

ones. In suspected cases of illicit drug abuse, beta-adrenergic blocking agents should be avoided. Patency of infarct-related artery can be achieved by PCI in patients with AMI associated with amphetamines or amphetamine-like substances.

Abdullah Uluçay, Canan Arpacık Kargı¹, Mehmet Faruk Aksoy
Clinic of Cardiology, Defne Hospital, Hatay
¹Clinic of Cardiology, Iskenderun State Hospital, Hatay-Turkey

Video 1, 2, 3. Pre-stenting, post-stenting and final coronary angiographic video/movie images in different views.

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Address for Correspondence/Yazışma Adresi: Dr. Abdullah Uluçay,
Özel Defne Hastanesi, Kardiyoloji Bölümü, Hatay-Türkiye
Phone: +90 326 221 11 00 Fax: +90 326 221 44 45
E-mail: ulucaytr@hotmail.com

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A case of iatrogenic hypothyroidism presented with cardio-inhibitory syncope and resolved by thyroxine supplementation

Tiroksin tedavisi ile düzelen kardiyoinhibitör senkop ile gelen iatrojenik hipotiroidili bir vaka

Introduction

Thyroid hormones have inotropic and chronotropic effects on heart functions (1). In hypothyroid situation, life-threatening dysrhythmias may occur (2).

We report a 15-year-old girl who presented with syncope and sinus bradycardia, low voltage, prolonged QT interval and first degree atrioventricular block on electrocardiogram due to iatrogenic hypothyroidism.

Case Report

A 15-year-old girl presented with syncope. She lost consciousness for approximately five minutes when she has been sitting, before her admission. No feces or urine incontinence or tonic contractions had been observed.

On physical examination she was conscious, her body weight was 42.5 kg (5-10 percentiles), height 158 cm (25-50 percentiles). The pulse rate was 52 beats/min, the blood pressure 85/40 mmHg. The auscultation of heart was normal except for bradycardia; neurological examination was normal. There was a transverse incision scar on the anteromedial neck; thyroid gland was nonpalpable. The blood testing showed macrocytic anemia (Hb: 9.6 gr/dl, Htc: 26.2%, MCV: 100.8 fl). Blood glucose, electrolytes telecardiogram and electroencephalogram were normal. Electrocardiography showed sinus bradycardia, low voltage, first degree atrioventricular block (PR interval was 0.20 s) (Fig. 1A). Echocardiographic examination was normal. Holter monitoring showed sinus bradycardia (minimum heart rate was 45 beats/min in sleepiness, 47 beats/min in wakefulness), first degree atrioventricular block, and prolonged QT interval (0.45 s). Head-up tilt testing was normal. Her exercise capacity was low for modified Bruce protocol. Her pulse rate maximally increased up to 109 beats/min with exercise.

Serum vitamin B12 and folic acid levels were normal. Thyroid functions were found as: free triiodothyronine 3 (T3) <0.26 pg/ml, free thyroxine (T4): 0.06 ng/dl, TSH: 604.6 µU/ml. Ultrasonographic examination and thyroid scanning did not show any thyroid tissue. When her past history was detailed, it was learned that she had easy fatigability, dry skin, constipations, reduced exercise capacity and academic performance since one year. One year ago she had been operated for an anteromedial neck mass in another hospital. Since histopathological examination of the mass reported thyroglossal duct cyst, her operator had not prescribed any drug therapy and had said that follow-up was not required. However, the pathologic specimens of the patient were investigated again by Pathology Department of our hospital and ectopic thyroid tissue and thyroid adenoma was seen. Levothyroxine therapy was started with a dose of 25 µg/day and was raised weekly up to 150 µg/day. At the end of first month of treatment skin dryness, constipations, anemia and effort intolerance improved, academic performance increased. The electrocardiographic findings returned to normal (Fig. 1B).

Discussion

Thyroid hormones have inotropic and chronotropic effects on heart function and enhance overall total protein synthesis in the heart (1). Adenosine-triphosphatase activity of myosin heavy chain α is markedly higher than myosin heavy chain β . T3 stimulates the expression of myosin heavy chain α but decreases the expression of myosin heavy chain β . This regulation may modulate myocardial contractility. Thyroid hormones increase cardiac actin and troponin I. Release of calcium and its reuptake into the sarcoplasmic reticulum are important for systolic and diastolic functions. The gene encoding calcium pump of the sarcoplasmic reticulum is markedly T3 responsive (2). Thyroid hormones can regulate β -adrenergic receptor number in the heart and may enhance sensitivity to catecholamines (1).

Atrioventricular blocks, sinus bradycardia, prolongation of the QT and QRS intervals and Torsades de Pointes were reported in hypothy-



Figure 1. Our patient's electrocardiogram on admission showing sinus bradycardia, low voltage, and first degree atrioventricular block (PR interval 0.20 s) (A) and after levothyroxine therapy showing resolution of these findings (B)

roid patients (1, 3, 4). Also 2:1 atrioventricular block was demonstrated in a patient with subclinical hypothyroidism (5). These electrocardiographic changes are resolved by thyroid hormone replacement (4, 5). In patients, low voltage may occur in electrocardiogram due to accumulation of fluid in the pericardium (6). Myocardial swelling, interstitial fibrosis and accumulation of mucopolysaccharides have been demonstrated on histological examination of hypothyroid heart (1, 7). These changes may lead to voltage suppression without pericardial effusion (8).

Ectopic thyroid is the presence of functioning thyroid tissue in aberrant location. It can be found anywhere between the foramen cecum and the normal pretracheal position. The ectopic thyroid is only source of functioning thyroid tissue in approximately 75% of such cases (9). Also in our patient, ectopic thyroid gland was the only thyroid hormone source.

Thyroid hormones play a role in normal hematopoiesis. Macrocytic or normocytic anemia may occur in hypothyroid status and anemia is resolved by thyroid hormone replacement (10).

Conclusion

Our patient was diagnosed as having iatrogenic hypothyroidism which was resulted from total excision of ectopic thyroid tissue. Because she had a short period of unconsciousness without feces or urine incontinence, tonic contractions or constitutional symptoms, electroencephalogram and head-up tilt testing was normal, neurological seizure and vasovagal syncope were excluded. Since all of her clinical and laboratory abnormalities including electrocardiographic findings improved with levothyroxine therapy, syncope was considered to be due to cardio-inhibitory effects of hypothyroidism. We would like to emphasize that careful evaluation of anteromedial neck masses for ectopic thyroid gland before excision is very important. On the other hand, thyroid hormone replacement and close follow-up of patient are essential after thyroidectomy.

Melike Sezgin Evim, Birsen Uçar¹, Zübeyir Kılıç¹, Birgül Kirel²
Department of Hematology, Faculty of Medicine, Uludağ
University, Bursa

¹Departments of Pediatric Cardiology and ²Pediatric Endocrinology,
Faculty of Medicine, Eskişehir Osmangazi University,
Eskişehir-Turkey

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Address for Correspondence/Yazışma Adresi: Dr. Melike Sezgin Evim,
Uludağ Üniversitesi Tıp Fakültesi, Hematoloji Anabilim Dalı, Bursa-Türkiye
Phone: +90 224 295 05 47 Fax: +90 224 442 81 43
E-mail: melikevim@yahoo.com

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