RECURRENT ACUTE PULMONARY OEDEMA ASSOCIATED WITH OBSTRUCTIVE SLEEP APNOEA

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ABSTRACT

A patient had recurrent acute nocturnal pulmonary oedema following an anterior myocardial infarction despite a normal maximal stress electrocardiogram. He had a history of chronic heavy snoring and other symptoms to suggest a diagnosis of obstructive sleep apnoea (OSA) which was supported by an overnight sleep study. The recurrent acute pulmonary oedema was most likely due to a combination of poor left ventricular function and obstructive sleep apnoea.

Keywords: *obstructive sleep apnoea, recurrent acute pulmonary oedema*

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INTRODUCTION

Recent data have linked habitual snoring and obstructive sleep apnoea (OSA) with increased risk of cardiovascular disease[1,2]. The effects of negative intrathoracic pressure swings during obstructive apnoeas and sleep-related hypoxia with its myocardial contractile depressant effects both promote the development of pulmonary oedema[3-5]. Patients with normal left ventricular function have been reported to develop acute nocturnal pulmonary oedema due to OSA[6,7]. The haemodynamic effects resulting from the intermittent falls in intrathoracic pressure to more negative values during the apnoeic episodes and the associated hypoxaemia would be especially pronounced in patients with underlying coronary artery disease with impaired left ventricular function[5,8]. We report a case of recurrent acute nocturnal pulmonary oedema due to a combination of poor left ventricular function and OSA in a patient with a recent anterior myocardial infarction.

CASE REPORT

A 48-year-old man who has 20 years of diabetes on gliclazide, was admitted to another hospital on 25th May 1992 for sudden chest pain and was diagnosed to have an acute anterior myocardial infarction (Fig 1). After an uneventful 7-day hospital stay he was discharged with isosorbide dinitrate, verapamil and trimetazidine following a normal submaximal exercise electrocardiograph test. On 6th June 1992 he developed acute pulmonary oedema at 2.00 am which awoke him from his sleep. He was re-admitted to the same hospital and investigations which included serial electrocardiographs and serial cardiac enzymes did not reveal evidence of a myocardial reinfarction. The heart failure was thought to be partly contributed by verapamil which was then discontinued. Subsequently, he had another 2 episodes of acute pulmonary oedema which woke him up from sleep at 2.00 am on 10th June and at 5.00 am on 20th June 1992 respectively, for which he was treated at the same hospital. Again, investigations did not reveal evidence of reinfarction on these 2 occasions. He was then referred to us while he was taking isosorbide dinitrate, captopril, trimetazidine and frusemide.

The patient had been a habitual heavy snorer as long as his wife could remember since their marriage 21 years ago. He snored in all positions of sleep and his wife had witnessed that there were periods of silence in between the snoring during which he appeared to have stopped breathing. He suffered from excessive daytime sleepiness and tended to fall asleep easily when he was alone in monotonous situations. He also tended to feel sleepy behind the wheel and had to pull his car to the roadside on many occasions to take short naps when he needed to drive long distance. He did not suffer from early morning headache or feel unrefreshed on waking up in the mornings.

The patient used to smoke 60 cigarettes a day but had stopped smoking since his myocardial infarction. Alcohol consumption was discontinued 2 years ago. He had benign ventricular extrasystoles for 15 years which did not require treatment. His father was also a heavy snorer.

Physical examination revealed a narrow oropharynx with a low palate and flabby posterior pillars. There was no macroglossia or jaw deformity. The soft palate and uvula were not oedematous or inflamed. His body mass index was 23 kg/m^2. The blood pressure was 120/80 mmHg and the jugular venous pressure was not elevated. S3 gallop was present and crepitations were heard over the lung bases during the first 2 days in our hospital.

The haemoglobin was 143 g/l and the blood urea and serum electrolytes were normal. The chest radiograph showed slight cardiomegaly. The forced vital capacity was 2.92 L (99% predicted) and the forced expiratory volume in one second was 88% of forced vital capacity. Arterial blood gas analysis while he was breathing room air during wakefulness yielded the following result: pH 7.47, PO_2 10.2 kPa, PCO_2 5.2 kPa and bicarbonate 28 mmol/l. The fasting lipid profile and the thyroid function test were normal.

