Cor Pulmonale due to Obstructive Sleep Apnoea

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Summary

A 47 year old man with a long history of chronic loud snoring and daytime sleepiness presented with hypercapnic respiratory failure and right ventricular failure. The diagnosis of obstructive sleep apnoea (OSA) leading to the 'obesity-hypoventilation syndrome', was supported by the findings of an overnight cardio-respiratory monitoring during sleep. His symptoms and arterial blood gases improved following treatment with nocturnal nasal continuous positive airway pressure (CPAP).

Key words: Obstructive sleep apnoea, 'obesity-hypoventilation syndrome'.

Introduction

Recurrent episodes of nocturnal hypoxaemia and hypercapnia due to severe obstructive sleep apnoea (OSA) may result in attenuation of hypoxic and hypercapnic ventilatory drives during wakefulness[1]. When obesity and daytime somnolence, both of which are features of OSA, plus right heart failure and hypoventilation with hypercapnia are present without intrinsic pulmonary disease, the 'obesity-hypoventilation syndrome' is constituted[2]. This is a report of a patient with severe OSA leading to the 'obesity-hypoventilation syndrome', whose symptoms and arterial blood gas profile improved following nocturnal nasal continuous positive airway pressure (CPAP) treatment.

Case Report

A 47 year old chronic smoker and newly diagnosed hypertensive was admitted because of progressive effort intolerance associated with bilateral leg oedema of 2 years' duration. He was a habitual snorer and suffered excessive daytime sleepiness. He worked as a driver but recently had great difficulty in staying awake while driving.

He weighed 98 kg and his body mass index was 34 kg/m^2. He was centrally cyanosed, had a short neck and fell asleep and snored in between answering questions. His uvula was oedematous and wrinkled at its base when he said "aah". The blood pressure was 160/110 mm Hg, jugular venous pressure was elevated and cardiomegaly was present with a gallop rhythm. The pulmonary component of the second heart sound was accentuated. Lung auscultation was normal. No anatomical abnormality of his upper airway was found to explain his sonorous breathing.

The haemoglobin was 164 g/l. The chest X-ray showed cardiomegaly but normal lung fields. The electrocardiograph revealed p 'pulmonale' and a right ventricular strain pattern. Daytime arterial blood gas analysis while he was breathing room air showed pH 7.34, partial pressure of oxygen (pO2) 5.6 kPa, pCO2 7.6 kPa, bicarbonate 31 mmol/l, and oxygen saturation 78%. Lung function test showed: forced expiratory volume in 1 second (FEV1) 1.96 l (75% predicted), forced vital capacity (FVC) 2.10 l (67%

predicted), FEV1/FVC 93%, residual volume 0.86 l (65% predicted), total lung capacity 2.96 l (65% predicted), and normal diffusing capacity. Thyroid function test was normal.

An overnight cardio-respiratory monitoring during sleep using MESAM 4[3], a microprocessor-based digital recording device which monitors oxygen saturation, heart rate, snoring and body position, was performed. The recording showed snoring was present throughout the night irrespective of his body position. Snoring was interrupted by periods of witnessed apnoea. The heart rate tracings showed marked cyclical variations (Fig 1). The oxygen saturation tracings showed low baseline saturation with cyclical further desaturations related to the intervals of silence due to apnoea.

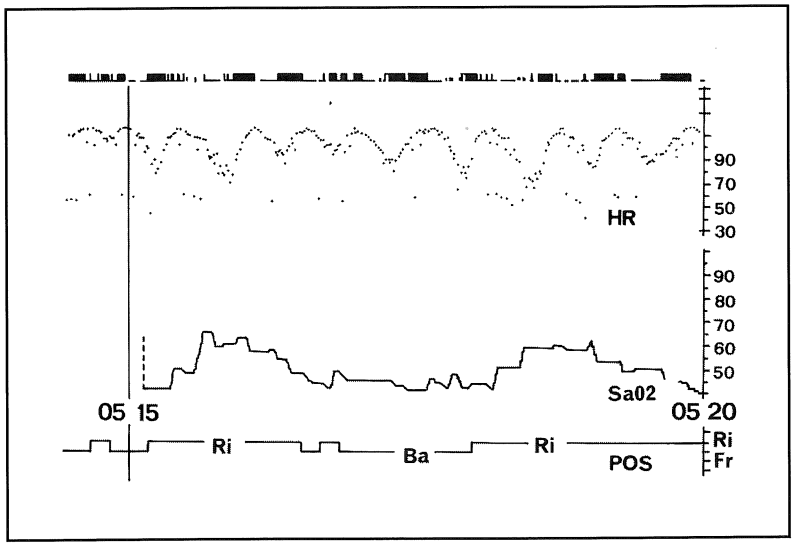

Fig 1: MESAM 4 recording showing a representative 5 minute epoch. All 4 recorded channels are displayed and labelled as HR (heart rate), SaO2 (oxygen saturation), and POS (body position). The height of the snoring tracing (top-most tracing) varied with the loudness and with the length of the snore. The HR tracing shows marked cyclical variations in heart rate. The SaO2 tracing shows very low saturation with cyclical desaturations. The body position indicates that the patient was lying on his right side (Ri) alternating with his back (Ba).

