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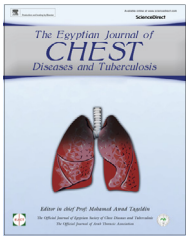

ORIGINAL ARTICLE

Sleep disordered breathing in patients with chronic kidney diseases: How far the problem?

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KEYWORDS

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Abstract *Background:* Sleep-disordered breathing (SDB) is highly prevalent in patients with advanced chronic kidney diseases (CKDs).

Objective: To describe and compare the prevalence, severity, and patterns of SDB and associated nocturnal hypoxia among patients with advanced CKD, hemodialysis (HD) patients, and control group.

Methods: Forty patients were recruited from outpatient nephrology clinics and hemodialysis units. Patients were stratified into two groups: conservative ($n = 25$), and HD ($n = 15$). 30 healthy individual enrolled as the control group. All participants completed polysomnography (PSG).

Results: Case control study of forty CKD patients (15 HD and 25 conservative) [13(86.7%) and 20 (80%) men, mean age 62.73 ± 5.43 and 55.76 ± 9.03 year, BMI 40.83 ± 8.75 and 36.12 ± 16.53 kg/m, mean ESS 18.46 ± 3.20 and 17.84 ± 2.79), respectively, and 30 healthy participants served as the control. The prevalence of SDB in CKD was 33/40(82.5%). In the conservative group, AHI was 148.84 ± 147/h, [80% obstructive, 13% central, and 5% mixed apnea]. Among these conservative groups with OSA patients, 56% had severe, 31% moderate, and 12.5% mild OSA. While in the HD group, AHI 133.26 ± 111/h, [84.6% obstructive, 7.7% central, and 7.7% mixed apnea]. Among these HD groups with OSA, 63% had severe, 27% moderate, and 9% mild OSA. GFR was significantly correlated with AHI and ODI ($r = -0.315$, $P < 0.05$, $r = -0.506$, $P < 0.001$) respectively. AHI correlated with urea concentration ($r = -0.094$, $P < 0.05$). Increased creatinine, and decreased eGFR were significant risk factors of severe OSA. Predictors that reduced renal function were, decreased TST, delayed latency to REM sleep, and increased AHI.

Conclusions: Severe OSA was highly prevalent among CKD. Urea was the stronger predictor of increased AHI.

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Introduction

The prevalence of chronic kidney diseases (CKDs) is increasing worldwide, and there is increasing evidence linking sleep-disordered breathing (SDB) with kidney disease [1]. Sleep-disordered breathing refers to a wide spectrum of sleep-related breathing abnormalities; those related to an increase in upper airway resistance include snoring, upper airway resistance syndrome, and obstructive sleep apnea–hypopnea syndrome [2]. CKD describes patients with a chronically decreased glomerular filtration rate (GFR) or other evidence of kidney damage. There are different levels of CKD, which provide the basis of an international classification system [3]. Sleep disorders are common in patients with chronic renal failure [4,5]. Sleep apnea occurs in 50% of patients with end stage renal disease (ESRD); [6] which is considerably higher than in the general population [7]. The potential importance of sleep disordered breathing in the CKD population is highlighted by the worldwide mortality in end stage renal disease (ESRD) patients as high as 20 per cent per annum. It is also possible that sleep apnea accelerates the deterioration of kidney function in patients with CKD either indirectly by increasing systemic BP, inflammatory cytokines and sympathetic nervous system activity all of which have been proposed to reduce kidney function [8–10] or directly through the effect of hypoxia on the kidney [11,12]. Severe hypoxic events in obstructive sleep apnea (OSA) might lead to kidney damage [13]. Sleep disorders tend to be under-recognized by renal healthcare providers [13]. Polysomnography remains the gold standard for diagnosing sleep-disordered breathing. Monitoring should be done in conjunction with a comprehensive sleep medicine evaluation. Portable monitoring can be performed in a patient who cannot be safely transported for laboratory polysomnogram [14].

