Obstructive sleep apnea in chronic kidney disease patients undergoing hemodialysis. Prevalence, severity, and treatment. A narrative review

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Abstract:

Background: Chronic kidney disease (CKD), also known as chronic renal insufficiency (CRI), can be defined by a glomerular filtration rate (GFR) less than 60mL/min/1.73m² associated with an albumin-to-creatinine ratio greater than 30mg of albumin per 1g of creatinine. CKD is a significant factor leading to a decline in the quality of life, increased morbidity, and a substantial reduction in life expectancy. Currently, it is estimated that there are 3.9 million patients worldwide on renal replacement therapy for CKD. Recent data indicates that in the United States of America (USA), more than 500,000 people suffer from renal failure or insufficiency. In Brazil, epidemiological data is incomplete and outdated. In 2016, there were 122,825 patients in Brazil on renal replacement therapy.

Objectives: This literature review aims to assess the prevalence, severity, and therapeutic recommendations for obstructive sleep apnea (OSA) in patients with CKD undergoing hemodialysis.

Methods: A literature review was conducted using PubMed, Web of Science, and SciELO databases with keywords including “Obstructive Sleep Apnea”, “Chronic kidney disease”, “End-stage renal disease”, “sleep disorders”, “hemodialysis”, “CPAP”, “Continuous positive airway pressure”, and “Physiotherapy.” Only studies published in the last twenty years and in the English language were included.

Results: Despite scientific evidence demonstrating a high prevalence of OSA in CKD patients undergoing hemodialysis, there are still a lack of studies examining the effects of Continuous positive airway pressure (CPAP) therapy on the clinical outcomes of these patients. In the scientific literature, only three randomized clinical trials were found that investigated the impact of CPAP therapy on improving kidney function. Consequently, there is a critical need for clinical studies to assess the effects of CPAP therapy in CKD patients undergoing hemodialysis. It is also essential to highlight the role of physiotherapy in managing sleep disorders and aiding in the adaptation and monitoring of patients undergoing CPAP therapy.

Conclusion: This literature review concluded that more randomized controlled studies are necessary to better define the optimal therapy for OSA in CKD patients, with the goal of preventing a decline in GFR progression, reducing morbidity and mortality, and enhancing overall quality of life.

Keywords: Respiratory sleep disorders; obstructive sleep apnea; end-stage renal disease; hemodialysis; physiotherapy; continuous positive airway pressure; CPAP.
BACKGROUND

Chronic kidney disease (CKD), also known as chronic renal insufficiency (CRI), can be defined by a glomerular filtration rate (GFR) less than 60mL/min/1.73m² associated with an albumin-to-creatinine ratio greater than 30mg of albumin per 1g of creatinine. CKD is a significant factor leading to decline in the quality of life, increased morbidity, and substantial reduction in life expectancy. Currently, it is estimated that there are 3.9 million patients worldwide on renal replacement therapy for CKD\(^1,2\). Recent data indicates that in the United States of America (USA), more than 500,000 people suffer from renal failure or insufficiency. According to the United States Renal Data System database in 2015, 124,411 new cases of end-stage renal disease (ESRD) were diagnosed, with an expected annual increase of 20,000 new cases, making it the ninth leading cause of death that year\(^2,3\).

In Brazil, epidemiological data is incomplete and outdated. In 1994, there were 24,000 patients with ESRD on renal replacement therapy, and by 2004, this number had risen to 59,153 patients. The annual incidence of CKD was 8%, with 18,000 new cases diagnosed in 2001. In 2004, the annual cost of the National Dialysis Program was R$1.4 billion Brazilian reais, and an estimated 1.5 million Brazilians were living with some degree of CKD\(^4\). In 2013, the prevalence of CKD in Brazil was 1.42%, rising to 2.68% among Brazilians aged 65 or older, with an estimated annual incidence of 119.8 cases per million inhabitants. In 2016, there were 122,825 Brazilians on renal replacement therapy\(^5\). In 2018, the estimated prevalence of patients on dialysis worldwide was approximately 298.4 per million inhabitants. The annual mortality among hemodialysis patients was 6.6% in Japan compared to 21.7% in the USA\(^6\).