His hypertension and heart failure were treated with enalapril and frusemide. Nasal continuous positive airway pressure (CPAP) at 12 cm H_20 was applied via a tight fitting bubble mask over his nose when he slept at night in the ward (Fig 2). Supplemental oxygen at 5 l/min was administered through an access port on the mask. An arterial blood gas analysis after an hour of nasal CPAP and oxygen therapy showed pH 7.46, pO_2 22 kPa, and pCO_2 5.1 kPa.

Following nasal CPAP treatment, he no longer had daytime hypersomnolence. After a week's treatment with nocturnal nasal CPAP without supplemental oxygen, daytime arterial blood gas analysis while he was breathing room air showed: pH 7.43, pO2 9.8 kPa, pCO2 4.5 kPa and bicarbonate 22 mmol/l. At the recommendation of the author, his company paid for a nasal CPAP machine for his nightly use at home.

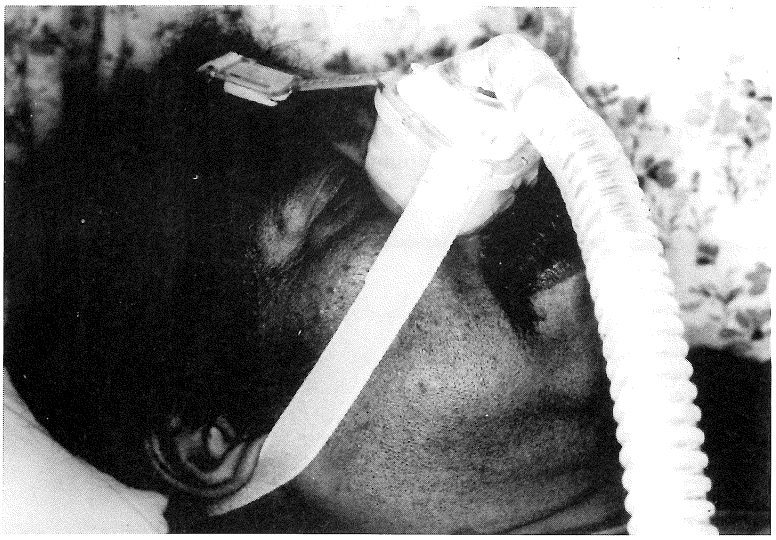

Fig 2: Patient wearing the nasal CPAP mask with a corrugated tube which extends to the blower.

Discussion

The presence of obstructive sleep apnoea (OSA) syndrome should be suspected when someone complains of excessive daytime sleepiness associated with chronic heavy nocturnal snoring like in this patient[3]. During sleep, pulmonary artery pressure is elevated due to an increase in pulmonary resistance resulting from hypoxic vasoconstriction. Daytime hypoxaemia plays a major role in the occurrence of permanent daytime pulmonary arterial hypertension and right heart failure, as happened in this patient.

Obstructed breathing during sleep may be viewed as a continuum. The most benign extremity of this continuum is represented by pure snoring. A step further is characterised by the occurrence of occasional apnoeas. The occurrence of repeated apnoeas due to obstructed breathing gives rise to the full-blown obstructive sleep apnoea syndrome. When daytime hypoventilation and pulmonary hypertension develop, the complete Pickwickian syndrome[2] or 'obesity-hypoventilation syndrome' is seen, as illustrated by this case.

In many ways, this patient with OSA syndrome resulting in the 'obesity-hypoventilation syndrome', resembles patients with chronic obstructive pulmonary disease with the 'blue and bloated' syndrome. However, his respiratory function test did not show evidence of airflow obstruction. The restrictive pattern noted was related to his obesity.

Nasal CPAP, which acts as a pneumatic splint to prevent the flaccid upper airway from collapsing to obstruct the airflow when the dilator pharyngeal muscles fail to contract during inspiration, used nightly during sleep, is the treatment of choice for most patients with OSA[2]. Nasal CPAP also eliminates snoring.

Impaired chemosensitivity, low pulmonary and chest wall compliance related to obesity, and underlying bronchopulmonary disease are possible factors contributing to persistent daytime hypoventilation. Repeated episodes of nocturnal hypoxaemia and hypercapnia due to severe sleep apnoea may result in attenuation of hypoxic and hypercapnic ventilatory drives during wakefulness[1]. Sleep apnoea itself can

cause long-term carbon dioxide retention even in the absence of lung disease, and the CO_2 retention in this circumstance can be reversed by preventing sleep apnoea, with nasal CPAP[2]. Although this patient's OSA requires long-term treatment with nocturnal nasal CPAP, his daytime hypoxaemia, hypercapnia and hypersomnolence improved after only a week's therapy.

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References

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