Subjects and methods

Forty patients with CKD, stages 5 as defined by an estimated glomerular filtration rate [eGFR] < 15 mL/min/1.73 m according to the National Kidney Foundation Staging System [15], attended outpatient nephrology clinics and the hemodialysis unit of the Internal Medicine Department of the Menoufiya University Hospital from January 2011 to 2013. And 30 apparent healthy control subjects [eGFR] > 90 mL/min/1.73 m were invited to participate in the study. Exclusion criteria included supplemental oxygen use, tracheostomy, CPAP therapy and inability to give informed consent. The study was approved by the Health Research Ethics Board of the Menoufia University. Informed consent was obtained from all participants. Patients were stratified according to their estimated glomerular filtration rate (eGFR) at the time of the study visit and classified into two groups based on the National Kidney Foundation staging system as follows: CKD (eGFR <13 mL/min/1.73 m^2 not on dialysis, $n = 25$), ESRD (on hemodialysis, $n = 15$), eGFR was calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation [15]. All patients completed a standardized sleep history questionnaire which included a history of snoring, witnessed apnea during sleep and nocturnal choking, un-refreshing sleep, morning headaches, insomnia, and memory impairment. Additionally, the questionnaire surveyed demographic information and medical history, including a history of obesity (body mass index [BMI] $\geqslant$ 30 kg/m), hypertension, cardiovascular disease (angina, myocardial infarction, coronary artery bypass surgery, or congestive heart failure), cerebrovascular disease (stroke or transient ischemic attack), diabetes, chronic obstructive pulmonary disease (COPD), and medications. All patients completed the Epworth sleepiness score (ESS) [16]. The ESS is a self-administered questionnaire designed to measure the general level of daytime sleepiness. Patients rate on a scale of 0–3 how likely they are to fall asleep in 8 different situations that are commonly encountered. Total ESS scores range from 0 to 24, with higher scores indicating more subjective daytime sleepiness. Specifically, an ESS score >10 is considered indicative of subjective daytime sleepiness [16]. All CKD patients performed an attended, overnight PSG in the sleep laboratory unit in chest Department Menoufia University. Dialysis patients were asked to perform the overnight study on a dialysis-free day. Overnight PSG was performed using (Embla S4000 Medcare, Iceland).The system has Somnologica studio 3.3.2 software, electrodes and cablestor record the electroencephalogram (EEG), electrooculogram (EOG),electromyogram (EMG) of the chin and bilateral tibialis anterior muscles, and electrocardiogram (ECG). Airflow was measured using nasal and oral thermistors, and a nasal pressure transducer. Respiratory effort was monitored with respiratory inductive polysomnography belts with thoracic and abdominal locks. Oximetry was measured using a disposable finger probe (oximeter flex sensor 8000J) placed on the index finger. Snoring was recorded using snore microphones (piezo snoring sensor) attached to the neck. Body position was monitored using a body position sensor. All studies were analyzed by trained PSG technicians and sleep physicians using the criteria of Rechtscaffen and Kales [17], and in close concordance with scoring updates given by the American Academy of Sleep Medicine [18].The traditional Rechtscaffen and Kales terminology for the 5 sleep stages (i.e. stages 1, 2, 3, 4, and REM sleep, with stages 1 and 2 collectively referred to as ''light sleep'', stages 3 and 4 collectively referred to as ''deep sleep'') were used in this study. Apneas were scored when there was a complete cessation of airflow or $\geqslant$90% drop in the peak thermal sensor excursion for at least 10 s. Hypopneas were scored when there was a drop in nasal pressure by $\geqslant$30% of baseline lasting at least 10 s with a $\geqslant$4% desaturation from pre-event baseline, or when there was a drop in nasal pressure signal excursion by $\geqslant$50% of baseline lasting at least 10 s with a $\geqslant$3% desaturation from pre-event baseline. The apnea–hypopnea index (AHI) which is the number of apnea–hypopnea events per hour was determined after the exclusion of periods with movements, which were considered to be wake periods. An apnea without chest or abdominal movements was classified as CSA and apnea with chest and abdominal movements was classified as OSA. Hypopneas were considered to be obstructive when there was evidence of upper airway obstruction, such as snoring, paradoxical respiratory band movement or inspiratory flow limitation through the nasal cannula; central hypopnea, in contrast, was associated with in-phase respiratory movements and no evidence of inspiratory flow limitation. SDB was considered to be central if greater than 50% of apnea/hypopnea events were central and obstructive if greater than 50% were obstructive [19].