While individuals between the ages of 40 and 44 can expect to live for about another 40 years, those at the same age with CKD on hemodialysis can only expect about another 10 years of life\(^5\). CKD patients have a significantly higher prevalence of various comorbidities compared to the general population, including sleep disorders\(^6\). In a study conducted in 2018 in São Paulo, it was found that 73% of CKD patients on hemodialysis were found to have obstructive sleep apnea (OSA). Other studies have shown prevalence rates of up to 50% in this patient population\(^7,9\).

The presence of sleep-disordered breathing (SDB), OSA, or central sleep apnea (CSA) in CKD patients increases the risk of cardiovascular events and overall mortality, regardless of the modality of renal replacement therapy and even in non-dialytic patients\(^6,7,9,10\). The presence of mixed sleep apnea can lead to a rapid decline in renal function in non-dialytic CKD patients, potentially resulting in stage alteration and the need for renal replacement therapy\(^9,10\). SDB is characterized by episodes of hypopnea and/or apnea that lead to repetitive hypoxia, elevated cytokine levels, and increased blood pressure, resulting in peripheral insulin resistance. These changes are risk factors for CKD progression, as diabetes and systemic arterial hypertension (SAH) are the main causes of CKD worldwide\(^10,11\). In addition to SDB, the presence of pruritus in cases of uremia and restless legs syndrome impairs sleep, compromises quality of life, and increases the risk of cardiovascular events, thereby increasing mortality in CKD patients\(^12,13\). Despite sleep disorders being a risk factor for CKD progression, CKD is also a risk factor for the development of sleep disorders\(^6\).

The high prevalence of SDB in patients with CKD on renal replacement therapy can be explained by uremic neuropathy and hypervolemia. A weight gain of more than 2kg between dialysis sessions is an independent risk factor for the development of SDB\(^14\). Despite their considerable impact on the life expectancy and quality of life of dialysis patients, sleep disorders present clinically differently in these patients from the general population, making their diagnosis and treatment challenging. This literature review aims to assess the prevalence, severity, and therapeutic recommendations for OSA in patients with CKD undergoing hemodialysis.
METHODS

A literature review was conducted using PubMed, Web of Science, and SciElo databases with keywords including “Obstructive Sleep Apnea”, “Chronic kidney disease”, “End-stage renal disease”, “sleep disorders”, “hemodialysis”, “CPAP”, “Continuous positive airway pressure”, and “Physiotherapy.” Only studies published in the last twenty years and in the English language were included. In the scientific literature, only three randomized clinical trials were found that investigated the impact of CPAP therapy on improving kidney function.

Definition and classification

CKD, also known as Chronic Renal Insufficiency (CRI), is defined according to the Kidney Disease Improving Global Outcomes (KDIGO) Foundation Guidelines as morphological or functional renal alterations lasting for more than three months, associated with a GFR less than 60 mL/min/1.73m² and an albumin-to-creatinine ratio greater than 30 mg of albumin per 1 g of creatinine. In stage G5 of the KDIGO classification, the patient is in ESRD, with a GFR of less than 15 mL/min/1.73m², requiring renal replacement therapy to survive\(^{(1,2,6)}\).

A respiratory event is considered apnea in adults if there is a ≥50% reduction in airflow compared to the graphed baseline prior to the event, as measured using an oronasal thermal sensor and/or pressure cannula, positive pressure airflow device, or an alternative apnea sensor. This reduction must last for at least 10 seconds or more to be considered apnea. To be classified as hypopnea, there should be a 30% or greater reduction in respiratory airflow for at least ten seconds, accompanied by a decrease of four percent or more in oxygen saturation\(^{(15)}\). The Apnea-Hypopnea Index (AHI) per hour of sleep is used to classify the severity of sleep apnea based on the number of events. A normal index ranges from 0 to 5 events per hour, mild from 5 to 14.9 events/hour, moderate from 15 to 30 events/hour, and severe with more than 30 events/hour\(^{(6,16)}\).

