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Hypothyroidism complicates bradyarrhythmic episodes in a heart-transplanted patient: Can it be treated with low-dose dopamine?

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Abstract

Extracorporeal circulation decreases thyroid hormone levels in peripheral blood. This clinical entity may complicate the postoperative period after heart transplantation if the recipient has taken thyroid hormone replacement therapy. Cardiac transplantation was performed on a patient in whom thyroid hormone levels decreased after surgery. Sinus bradycardia was seen after surgery (30-40 bpm). Thyroid hormones were replaced in the patient. Due to the fact that temporary pacing decreased blood pressure, dopamine was safely given in very low doses. This case was discussed under the literature knowledge.

Key words: bradyarrhythmia, hypothyroidism, cardiac transplantation.

Streszczenie

Krążenie pozaustrojowe zmniejsza stężenie hormonów tarczycy we krwi obwodowej. Opisane zjawisko kliniczne może być źródłem powikłań pooperacyjnych po przeszczepie serca, jeśli biorca jest poddawany hormonalnej terapii zastępczej tarczycy. Wykonano przeszczep serca u pacjenta, u którego po operacji stężenie hormonów tarczycy zmniejszyło się. Pooperacyjnie zaobserwowano bradykardię zatokową (30-40 bpm). Pacjent otrzymał hormonalną terapię zastępczą tarczycy. Jako że tymczasowy rozrusznik serca zmniejszył ciśnienie krwi pacjenta, dopaminę podano ostrożnie w bardzo niskich dawkach. Prezentowany przypadek został omówiony w kontekście relewantnych publikacji.

Słowa kluczowe: bradyarytmia, niedoczynność tarczycy, przeszczep serca.

Introduction

Thyroid hormones have an important effect on the cardiac and peripheral vascular systems and they regulate basal metabolism [1-3]. Cardiopulmonary bypass decreases thyroid hormones especially in free triiodothyronine (FT_3) levels within the first postoperative 24 hours [3]. A mean percentage of 30-50% of hypothyroidism patients with circulatory insufficiency have deterioration in myocardial contractility and a decrease in cardiac output [4]. In this report, we present an orthotopic cardiac transplant patient with a severe and treatment-resistant bradyarrhythmia episode in spite of thyroid hormone replacement in the postoperative period. She was treated with very low-dose dopamine. This case was discussed under the literature knowledge.

Case report

A 42-year-old female patient with dilated cardiomyopathy underwent orthotropic cardiac transplantation by biatrial technique. Thyroid hormone function tests of the donor were in the normal range. Thyroid hormones levels were within normal levels preoperatively (FT_3: 2.28 pg/ml, free thyroxin [FT_4]: 3.01 ng/dl, thyroid-stimulating hormone [TSH]: 0.212 µIU/ml). The patient had been taking levothyroxine 50 mg/day. Usual drug administration of triple-therapy of cyclosporine, azathioprine, and methyl-prednisolone was applied. The patient's haemodynamic status was uneventful within 20 hours. The patient was extubated in the 15^{th} hour after heart transplantation. But after this time arterial blood pressures decreased with bradyarrhythmic episodes. Arterial blood pressure was around 70 mmHg systolic, 40 mmHg diastolic, and heart rates were within 30-50 bpm.

Temporary pacing was immediately initiated with 70 bpm to provide haemodynamic stability. However, after pacing, in spite of the occurrence of sufficient heart rate levels, arterial blood pressure continued to fall, whereupon the pacemaker was stopped, and adrenalin was given in 0.1-3 µg/kg/min doses. After initiating of adrenalin, sinus tachycardia and hypertension were noted. The same clinical condition was observed with dobutamine infusion. Because of these entities, inotropic agents were obliged to

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stop. Dopamine of 5 µg/kg/min was ordered but again it resulted in tachycardia and hypertension. Then the dopamine dose was reduced to around 1 µg/kg/min. Appropriate haemodynamic stability was provided and maintained with dopamine doses from 1.1 µg/kg/min to 0.8 µg/kg/min. Thyroid hormone levels were low (FT_3: 1.89 pg/ml, FT_4: 4.03 ng/dl, TSH: 0.024 µIU/ml) during this period. Thyroid hormone replacement therapy doses were increased. Dopamine infusion therapy continued for six days. After achieving an adequate pulse rate at the end of the sixth day, dopamine was stopped. Thyroid hormone values were FT_3: 2.4 pg/ml, FT_4: 2.57 ng/dl, and TSH: 0.22 µIU/ml on the postoperative seventh day. The patient was discharged uneventfully on the 30th postoperative day. There were no signs related with rejection or infection during this period.

Discussion

Recipients meet three different problems after heart transplantation. Firstly, the plasma FT_3 level rapidly falls due to the beginning of brain-death neuroendocrine changes, which end with aerobic metabolism breakdown, which finally leads to stress on the donor heart. This condition may relate to acute graft insufficiency, which is a special term used in cardiac transplantation. Secondly, cardiopulmonary bypass suppresses thyroid hormone levels. Thirdly, corticosteroids use of immunosuppressive regimes in cardiac transplantation reduces thyroid hormone levels as well. Postoperative usage of high-dose steroids reduces the plasma levels of TSH, FT_3, and FT_4. It is reported that oral use of methyl prednisolone of 20 mg once a day for seven days causes a reduction in TSH with no change in FT_3, FT_4, or thyroid-binding-globulin [3-5].

Thyroid hormones play an important role on the myocardial contractility in transplant patients [1-3]. Thyroid hormone replacement therapy reduces the dose and usage time of inotropic agents [3, 4]. It enhances postoperative haemodynamic stability and reduces arrhythmia incidence. Hypothyroidism reduces average and maximum heart rate and usually causes sinus bradycardia and atrioventricular blocks [4, 5]. It leads to haemodynamic deterioration via cardiac dysfunctions. For these reasons it is important to ensure adequate thyroid hormone levels after transplant surgery to maintain optimal cardiac function.

Bradycardia can generally be seen after cardiac transplantation and is a foreseeable complication of surgery. It is preferred to use beta mimetic, temporary pacing, or inotropic agents as a first-line treatment. In our case, inotropic agents administered, adrenalin and dobutamine, respectively, were inadequate and hypotension resumed despite pacemaker implantation. Although we tried to adjust the drug doses, we could not break tachycardia, and hypertension sustained. Haemodynamic stability was achieved with low-dose dopamine (1 µg/kg/min). Thyroid hormone test results conducted at the same time were in the normal ranges. Hormone replacement therapy was then administered, and by starting this replacement therapy we were able to decrease the dopamine dose, and then it was stopped on the seventh day of operation. Although there was no relationship between thyroid hormones and dopamine, as reported before, the satisfactory clinical process of the patient was unexpected.

As a result, in the early postoperative period of cardiac transplant patients, bradycardia-triggered haemodynamic instability may occur because of a decrease in thyroid hormone levels. In this situation, instead of excessive treatment, only low-dose dopamine may be enough to achieve a haemodynamic stability with no complications.

Disclosure

Author reports no conflict of interest.

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