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ORIGINAL ARTICLE

Sleep apnoea syndrome prevalence in chronic kidney disease and end-stage kidney disease patients: a systematic review and meta-analysis

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ABSTRACT

Background. Several studies have examined the frequency of sleep apnoea (SA) in patients with chronic kidney disease (CKD), reporting different prevalence rates. Our systematic review and meta-analysis aimed to define the clinical penetrance of SA in CKD and end-stage kidney disease (ESKD) patients.
Methods. Ovid-MEDLINE and PubMed databases were explored up to 5 June 2023 to identify studies providing SA prevalence in CKD and ESKD patients assessed by different diagnostic methods, either sleep questionnaires or respiration monitoring equipment [such as polysomnography (PSG), type III portable monitors or other diagnostic tools]. Single-study data were pooled using the random-effects model. The Chi^2 and Cochrane-I^2 tests were used to assess the presence of heterogeneity, which was explored performing sensitivity and/or subgroup analyses.
Results. A cumulative analysis from 32 single-study data revealed a prevalence of SA of 57% [95% confidence interval (CI) 42%–71%] in the CKD population, whereas a prevalence of 49% (95% CI 47%–52%) was found pooling data from 91 studies in ESKD individuals. The prevalence of SA using instrumental sleep monitoring devices, including classical PSG and type III portable sleep monitors, was 62% (95% CI 52%–72%) and 56% (95% CI 42%–69%) in CKD and ESKD populations, respectively. Sleep questionnaires revealed a prevalence of 33% (95% CI 16%–49%) and 39% (95% CI 30%–49%).
Conclusions. SA is commonly seen in both non-dialysis CKD and ESKD patients. Sleep-related questionnaires underestimated the presence of SA in this population. This emphasizes the need to use objective diagnostic tools to identify such a syndrome in kidney disease.

Keywords: chronic kidney disease, end-stage kidney disease, meta-analysis, sleep apnoea, systematic review

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INTRODUCTION

Sleep apnoea (SA) is one of the most clinically important forms of sleep-related breathing disorders, possibly contributing to the deterioration of kidney function and the high incidence of cardiovascular (CV) sequelae in chronic kidney disease (CKD) patients [1–3]. This population is often found to have concomitant sleep disorders, including but not limited to SA syndrome, such as insomnia, excessive daytime sleepiness or periodic limb movement disorder, all of which have a higher prevalence in CKD patients [4, 5] than in the general population [6].

SA is defined as intermittent episodes of partial or complete interruption of respiratory airflow during sleep; it can be distinguished in central SA, a transient abolition of the neural drive to respiratory muscles, obstructive SA, characterized by periods of breathing cessation (apnoea) and periods of reduced breathing (hypopnoea), or a mixed form [2].

SA has a relevant economic burden on the adult population. In a recent cost-of-illness analysis with a societal perspective, 26 clinical (e.g. diabetes) and non-clinical (e.g. car accidents) conditions were found to be significantly influenced by SA, contributing to an economic burden ranging from €10.7 to €32.0 billion/year in Italy [7]. In the same study, the cost of impaired quality of life due to SA under-treatment was between €2.8 and €9.0 billion/year. Knowing the prevalence of the disease in high-risk populations, such as CKD and ESKD, is of obvious importance for health planning.

Several studies have examined the frequency of SA in patients with CKD and ESKD, reporting a wide ranging prevalence. Although there is no consensus regarding the occurrence of this syndrome in CKD, comparative studies showed that the prevalence of SA in CKD and ESKD is much higher than that observed in the general population [3]. The wide range of SA prevalence in CKD and in ESKD patients is probably due to the inherent variability of this population, which is characterized by the highest burden of comorbidities among major chronic diseases [8], the use of different definitions of the disease [9] and the heterogeneity in the diagnostic methodologies. These methodologies include polysomnography (PSG), a highly reliable method, home sleep studies and less reliable tests like registration of pulse oximetry overnight and clinical questionnaires, and variable reliability of these tests makes it difficult to ascertain the true prevalence of SA in CKD and in ESKD [9, 10]. Several validated questionnaires (e.g. Berlin and STOP-Bang) have been proposed to screen people at risk for obstructive SA [11, 12]. These questionnaires, although sensitive (about 85%), have low specificity (50%–60% in CKD and 50%–70% in ESKD) for SA as defined by the gold standard (PSG) [13].