Epidemiology

The prevalence of OSA and CSA in the general population varies according to sex and age, being more common in men and older people. For moderate to severe disorders, the prevalence is in the age group of 30 to 49 years, at 10% for men and 3% for women. In the age group between 50 and 70 years old, there is a 17% prevalence in men and 9% in female patients\(^{(17)}\). In the population with CKD, the prevalence of sleep disorders is significantly higher, and the prevalence increases according to the worsening of GFR. According to some studies, this prevalence varies from 50 to 73% in stage 5 patients according to KDIGO\(^{(1,7,8,18,19)}\). Patients with CKD with a GFR greater than 59mL/min/1.73m² have a prevalence of 27% for moderate to severe disorders, while patients with a GFR of 59 to 15mL/min/1.73m² have a prevalence of 41%, and in renal replacement therapy patients (GFR less than 15mL/min/1.73m²) the prevalence rises to 57%. The influence of weight, age, sex, and comorbidities was excluded in the studied population, leaving CKD as the main influence\(^{(18,20)}\).

Sleep apnea as a risk factor for CKD and worsening renal function

According to the scientific literature, it is known that the presence of OSA can increase the risk of developing CKD and even lead to progressive impairment of renal function in these patients\(^{(6)}\). In a cohort study involving 6,866 patients, Lin et al., demonstrated that patients with OSA had an odds ratio of 1.37 for developing CKD, and they developed CKD earlier than patients without OSA\(^{(21)}\). In an intriguing cohort study involving three million veterans, Molnar et al., observed a strong association between OSA and CKD. According to the study, patients with OSA had about twice the odds of developing CKD compared to patients without OSA\(^{(22)}\). Besides increasing the risk of developing CKD, the presence of sleep disorders also raises the risk of worsening renal function.
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In a prospective study of GFR with an 11-year follow-up, Jaussent et al., demonstrated that the presence of excessive daytime sleepiness (EDS) and periodic leg movements (PLM) increased the risk of worsening renal function by 1.7 and 2 times, respectively. In this study, Jaussent et al., showed that severe apnea increased the risk of worsening renal function independent of the presence of diabetes Mellitus, systemic arterial hypertension (SAH), smoking, body mass index (BMI), age, gender, and the presence of EDS. The presence of EDS may result from reduced nighttime sleep duration, which can cause low-grade systemic inflammation, increasing the likelihood of worsening or developing cardiovascular disease and consequently worsening renal function.

In patients with SAH without CKD, the presence of OSA increased the risk of worsening GFR and progressing to CKD by 1.21 times more than in patients without OSA.

Sakaguchi et al., in a multicenter retrospective study, assessed the association between nocturnal hypoxia and the progression of CKD in patients classified as moderate and severe according to the KDIGO stages 3 and 4. Patients with a BMI above 25kg/m² were excluded to avoid obesity as a confounding factor. Among the patients involved in the study, those who had moderate to severe nocturnal hypoxemia (fifteen or more points on the oxygen desaturation index) experienced a three to four times faster decline in renal function.

Among the theories proposed, it is known that nocturnal hypoxia can lead to hyperactivation of the renin-angiotensin-aldosterone system (RAAS), which in turn can cause direct renal injury with increased glomerular pressure and worsening of renal function. This hyperactivation can be evidenced by the lack of an increase in effective renal plasma flow during the administration of angiotensin II tests in patients with OSA. The RAAS hyperactivation due to severe intermittent hypoxia (IH) (nighttime peripheral oxygen saturation below 90% due to OSA) leads to direct renal damage. Additionally, it results in SAH due to water and sodium retention.

