Central Sleep Apnea Presenting As Nocturnal Angina

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Abstract

Sleep disordered breathing is a common under diagnosed problem in patients with cardiac failure. Common forms of sleep disordered breathing seen in patients with cardiac failure are obstructive sleep apnea and central sleep apnea with Cheyne Stokes breathing. Central sleep apnea (CSA) can lead to greater cardiovascular outcomes and mortality. Patients with central sleep apnea are usually thin and do not snore heavily. Symptoms of CSA and cardiac failure often overlap. So a high index of suspicion is required to recognise CSA in cardiac failure patients. Standard overnight polysomnography should be considered in patients with risk factors for CSA such as male sex, age > 60, atrial fibrillation, hypoxia, left ventricular ejection fraction < 40%, NYHA class III - IV and refractory cardiac failure with standard medical management. Treatment of CSA can reduce cardiovascular mortality and improve quality of life.

Keywords: Central sleep apnea, Cheyne-Stokes breathing, nocturnal angina, cardiac failure, continuous positive airway pressure therapy.

Case Report

Elderly male diabetic, presented with sudden onset of grade 3 breathlessness which started at night, associated with orthopnoea and paroxysmal nocturnal dyspnoea. He was admitted in a nearby hospital, diagnosed as acute pulmonary oedema, treated with non-invasive ventilatory support, other supportive measures and was referred to our hospital for further evaluation. On further inquiry patient revealed history of recurrent episodes of central chest pain especially in night associated with sweating. He also gave history of recurrent episodes of breathlessness in night associated with orthopnoea and paroxysmal nocturnal dyspnoea (PND). History of pedal oedema was present. No history of wheezing. Patient gave history of snoring, but no history of excessive day time sleepiness. Past history revealed history of central chest pain in the past for which he underwent coronary angiogram and coronary artery bypass grafting (CABG). But his symptoms persisted even after CABG. He was a smoker. Epworth sleepiness score was 10.

On examination, his body mass index (BMI) was only 22.6 and neck circumference was 42 cms. JVP was elevated and pedal oedema present. Vitals stable. No craniofacial abnormalities or thyroid swelling. Mallampatti score was class 3. He had bilateral fine basal crackles.

Routine blood counts, hepatic, renal and thyroid function tests were within normal limits. Chest X-
ray was suggestive of pulmonary oedema. Echocardiography revealed left ventricular ejection fraction of 45-50%. Since the patient's symptoms persisted even after optimal management of cardiac failure and history of angina was mainly nocturnal, screening sleep study was done which showed significant desaturation. So detailed polysomnographic evaluation was done.

Polysomnographic report showing central sleep apnea with an Apnea hypopnea index of 49 (Figure 1).

![Figure 1](image)

He was diagnosed to have central sleep apnea syndrome. Continuous positive airway pressure titration study with 9 cm of water abolished all apnoeic events. Patient has been started on treatment with continuous positive airway pressure (CPAP) therapy and is on regular follow up. His nocturnal anginal episodes and paroxysmal nocturnal dyspnoea disappeared after initiation of treatment.

**Discussion**

Sleep disordered breathing occurs with increased frequency compared with general population especially in patients with left ventricular ejection fraction < 40% [1]. Sleep disordered breathing disorders in heart failure include central sleep apnea, CSA with Cheyne Stokes respiration (CSR), obstructive sleep apnea and mixed type (central and obstructive) [2,3].

Central sleep apnea is a disorder characterized by repetitive cessation or decrease of both airflow and ventilatory effort during sleep. CSA with CSR is a form of breathing in which central apneas or hypopnea alternate with periods of hyperventilation. Patients with CSA are usually thin and do not snore heavily. They may complain of fragmented sleep due to arousals, but may not complain of excessive day time sleepiness. So Epworth sleepiness scale is probably not useful in evaluating cardiac failure patients. Patients can complain of PND when they wake up during peak of ventilation after apnea which again can be attributed to cardiac failure. Moreover the symptoms resulting from CSA usually overlap with symptoms of cardiac failure such as nocturnal cough, orthopnea, PND, nocturia, sleep fragmentation and fatigue. So CSA remains occult in patients with cardiac failure.

Patients can report with chest pain, dyspnoea, syncope, hypertension, arrhythmias and history of repeated cardiac catheterizations in the past. But catheterizations may have disclosed less coronary artery occlusive disease than expected on the basis of patient's symptoms. Sleep apnea can lead to
nocturnal angina in patients with heart failure presumably due to extreme hypoxia induced by sleep apnea and catecholamine release.

Treatment:

1. Optimising treatment of cardiac failure
2. Positive airway pressure therapy [4]. Continuous positive pressure reduces frequency of central apneas, probably by preventing pharyngeal airway narrowing and occlusion during a central apnea.
3. If patient doesn't tolerate or fail in treatment with CPAP: If patient's ejection fraction is > 45%, Adaptive servo ventilation or Bi-level positive airway pressure with back up rate can be tried. If patients ejection fraction is <45%, nocturnal oxygen supplementation may be the best approach.
4. For patients who do not tolerate or benefit from positive airway pressure therapy or oxygen, may benefit from respiratory stimulant drugs such as Acetazolamide [5] or Theophylline.

Conclusion

A high index of suspicion is required to recognize CSA and CSR in patients with cardiac failure, because symptoms can often overlap. Since sleep disordered breathing contributes to progression of heart failure and death, early diagnosis and treatment is necessary to improve prognosis and quality of life in these patients.

References


