The Management of Co-Morbidities in Patients with Heart Failure – Obstructive Sleep Apnoea

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Abstract

Heart failure (HF) patients are older and frequently present with multiple co-morbidities. Co-morbidities worsen patient symptoms and may contribute to the progression of heart failure, increase mortality or limit the therapeutic response to treatment. Obstructive sleep apnoea (OSA) affects 2–4% of the adult population world-wide and is associated with similar risk factors to HF; meaning it is a frequent finding in HF patients, including HFrEF, HFrEF and HFrEF. OSA has consistently been shown to be associated with hypertension, coronary artery disease, arrhythmias, heart failure, and stroke. A thorough understanding of the diagnosis and treatment options of OSA is of paramount importance to the practising HF clinician. Patients may present to the HF specialist having been diagnosed by a formal sleep study or may be suspected of OSA because of symptoms of snoring, reports of obstructed breathing by the sleep partner or day-time sleepiness.

The mainstay of treatment for OSA is a positive airway pressure mask which can be used in mild moderate and severe OSA. The need for therapy should be discussed with the patient and if the AHI is above 15/hr then treatment is indicated to reduce this to below 15. This is a consensus recommendation and no adequately powered clinical trials have shown this improves either mortality or the risk of disease progression. Other options are discussed

Keywords: Heart Failure; Sleep Apnoea; Guidelines

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Introduction

Heart failure (HF) patients are older and frequently present with multiple co-morbidities. Co-morbidities worsen patient symptoms and may contribute to the progression of heart failure, increase mortality or limit the therapeutic response to treatment. Obstructive sleep apnoea (OSA) affects 2–4% of the adult population world-wide and is associated with similar risk factors to HF; meaning it is a frequent finding in HF patients, including HFrEF, HFrEF and HFrEF. OSA has consistently been shown to be associated with hypertension, coronary artery disease, arrhythmias, heart failure, and stroke.[1–2] A thorough understanding of the diagnosis and treatment options of OSA is of paramount importance to the practising HF clinician. Patients may present to the HF specialist having been diagnosed by a formal sleep study or may be suspected of OSA because of symptoms of snoring, reports of obstructed breathing by the sleep partner or day-time sleepiness.

OSA is caused by upper airway obstruction, usually associated with upper airway collapse. OSA manifests as intermittent airway occlusions, accompanied by consequent loss of airflow, arterial deoxygenation and arousal due to the stimulatory effects of chemoreflex firing secondary to the disturbed arterial blood gases. These obstructive apnoeas or hypopnoeas can occur throughout sleep and are somewhat irregular and are quite distinct form another sleep disordered breathing pattern seen in CHF, central sleep apnoea.[3] The mechanisms underlying the development of OSA in HF are no different to those in patients without HF, but a mixed apnoea pattern is more likely due to respiratory control system instability with subsequent oscillations of the arterial blood carbon dioxide levels (PaCO₂) and ventilatory drive due to lung congestion, reduced cardiac output, prolonged circulation times and elevated chemosensitivity.[4–5] Although large-scale trial evidence is lacking physicians are advised to treat HF patients with sleep disordered breathing as OSA if the hypoxias are predominantly obstructive in nature and as CSA (see central sleep apnoea chapter) if the hypoxias are predominantly central in nature.

Pathophysiology

Each OSA cycle of apnoea or hypopnoea is to an extent independent. The initiating factor is usually airway collapse causing an obstruction to airflow and consequent loss of effective breathing. Hypoxia and consequent arousal (and a surge in sympathetic activity) increases sufficiently to awaken the patient enough such that the airway is opened and effective breathing can resume. Frequent episodes of airway obstruction, hypoxia, and arousal during sleep impact significant sympathetic...
The mainstay of treatment for OSA is a positive airway pressure (CPAP) route to assess HF patients for the likelihood of OSA. Based on current guidelines, the PSG is diagnostic for OSA if respiratory monitoring demonstrates at least five obstructive hypoxaemic events per hour of sleep (or more than 15 in the absence of sleep related symptoms) and this frequency is defined as the apnoea-hypopnoea index (AHI).[8]

Treatment options for OSA
The mainstay of treatment for OSA is a positive airway pressure mask which can be used in mild moderate and severe OSA.[8] The need for therapy should be discussed with the patient and if the AHI is above 15/hr then treatment is indicated to reduce this to below 15. This is a consensus recommendation and no adequately powered clinical trials have shown this improves either mortality or the risk of disease progression. Compliance with mask therapy is limited and the need for on-going therapy and the willingness of a patient to continue may depend on whether there is a noticeable improvement in sleep related or daytime sleepiness symptoms induced by therapy. There are currently three primary types of these therapies: continuous positive airway pressure (CPAP), bi-level positive airway pressure (BiPAP) in which pressure levels decrease during exhalation, and BiPAP-adaptive servoventilation (ASV) in which the two pressures change due to sensors in the device.[9] These adverse effects of ASV in HFrEF patients with predominantly central sleep apnoea mean that this therapy should be reserved only for those with predominant OSA-type sleep apnoea (see chapter on CSA). Mask based therapy should be combined with life-style changes such as weight loss in the obese, regular exercise and the avoidance of alcohol and other sedatives prior to retiring for the night. Positioning the sleeping patient head-up can also help. Custom-made oral appliances that push the mandible forward or specially designed mouth guards can assist to a limited extent. Upper airway reconstructive surgery is occasionally used in severe cases unresponsive to or intolerant of mask therapy.

Declaration of Interest
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References