Adding to this clinical picture of RAAS hyperactivation is the role of the sympathetic autonomic nervous system (SANS). In an interesting experimental study with rats, IH for approximately seven hours daily was shown to activate the SANS through carotid chemoreceptors, increasing the action of the RAAS through type 1 angiotensin II receptors.

Patients with IH nocturnal tend to have higher blood pressure levels, with the elevation being related to increased SANS activity that occurs both during sleep and wakefulness. This increase in blood pressure is related to peripheral vasoconstriction caused by the action of the SANS, SAH associated with vasoconstriction causes damage to renal function through tubulointerstitial injury, as well as injury to the renal microvasculature (fibrosis of peritubular capillaries), resulting in mitochondrial impairment and apoptosis.

IH in renal tissue alone is capable of generating oxidative stress, with an increase in the production of oxygen-free radicals that cause tissue damage and endothelial dysfunction, stimulating the activation of fibroblasts leading to fibrosis of renal tissues, as well as hypertrophy of tubular epithelial cells and dilation of the glomerulus. The paradox between CKD and OSA as cause and effect still persists, as OSA appears to be a risk factor for CKD development and a contributor to the worsening of CKD patients, but CKD is also a risk factor for the development of OSA. According to various published studies, it is known that the prevalence of OSA in CKD patients undergoing renal replacement therapy is approximately 55%. It has also been demonstrated that when dialysis sessions (peritoneal or hemodialysis) are intensified, there is an improvement in the OSA condition with a reduction in the AHI. The same improvement is observed after kidney transplantation. Based on these data, it becomes clear that the clinical picture of CKD can be an independent cause of OSA.
Patients with CKD present a metabolic acidosis that stimulates bulbar chemoreceptors, leading to hyperventilation with hypocapnia, causing instability in central respiratory control due to a reduction in pCO$_2$ below bulbar activation levels. In association with central dysregulation and reduced chemosensitivity, there is sodium and water retention, both due to impaired renal excretion and hyperactivation of the RAAS, causing pharyngeal narrowing and an increase in tongue volume, leading to upper airway obstruction$^{(30, 39)}$. The accumulated hypervolemia in the lower limbs during the day shifts to the chest and neck region when the patient assumes the supine position to sleep. The migration of excess extracellular fluid rostrally (from the lower limbs to the trunk, cervical region, and cranial pole) results in external compression of the upper airway, consequently reducing its caliber$^{(39-42)}$.

According to various studies, neck circumference is a predictive factor for the magnitude of OSA related to fat accumulation and/or hypervolemia$^{(42-44)}$. The displacement of fluid from the lower limbs to the thoracic and cervical region also affects the extravascular lung space, stimulating capillary pulmonary receptors, leading to a cycle of hyperventilation and predisposing to OSA and especially CSA$^{(40, 41, 43, 44)}$. Studies evaluating the diameter of the internal jugular vein using magnetic resonance imaging have shown that the greater the vein’s distension (caused by increased intravascular volume), the worse the sleep apnea condition. Other markers that are also proportionally related to the magnitude of hypervolemia and consequently OSA include brain natriuretic peptide and cardiothoracic index, as well as the diameter of the inferior vena cava$^{(40, 42, 44, 45)}$.

The presence of uremic neuropathy in CKD patients reduces the sensitivity of the upper airways, increasing the likelihood of collapse and subsequent obstruction. Additionally, uremic myopathy contributes to a reduced exhaustion threshold of ventilatory muscles, leading to a decrease in the tone of these muscles, thereby contributing to sleep-related respiratory events$^{(46, 47)}$.

Despite the aforementioned, in a meta-analysis conducted by Kanbay et al., in 2023, the authors assessed the changes in polysomnographic (PSG) parameters in patients undergoing kidney transplantation and demonstrated that there was no improvement in the AHIl, total sleep time, or rapid eye movement (REM) sleep time even after transplantation$^{(48)}$. For CKD patients on peritoneal dialysis, who are older, male, have coronary artery disease, dyslipidemia, chronic obstructive pulmonary disease (COPD), or SAH, the risk of OSA is even higher$^{(49)}$.

