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CASE REPORT

Thyrotoxicosis-facilitated bridge to recovery with a continuous-flow left ventricular assist device

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Abstract

The HeartMate II is a continuous-flow left ventricular assist device that can be explanted from patients after cardiac recovery. We implanted a HeartMate II in a 21-year-old man who had idiopathic cardiomyopathy. A year later, he developed thyrotoxicosis, presumably secondary to amiodarone administered for ventricular fibrillation. Four months after the diagnosis of thyrotoxicosis, thyroid hormone levels had returned to normal, and native cardiac function had improved remarkably. After a support period of 24 months, the HeartMate II was explanted. Six years later, the patient continues to be in New York Heart Association functional Class I. Amiodarone-induced thyrotoxicosis may have contributed to myocardial recovery.

Keywords: Amiodarone • Cardiomyopathy • Left ventricular assist device • Thyrotoxicosis

The HeartMate II left ventricular assist device (LVAD) (Thoratec Corporation, Pleasanton, CA, USA) is one of a number of continuous-flow blood pumps being utilized clinically. We describe a patient who received a HeartMate II for idiopathic cardiomyopathy and later required medical management of amiodarone-induced thyrotoxicosis. The thyrotoxicosis appeared to accelerate improvement in his native cardiac function, allowing the LVAD to be explanted after 24 months of support.

CASE REPORT

A 21-year-old man was transferred from his community hospital to our institution for heart failure of ~1 month's duration, with renal and hepatic dysfunction. On his admission to our hospital, echocardiography confirmed the presence of global hypokinesia compatible with idiopathic, chronic heart failure. The left ventricular ejection fraction was 10%. Because of the patient's deteriorating condition, he did not respond to medical therapy, so a HeartMate II LVAD was implanted in a routine fashion. Intraoperative transesophageal echocardiography confirmed a depressed ejection fraction of 10%, with a left ventricular end-diastolic diameter of 6.1 cm. At the time of implant, a biopsy of the core showed moderate hypertrophy and interstitial fibrosis with minimal replacement fibrosis.

Over the next 2 weeks, the patient recovered uneventfully and became ambulatory. Therapy with beta blockers and angiotensin-converting enzyme inhibitors was initiated commensurate with his heart failure. Echocardiographical studies showed adequate left ventricular unloading by the HeartMate II LVAD. His haemodynamic values remained stable, and he was discharged home on postimplant Day 35. Thyroid function test results were normal during this initial admission.

Six months later, the patient fainted while doing yard work. He regained consciousness and was able to drive to his community hospital, where he remained alert and oriented. In the emergency room, electrocardiography showed ventricular fibrillation. Sinus rhythm was restored after cardioversion, and amiodarone therapy was initiated as a precautionary measure. Thyroid function tests were not obtained during this admission.

During a follow-up visit to our clinic, 1 year after LVAD implantation (and 4 months after amiodarone initiation), the patient underwent routine transthoracic echocardiography, which showed sinus tachycardia (up to 120 bpm) with an adequately decompressed left ventricle and improved ejection fraction of 40% with LVAD support. Further evaluation revealed that over the preceding 6 weeks, he had a progressive onset of weight loss, tremors, heat intolerance, hyperactivity, insomnia and palpitations. Thyroid tests showed the following values: thyroid-stimulating hormone, <0.01 mcU/ml; T4, 15.5 mcg/dl; free T4, 4.2 mcg/dl; T3, 769 ng/dl. A radioactive iodine scan, with an 81% diffuse uptake at 24 h, confirmed a diagnosis of thyrotoxicosis (Graves' disease). The patient was given 12.5 mCi of radioactive iodine and, later, methimazole.

Over the ensuing 6 months, the patient's thyroid hormone levels normalized: T3 decreased to 237 ng/dl, free T4 fell to 1.9 mcg/dl and thyroid-stimulating hormone rose to 0.23 mcU/ml. During the same period, his myocardial contractility improved. He underwent right-sided heart catheterization and dobutamine stress testing with LVAD pump speed reduced to 6000 rpm. Under these conditions, the left ventricular ejection fraction was normal. Therefore, after a support period of 24

months, the HeartMate II was explanted via a sub-costal approach that precluded myocardial biopsy. The patient tolerated the procedure well and was discharged home 12 days later, on a regimen of beta blockers and angiotensin-converting enzyme inhibitors. After device explantation, transthoracic echocardiography revealed an ejection fraction of 60%. Six years after LVAD removal, the patient continues to do well and remains in New York Heart Association functional Class I, with normal left ventricular function on echocardiography.

DISCUSSION

This patient developed thyrotoxicosis, a known complication of amiodarone administration, while being supported by a HeartMate II LVAD. During the period of thyrotoxicosis, ventricular function improved dramatically.

Although usually considered deleterious, elevated thyroid hormone levels may be beneficial to ventricular function in selected patients. Indeed, Khalife *et al*. [1] found that thyroid hormones improved left ventricular function in hypothyroid hamsters with dilated cardiomyopathy. Using a similar model, Kuzman *et al*. [2] showed that thyroid hormones decreased the left ventricular end-diastolic dimension. In a hypertensive rat model of heart failure, Thomas *et al*. [3] found that after administration of thyroid hormones, left ventricular systolic wall stress decreased because of a dose-dependent decrease in the left ventricular chamber diameter and an increase in wall thickness. The potentially positive effects of thyroid hormones, including remodelling and improved haemodynamic status, have also been observed in rats after a myocardial infarction [4].

Administration of agents capable of causing myocardial hypertrophy as a second-phase treatment is not novel in patients with failing, dilated ventricles that have already undergone reverse remodelling after a period of mechanical and pharmacological unloading. Combined with mechanical left-ventricular assistance, clenbuterol, a beta 2-adrenergic receptor agonist, has been successfully used for myocardial recovery [5]. Thyroid hormone has also been proposed as an agent to improve cardiac function in organ donors. In our patient, amiodarone-induced thyrotoxicosis may have contributed to myocardial recovery.

The role of thyroid hormones in aiding ventricular recovery is uncertain. Many patients have undergone LVAD removal without the aid of thyroid or other beta agonist therapy. However, thyroid hormone therapy and other approaches for treating dilated cardiomyopathy deserve continued study, particularly in young patients.

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