62 mIU/ml and free thyroxine level was 0.38 ng/dl. Thyroid ultrasonography was normal. Levothyroxine treatment was started at a dose of 10 µg/kg. The mother was euthyroid and had no autoimmune diseases. Supraventricular tachycardia and atrial flutter attacks did not recur after the 10th postnatal day. He was discharged on the 13th postnatal day with propranolol and levothyroxine.

Case 4

The male infant of non-consanguineous parents was born via C/S at 32 weeks of gestational age. Apgar scores were 5 and 7 at 1st and 5th minutes of life, respectively. He was intubated in the delivery room due to respiratory distress and surfactant was administered. Body weight and length were 2,500 g (75-90%) and 47 cm (75-90%), respectively. On admission, he had supraventricular tachycardia. On electrocardiography, heart rate was 240-260 beats/min with narrow QRS complexes. Adenosine was not effective in induction of a sinus rhythm, and synchronized cardioversion was performed on the first postnatal day. After return to normal sinus rhythm following cardioversion, maintenance digoxin was started. On the 5th postnatal day, he was extubated. Echocardiography yielded no pathologic findings. On the 7th postnatal day, thyroid stimulating hormone level was 89 mIU/ml and free thyroxine level was 0.3 ng/dl. Thyroid ultrasonography was normal. Levothyroxine treatment was started at a dose of 10 µg/kg. The mother was euthyroid and had no autoimmune diseases. Supraventricular tachycardia occurred on the 15th postnatal day and adenosine was ineffective, so esmolol infusion was started. As esmolol was gradually stopped, propranolol was administered at the 16th postnatal day. He was discharged on the 25th postnatal day with propranolol and levothyroxine.

Discussion

Supraventricular tachycardia is the most common arrhythmia in childhood, including the neonatal period. It can manifest as tachycardia antenatally and restlessness, sucking disorder, tachypnea, tachycardia, and heart failure postnatally. Although many newborns tolerate supraventricular tachycardia in the first hours, in cases lasting more than 6-12 hours, heart failure may develop after decreasing heart rate [4]. 15% of patients have a history of sepsis and drug use [5]. Wolf

Parkinson White (WPW) pattern is available on ECG in 10-20% of cases. Some congenital heart anomalies (Ebstein anomaly, single ventricle, large artery transposition) tend to manifest with supraventricular tachycardia [4]. Gilljam et al. [6] reported that the median age of onset in supraventricular tachycardia was 1 day (1-30 days) and mean heart rate was 270±27 beats/min by retrospectively examining the files of 109 newborns hospitalized with the diagnosis of supraventricular tachycardia for 27 years. In 52 (48%) of the patients, they found heart failure during the first referral and reported that 17% were resistant to treatment. In antenatal follow-up of patients with heart failure, hydrops fetalis was found in 10 patients and intrauterine supraventricular tachycardia, in 9 patients. In this study, the electrocardiographic findings of our patients were compatible with supraventricular tachycardia, and there were no WPW pre-excitation findings in the ECG obtained during sinus rhythm. Echocardiographic examinations did not any heart abnormalities. It was stated that the antenatal follow-up of the patients was normal. There was no evidence of sepsis, drug intake history, or placement of any central catheters which could cause pain and supraventricular tachycardia.

These are the first neonates with tachyarrhythmia and hypothyroidism in the literature to our knowledge. There are reported adult patients with this concomitance [7,8].

The heart is a major target organ for thyroid hormone action. Normal thyroid hormone levels are essential for maintaining normal heart structure and function. In hypothyroidism, the muscle of the heart is weakened in contraction and relaxation phases. This means that the heart cannot pump, and the stroke volume is reduced [9].

Changes in thyroid hormone levels also exert influence on electrophysiological function of the heart. The basic reason of alteration in electrophysiological function that is caused by changes in different thyroid hormone levels has not yet been completely explored. To the best of our knowledge, tachyarrhythmia is an unusual finding in hypothyroidism.

In neonates with congenital hypothyroidism, left systolic and diastolic functions were lower [10]. Although our patients had hypothyroidism, all of them had tachyarrhythmia. Thyroid hormone exerts its effect by influencing thyroid hormone regulating gene expression via interactions with the high affinity thyroid hormone receptor located in the nucleus [9]. Thyrotropin-releasing hormone triggers the pituitary gland to release thyroid stimulating hormone. Thyrotropin-releasing hormone-containing fibers innervate autonomic motor and premotor nuclei of the brainstem and spinal cord which regulate functions of cardiovascular system [11]. It is reported that thyrotropin-releasing hormone itself can cause high blood pressure, tachyarrhythmia, palpitations. It can increase the release of noradrenalin; and it also acts like adrenalin [12]. We can speculate that thyrotropin-releasing hormone in our patients may have caused these supraventricular tachycardia attacks, but on the other hand it is not clear why all newborns with hypothyroidism do not have tachyarrhythmia. Measuring of thyrotropin-releasing hormone level is not used in practice.

Thyroid hormone receptor plays a key role in mediating the physiologic actions of thyroid hormone so pre or post receptor effects might play a role in those patients with tachycardia. Case 2 had atrioventricular block that might be detected in hypothyroidism [13]. However, the physiopathological basis of tachyarrhythmia in these patients is not elucidated.

Conclusion

Hypothyroidism should be kept in mind in cases with tachyarrhythmia as well as bradycardia.

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