Clinical picture of OSA in patients with CKD

Many CKD patients with OSA are not initially diagnosed. The symptoms of OSA that affect the population without kidney disease are generally not present in CKD patients. Furthermore, sleep-related symptoms are present in CKD patients whether they have OSA or not, with the exception of EDS. Symptoms such as EDS, snoring, choking or witnessed apnea, non-refreshing sleep, morning headache, and memory difficulties are more common in the population with OSA without CKD than in CKD patients. Therefore, CKD patients with coexisting OSA may initially go undiagnosed due to the absence of characteristic symptoms. Additionally, it should be noted that patients with ESRD have lower BMI and neck circumference values than OSA patients without CKD$^{(50)}$.

Both BMI and neck circumference are significant risk factors for the development of OSA and are used in screening questionnaires for OSA. However, these screening tools are less applicable in the CKD population$^{(30, 50, 51)}$. Other factors that may contribute to masking the symptoms of OSA in CKD patients include the presence of various comorbidities, the use of various medications, and the typical symptoms of CKD itself, such as fatigue and poor sleep quality. Insomnia, PLM and, restless legs syndrome (RLS) are also common symptoms in CKD$^{(13, 30, 52, 53)}$. 
Given the high prevalence of sleep disorders in CKD, the scarcity of symptoms, and the ineffectiveness of screening questionnaires in this population, the use of highly sensitive diagnostic tests such as overnight polysomnography or cardiopulmonary sleep monitoring tests becomes important for detecting OSA, as it increases overall mortality and is a risk factor for cardiovascular events in CKD.(7, 54).

The use of CPAP in CKD patients and its impact on kidney function

The use of CPAP is the first-line treatment for patients with moderate to severe OSA, effectively maintaining open airways during sleep and preventing hypopnea and apnea events.(55,56). Some studies have shown that using CPAP in stage 5 CKD patients for one week decreased renal plasma flow, which can be seen as a partial reversal of renal hyperperfusion, one of the contributing factors to declining eGFR in CKD.(55,56).

Moriya et al., in a retrospective study, correlated the ratio of creatinine to urinary N-acetyl-β-D-glucosaminidase (UNCR) with worsening oxygen desaturation index (ODI) in patients with OSA and CKD. Additionally, CPAP therapy was applied to patients with an AHI of 20 or higher. The use of CPAP reduced diastolic blood pressure, decreased UNCR, and maintained stable eGFR in these patients.(57).

Rimke et al., initiated a randomized controlled clinical study applying CPAP therapy to CKD patients with OSA in the study group, in addition to standard therapy for other comorbidities and CKD, to assess changes in eGFR over one year of treatment and compare them to a control group.(58). The results of the study are not yet available. In a case report of a 51-year-old peritoneal dialysis patient, one year of CPAP use reduced EDS and improved sleep quality, cognitive ability, and quality of life, but no changes in blood pressure levels were evaluated.(59). Fu et al., published a meta-analysis in 2023 that assessed the use of CPAP in non-dialysis CKD patients using randomized clinical trials, a large sample (519 in total), and CPAP use for at least 4 hours per night.(60). The use of CPAP did not effectively modify eGFR.

In patients with OSA without impaired kidney function, the use of CPAP has been shown to reduce the overactivation of the RAAS, as evidenced by increased renal flow after angiotensin II administration, decreased serum aldosterone levels, and reduced proteinuria. Additionally, blood pressure levels in these patients decreased after 30 days of CPAP use.(61). Since OSA in CKD patients causes a gradual decline in kidney function, treating the respiratory disorder could prevent the progression of CKD caused by the respiratory disorder. An interesting study conducted by Marrone et al., compared the decline in eGFR in patients with untreated OSA, those treated with standard CPAP, and those treated with auto-adjustable CPAP.(62). After 541 days of follow-up, the authors found that the decline in eGFR was greater in the untreated and auto-adjustable CPAP-treated groups than in the standard CPAP-treated group.