PSG represents the gold standard for diagnosing SA [14, 15]. This method records sleep time, the average number of apnoeas and hypopnoeas per hour of sleep, and sleep efficiency, and assesses SA severity, categorizing the disorder into mild SA, defined as an apnoea–hypopnoea index (AHI) of 5–15 events/h, moderate (15–30 events/h) and severe (>30 events/h). However, PSG is a time-consuming, expensive investigation [16, 17] and it is applied only in selected cases. Simpler and cheaper home sleep tests (a sleep recorder equipped with an oximeter to record arterial oxygen saturation and heart rate, a pressure transducer to record nasal airflow and possibly a microphone to record snoring) have been developed to assess SA in CKD as an alternative to PSG [18, 19]. Pulse oximetry records hypoxia episodes during sleep [2] rather than episodes of apnoea and hypopnoea, and nocturnal hypoxemia (a surrogate marker of SA) is a predictor of the CV sequelae commonly found in this population [18–20]. However, nocturnal hypoxemia does not necessarily reflect SA [21] and may miss up to 50% of diagnoses [13, 22].

This comprehensive, evidence-based review provides a systematic analysis of the literature aimed at defining the clinical prevalence of SA in CKD and ESKD population as well.

MATERIALS AND METHODS

Data source and search strategy

This systematic review followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines [23] and was conducted according to a pre-published protocol (crd42020151452) [24]. Due to the length of the originally planned systematic review and meta-analysis, we decided to split it in two different systematic reviews. For this reason, pre-specified outcomes reported in the protocol (i.e. the relationship between SA and renal function decline and the risk of mortality and/or adverse CV outcomes) will be presented in a second manuscript.

Ovid-MEDLINE and PubMed databases were searched for English-language articles without time restriction up to 5 June 2023 by focused, highly sensitive search strategies (Supplementary data, Table S1). Bibliographies of relevant studies and reviews were screened for additional articles. Two authors designed and performed the literature search (A.P., D.B.).

Study selection and data extraction

We included any interventional (randomized and non-randomized controlled trials or uncontrolled trials) or observational (prospective or retrospective study) studies providing information on the prevalence of SA in CKD or ESKD patients. Studies were included if matching the following criteria:

(i) Adult CKD patients. CKD was defined according to the National Kidney Foundation-Kidney Disease Outcomes Quality Initiative (NKF KDOQI) guidelines [25], by a reduced glomerular filtration rate (GFR <90 mL/min/1.73 m^2) and/or pathological evidence of kidney damage (persistence of urinary abnormalities, such as albuminuria, proteinuria or haematuria) with a preserved GFR (≥90 mL/min/1.73 m^2), for at least 3 months. Studies performed on patients needing haemodialysis (HD) or peritoneal dialysis (PD) were also included.

(ii) Presence of SA, identified by different diagnostic methods, either sleep questionnaires or respiration monitoring equipment (classical PSG in the sleep laboratory, Type III portable monitors as identified by the American Academy of Sleep Medicine (AASM) including the Apnea Link and other devices). The presence and severity of SA were determined by the AHI and/or respiratory disturbance index (RDI) during sleep respiration monitoring. Standard definition of apnoea is according to the AASM criteria 2012 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3459210/) [26], recently reviewed by Gottlieb and Punjabi [27]. For Berlin questionnaire, a positive score (≥2 points) in at least two out of the three categories (symptom domains) was deemed high risk for SA (https://www.ukcpap.co.uk/info-questionnaire-berlin-score.php) [11]. In STOP-Bang questionnaire, a total score of three or more ≥3/8 places the individual at high risk (http://stopbang.ca/osa/screening.php) [28].

Studies were excluded if they (i) dealing with kidney transplant patients and (ii) not providing data on SA prevalence.

Studies where at least part of the population fulfilled the above criteria were included in the review.

Two investigators (A.P., D.B.) independently screened titles and abstracts, excluding studies not pertinent to the topic, and assessed the retrieved full texts to determine eligibility according to the pre-specified inclusion/exclusion criteria. Possible discrepancies in study judgments were solved by another author (F.M.) who was not involved in the selection process. Reviews, editorials, letters, case reports and studies performed on children (age <18 years) were excluded from qualitative analyses but screened for potential additional references. If more than one article from a single study was retrieved, eligible data from all reports were considered, but each study was included only once.

Data analysis

Aggregating single-study prevalence data, the pooled SA prevalence with 95% confidence intervals (CIs) was obtained. Data were pooled using the random-effects model, and also analysed with the fixed-effects method to guarantee the strength of the model. To maximize the information, data provided by single studies or in a descriptive way were reported narratively. The Chi^2 test on N-1 degrees of freedom, with an alpha of 0.05 considered for statistical significance and the Cochrane-I^2 [29] were used to assess the presence of heterogeneity. I^2 values of 25%, 50% and 75% were assumed to correspond to low, medium and high levels of heterogeneity, respectively. Potential sources of heterogeneity were explored performing sensitivity and subgroup analyses according to SA diagnostic criteria (questionnaires or instrumental monitoring), different cut-off values for SA diagnosis and population characteristics (CKD stages). Meta-regression analyses were performed for identifying possible effect modifiers. