Sleep apnoea and the potential benefits of a good night’s sleep in patients receiving maintenance dialysis

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Premature cardiovascular (CV) death remains the leading cause of mortality in patients with endstage renal disease (ESRD). Modification of conventional CV risk factors, such as dyslipidaemia, has only limited impact on CV disease in this population [1, 2], an observation that has prompted the search for novel mechanisms and therapeutic targets. The publication in this issue [3] provides a potential link between sleep apnoea and fluid overload in patients receiving haemodialysis, and identifies a novel, potentially remediable, risk factor for CVD in this population.

Obstructive sleep apnoea (OSA) is a condition characterized by transient episodes of apnoea or hypopnoea due to obstruction of the upper airway during sleep. Sufferers are often awoken from sleep due to paroxysmal asphyxia with prompt resolution of hypoxaemia and hypercapnia on arousal. In the general population, OSA has been associated with adverse CV outcomes and is linked with systemic and pulmonary hypertension, nocturnal cardiac arrhythmias, coronary artery disease, heart failure and cerebrovascular disease [4]. In ESRD patients, a number of studies have implicated OSA with markers of CV disease and poorer survival [5]. Furthermore, OSA is significantly more common in ESRD patients compared with the general population, and the usual risk factors for OSA (such as obesity, craniofacial abnormalities, nasal congestion and smoking) do not usually apply to dialysis patients. Potential mechanisms implicated with higher prevalence of OSA in ESRD patients include the desensitizing effects of uraemia or metabolic acidosis on higher respiratory control centres. These hypotheses originate from observational studies demonstrating improvement in OSA after removal of these biochemical abnormalities by renal transplantation or more intensive dialysis. Furthermore, missing

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or shortening the duration of dialysis is associated with greater sleep disturbance (measured by wrist actimetry), suggesting an etiological role of uraemic toxin accumulation or fluid accumulation within the pharyngeal wall when patients are supine [6]. The relationship is not as well defined in patients with chronic kidney disease not requiring dialysis who have only a weak correlation between the severity of sleep disturbance and the degree of renal impairment.

In this edition, Elias et al. have attempted to further explain the higher rates of OSA in 20 haemodialysis patients by demonstrating a link with fluid overload [3]. Interstitial oedema commonly redistributes from the lower limbs to the neck during nocturnal recumbency (rostral fluid redistribution), and this can be artificially reproduced by applying external pressure to the legs to reduce peripheral fluid volume [7]. Using upper airway magnetic resonance imaging (MRI), the investigators have implicated mucosal water content and internal jugular vein volume (which are located lateral to upper airway walls) with severity of OSA. More severe OSA, measured using established polysomnography scoring criteria, was independently and significantly associated with greater upper airway mucosal water content and jugular vein volume.

This study highlights a number of important points. As the authors acknowledge, an association was only demonstrated between OSA and factors associated with fluid retention. No causal or pathological relationships were demonstrated, and this can only be achieved by robust, well-controlled longitudinal studies investigating the effect of reduction of measured parameters on OSA. Interventions to improve fluid control may include more frequent or nocturnal haemodialysis. Alternatively, ultrafiltration guided by bioimpedance spectroscopy may provide a practical method of assessing a patient’s fluid status in the haemodialysis unit [8]. Additionally, the factors associated with OSA in ESRD patients were different from the general population. In particular, obesity did not contribute to higher rates of OSA in ESRD patients. Similar results have been found in patients with heart failure strengthening the role of fluid accumulation, which is also common in this patient cohort [7]. Finally, the technique to measure internal jugular vein volume and upper airway mean water content using upper airway MRI remains relatively novel. Little data are available investigating the effect of interdialytic fluid shifts on the precision and accuracy of these measurements, and thus, care must be taken when extrapolating these results to ESRD population as a whole. In this study, in-laboratory polysomnography was performed to diagnose and determine the severity of OSA. Although this technique is considered to be the gold standard for assessment of OSA, it remains expensive and would be unpopular in haemodialysis patients who already attend hospital thrice weekly. Alternative methods of detection of OSA include portable monitoring, which measures fewer physiological parameters compared with polysomnography but overcomes difficulties of cost and convenience (portable monitoring can be performed at home or in any patient room). The American Academy of Sleep Medicine has recommended portable monitoring as an alternative to polysomnography in patients with a high pretest probability of moderate-to-severe OSA which would include ESRD patients [9]. Nocturnal pulse oximetry, which forms part of the polysomnography and portable monitoring, has limited usefulness in isolation given the high variability of sensitivity and specificity for OSA diagnosis.

These data provide further incentive for ensuring tight fluid control in ESRD patients. Increased interdialytic fluid gains have been associated with adverse CV outcomes due to a number of characteristics including alteration in left ventricular systolic and diastolic function and higher blood pressure [10]. Obstructive sleep apnoea provides an additional mechanism associated with adverse outcomes in ESRD patients which may be amenable to monitoring and intervention.


REFERENCES


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