Case report

Sleep apnoea/hypopnoea syndrome: a potential cause of graft failure following heart transplantation

Udim U. Nkere *, M.C.S. Hall, P.A. Corris

Cardiothoracic Surgery Department, Freeman Hospital, High Heaton, Newcastle Upon Tyne, NE7 7DN, UK

Received 24 April 1997; received in revised form 3 November 1997; accepted 9 December 1997

Abstract

A 54-year-old man presented 54 months after a successful heart transplant with cor pulmonale secondary to obstructive sleep apnoea/hypopnoea syndrome (SAHS). This unusual cause of reversible graft failure following heart transplantation is presented in this case report. © 1998 Elsevier Science B.V. All rights reserved.

Keywords: Sleep apnoea/hypopnoea syndrome; Heart transplantation; Pulmonary function test

1. Introduction

Sleep apnoea/hypopnoea is defined as an interval during sleep during which respiratory airflow is stopped/reduced (by 30–50%) for 10 s or more. A precise definition of sleep apnoea/hypopnoea syndrome (SAHS) is difficult, but the consensus is that a sleep apnoea index (episodes of sleep apnoea per hour of sleep) of greater than 10 defines a person with the syndrome. These episodes of apnoea and hypopnoea, associated with falls in arterial oxygen saturation, frequently occur in the same patient and may be reported as a single apnoea/hypopnoea index. The prevalence of the syndrome is reported to be around 1–4% of the adult population [1,2,10].

There are three classifications of SAHS. Obstructive SAHS (OSAHS) (the interruption of respiratory flow despite the persistence of respiratory efforts) accounts for most cases of sleep apnoea/hypopnoea diagnosed in sleep disorder centres. Central SAHS (CSAHS) (an interruption of the drive to breathe induced by a decrease or increase (paradoxically) in the activity of the respiratory centres) occurs at about 10% the rate of OSAHS. Thirdly, mixed is a combination of obstructive and central. Additionally, Cheyne-Stokes respiration during sleep, in association with end-stage heart disease, can cause SAHS [1].

Though a relatively ‘new’ disease, SAHS has received considerable literature review regarding its association with cardiovascular disease [2]. It has been implicated as a cause of severe cardiac dysfunction that improves with the control of the apnoeas, and also has significant neurological, psychological, endocrine, haematological and nephrological consequences [5]. However, despite a documented incidence of 2.5% in post-heart transplant patients [6], the role of SAHS as a cause of graft dysfunction after heart transplantation is a relatively new phenomenon [3,6].

2. Case report

A 54-year-old man diagnosed with ischaemic cardiomyopathy underwent successful heart transplantation in September 1993. He was discharged from
hospital 2 weeks posttransplantation with triple therapy maintenance immunosuppression and oral antidiabetic medication. In December 1993 he was admitted for control of diabetes and was commenced on insulin. From March 1994 he became hypertensive and was commenced on antihypertensives. He also gained weight, going from 94 kg preoperatively to 135 kg.

In March 1996 he presented with increased shortness of breath, decreased exercise tolerance, daytime sleepiness, orthopnoea, paroxysmal nocturnal dyspnoea, and pedal oedema. His wife reported the onset of severe nocturnal snoring.

On examination he was short of breath at rest, cyanosed and clearly overweight. His heart rate was 100 beats/min and blood pressure was 130/80 mmHg. The jugular venous pressure was raised and he had dependent oedema extending to his umbilicus. Auscultation revealed first and second heart sounds of normal intensity and an additional S3. Pulmonary auscultation revealed decreased air entry at both lung bases. His arterial blood gases on room air were: pH 7.4, pCO₂ 8.06 kPa, pO₂ 7.0 kPa, HCO₃ 38.6 mmol l⁻¹, and base excess 11.6 mmol l⁻¹. His pulmonary function tests (PFTs) showed a restrictive defect with: FEV₁ 1.15 (31.5% of predicted), and FVC 1.40 (28.9% of predicted). There had been a significant reduction in volume from his pretransplant PFTs which were: FEV₁ 2.20 (56.7% of predicted) and FVC 2.65 (53% of predicted).

An echocardiogram on admission indicated impaired overall right ventricular (RV) function, mild tricuspid regurgitation, and good left ventricular (LV) function. In contrast, an echocardiogram done 1 year prior showed good RV function, no tricuspid regurgitation, and good LV function. Endomyocardial biopsy showed no signs of rejection. Abdominal ultrasonography showed systemic venous congestion, but no ascites. Cardiac catheterisation excluded coronary artery disease.
Sleep studies were carried out using a computerised sleep analysis system (Fig. 1) (Medilog Rapide sleep analysis system, Oxford Instruments, Abingdon, UK). Severe obstructive sleep apnoea was diagnosed.

This patient’s cor pulmonale responded well to medical treatment of heart failure, weight loss, and avoidance of alcohol and nocturnal sedatives. Continuous positive airways pressure (CPAP) was not well tolerated. At 18-months follow-up the patient remains well with no signs of his presenting problems.

3. Discussion

The proposal that SAHS may be a reversible cause of late graft failure in heart transplant patients is a relatively new concept. More commonly encountered late complications include infections, rejection, malignancy, and graft atherosclerosis. Each of these were clinically excluded in the case presented. A diagnosis of SAHS giving rise to cor pulmonale [1] represents an unusual problem in heart transplant patients.

A 45% prevalence of sleep-disordered breathing, including SAHS, has been reported in patients on a heart transplant waiting list [8]. At the time of presentation this patient’s steroid regime was at a low dose level (0.1 mg/kg pretransplant body weight). His weight gain continued despite repeated professional input. To avoid the problems of weight gain/obesity, and other steroid-related complications, a number of transplant centres adopt a steroid-free immunosuppressive regime in selected patients rather than the standard triple therapy maintenance [7]. However, considerable weight gain may be attributed to the improvement in global well-being rather than the administration of steroids [7].

The main symptoms found in those with SAHS are daytime sleepiness, nocturnal choking, impotence, and bed partner’s observation of snoring and apnoeas [4]. Typical patients are male, obese and hypertensive with a larger neck circumference and enlarged uvula. Such non-specific clinical findings necessitate further definitive investigation.

The use of full polysomnography to diagnose SAHS may include the following: (1) electroencephalography, electromyogram and electro-oculogram to allow sleep staging, (2) chest and abdominal wall excursion and oronasal air flow to detect breathing pattern, and (3) oximetry, heart rate, sound recording of snoring, and video recording. Fortunately the use of such equipment is only necessary in the diagnosis of more difficult cases and, as in the case presented, the use of a more limited range of equipment will suffice [4,9].

In the management of OSAHS, all patients should be encouraged to lose weight, and avoid sedatives and alcohol. Patients who fail to respond to these general measures should be offered CPAP on a nightly basis. Surgery is sometimes indicated to correct any specific defect in the upper airway [4].

This and other reports [3,6,8] suggest that SAHS should be considered as a potential cause of graft failure in heart transplant recipients and should be identified as a preexisting abnormality during heart transplant evaluation. In the case presented we believe that OSAHS was the primary cause of heart failure, however, it is conceivable that SAHS may be part of a multifactorial cause of heart failure both pre- and post-heart transplantation.

References