Exercise electrocardiography up to stage 5 of the Bruce protocol did not reveal any ST-T wave changes and the unifocal ventricular ectopics disappeared when the heart rate exceeded 165 per minute. The proximal segment of the left anterior descending artery was found to be stenosed on coronary angiography. The left ventricular end-diastolic pressure was 30 mmHg. Left ventriculogram showed a dyskinetic anterolateral and apical segments. The left ventricular ejection fraction was estimated to be 57%.

Overnight sleep monitoring was carried out with the use

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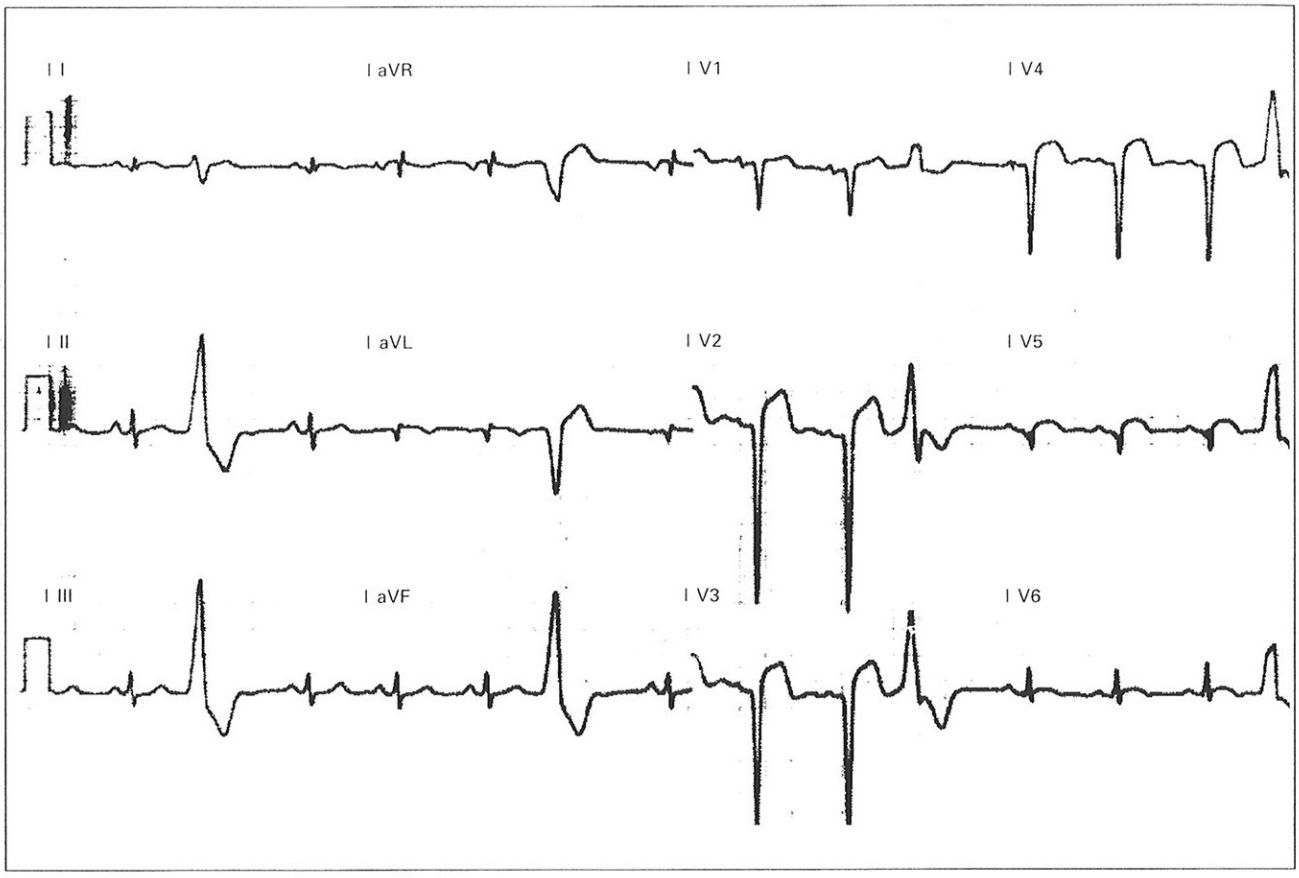
Fig 1 - Twelve lead electrocardiograph showing changes due to acute anterior infarct and uniform ventricular ectopics.



of the Vitalog, an ambulatory microprocessor based system which consisted of a portable recorder which continuously recorded, processed and stored data including breathing, heart rate, oxygen saturation, body position and arousal events. The overnight sleep study revealed moderately severe desaturation with arterial oxygen saturation between 88% and 96% and a nadir of 80%. While there were 5 more than 4% dips in oxygen saturation per hour and 27 more than 2% dips in oxygen saturation per hour, he experienced over 50 apnoea/hypopnoeas per hour of sleep. Together with the history of chronic heavy snoring and witnessed apnoeas, the overnight sleep study strongly supported the diagnosis of obstructive sleep apnoea syndrome.