There are few studies in the scientific literature that have assessed the effectiveness of CPAP in preventing or reducing the decline in eGFR in CKD patients. In a meta-analysis conducted in 2017, Chen et al., demonstrated that the use of CPAP therapy in CKD patients did not lead to a decline in eGFR.(63). When subgroups were evaluated, the authors observed a significant improvement in eGFR in patients aged 55 and older and those treated for 3 months or more. Conversely, in one arm of the SAVE (Sleep Apnea Cardiovascular Endpoints) study, patients with cerebrovascular or coronary artery disease, both with moderate to severe OSA, were randomized into two groups: one receiving standard treatment for the underlying disease and the other receiving standard treatment plus CPAP. The two groups were followed up for 4.4 years, with no difference in terms of eGFR decline, the albumin-to-creatinine ratio, or the number of severe renal complications. However, the study was not primarily designed to evaluate renal repercussions or complications. The study design also failed to include patients whose initial eGFR was normal, preventing the evaluation of OSA control as a protective factor against CKD progression.(64).
In another study, Puckrin et al., compared CKD patients with eGFR less than 59 mL/min/1.73m² who used CPAP therapy\(^{(69)}\). Among the patients involved in the study, those who used CPAP for more than 4 hours per night and on more than seventy percent of nights showed a reduction in eGFR decline and worsening proteinuria compared to patients who used CPAP for shorter periods or less frequently.

**FINAL CONSIDERATIONS**

Despite the high prevalence of sleep disorders in CKD patients, the symptoms they may present are nonspecific and can complicate diagnosis. When diagnosed, CKD patients with OSA should be treated with CPAP, as there are studies demonstrating the stabilization of renal function and proteinuria in treated patients, although there is no randomized controlled trial providing clear evidence of the benefit of this therapeutic modality in maintaining renal function.

In contrast, there is evidence that intensifying dialysis therapy, especially with the use of nocturnal dialysis sessions, improves the respiratory status of these patients with a reduction in AHI. However, there are authors who disagree with this assertion, and such intervention requires resources and may generate long-term complications. Moreover, it is feasible only for patients already on dialysis.

According to the literature, there is a worldwide consensus that CPAP is the gold standard in the therapeutic approach to moderate to severe OSA. The effects of its use include improving cognitive function, daytime drowsiness, subjective sleep quality, mood, and health-related quality of life\(^{(66-68)}\).

Patients with CKD have a high risk factor for cardiovascular disease, often progressing to ESRD. At this stage, the only therapeutic options are dialysis or kidney transplant, with significant financial implications for patients and the yours health conditions\(^{(69)}\). Given the high prevalence of CKD in the world population, it would be of great importance to identify new risk factors for the development of the disease and new clinical approaches. In this scenario, OSA, observed in up to 40% of patients with CKD\(^{(69)}\), has contributed to the progression of the disease due to IH and sleep fragmentation caused by apneic events, as demonstrated in the scientific literature in experimental animal models\(^{(70)}\) and also in studies involving patients\(^{(71)}\).

Even with the scientific demonstration of the high prevalence of OSA in patients with CKD undergoing hemodialysis, there is still a lack of studies demonstrating the effects of CPAP therapy on the clinical evolution of these patients. In the scientific literature, only three randomized clinical trials were found that verified the effects of using CPAP on improving kidney function\(^{(58, 64, 72)}\). Therefore, clinical studies that show the effects of using CPAP in CKD patients undergoing hemodialysis are extremely necessary. It would be extremely important to draw attention to the role of physiotherapy in sleep disorders in the adaptation and monitoring of these patients undergoing CPAP therapy.

Through this literature review, it can be concluded that more randomized controlled studies are needed to better define the preferred therapy for OSA in patients with CKD, to prevent the progression of the fall in GFR, and consequently, reduce morbidity and mortality while improving the quality of life.

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