Continuous ECG monitoring did not reveal any increase in frequency of his ventricular ectopy or any other forms of tachyarrhythmia during sleep. Simultaneous intrathoracic pressure monitoring using oesophageal balloon catheter and pulmonary capillary wedge pressure monitoring were not performed.

The patient did not desire the use of nasal continuous positive airway pressure (nasal CPAP) to treat the OSA. He was treated with enalapril, frusemide, isosorbide dinitrate and amlodipine.

DISCUSSION

Snoring per se has been considered more of social nuisance than a significant health problem. However, the fact that chronic heavy snoring is a clinical marker of obstructive sleep apnoea (OSA) means that apart from being an acoustic annoyance, snoring may be a health risk. The prevalences of OSA in habitual snorers have been reported to range between 35 and 64 percent[1,9,10]. The presence of OSA syndrome should be suspected when someone complains of excessive tiredness and/or excessive daytime sleepiness together with chronic nocturnal heavy snoring[11].

The primary pathophysiological event in the OSA syndrome is the recurrent upper airway occlusion during sleep because of a reduction in activation of the pharyngeal dilating musculature during sleep. Increased inspiratory efforts against the occluded airway during an obstructive apnoea only worsen the situation by creating more negative airway pressures. Occlusion persists until arousal occurs and increased tone of the pharyngeal muscles reopens the airway. The recurrent arousals result in a fragmented sleep which accounts for the daytime sleepiness.

One of the consequences of airway occlusion is progressive asphyxia and the accompanying hypoxaemia which can be of varying severity depending on the duration of apnoea[12]. The second consequence results from the intermittent falls in intrathoracic pressures to more negative values generated by inspiratory efforts that are made against an occluded upper airway. During obstructive apnoeas, intrathoracic pressures may fall as low as -60 cm H_2O. The decrease in intrathoracic pressure decreases right atrial pressure, thereby increasing venous return to the right side of the heart. By the mechanism of ventricular interdependence, increased right ventricular volume causes a shift in the interventricular septum that reduces left ventricular compliance, volume, and stroke output[3,13]. In addition, reductions in intrathoracic pressures increase left ventricular afterload by increasing the left ventricular transmural pressure gradient (which is the difference between left ventricular systolic pressure and intrathoracic pressure)[14].

Both the increase in left ventricular afterload and decrease

in left ventricular compliance could, in combination, further aggravate the elevated left ventricular end-diastolic pressure and therefore pulmonary capillary wedge pressure which would lead to pulmonary oedema[15]. Furthermore, reduction in intrathoracic pressure decreases pulmonary interstitial pressure which may also favour the formation of pulmonary oedema[16].

Despite the mild nocturnal oxygen desaturation in this patient the sleep-rated breathing disorder seemed to have a severe impact on his left ventricular function. His arousal following an apnoeic event might not be due to oxygen desaturation but rather to high subatmospheric pressure in his oropharynx[17], and the development of excessive tension in the contracting inspiratory muscles as suggested by Vicken et al[18]. A study by Wilcox and associates[19] shows that patients with the most vigorous inspiratory muscle contraction may have the least hypoxaemia while some patients with the most severe hypoxaemia may be related to poor ventilatory response during apnoea. It is likely that our patient developed wide negative swings in intrathoracic pressure due to vigorous inspiratory muscle contraction during obstructive apnoeas. This might have accounted for his relatively mild hypoxaemia and the ease with which he developed pulmonary oedema.

It is most likely that a combination of poor left ventricular function following his myocardial infarct and the OSA which have caused the recurrent pulmonary oedema in our patient. The fact that he did not develop acute pulmonary oedema prior to his myocardial infarct despite the presence of symptoms of OSA for many years implies that his OSA was not severe enough to cause pulmonary oedema in the presence of normal left ventricular function. His excellent effort tolerance on the treadmill means that poor left ventricular function alone was not the sole contributing factor to the episodes of pulmonary oedema. This emphasises the importance of being aware of the association of OSA and recurrent acute pulmonary oedema in patients with poor left ventricular function.

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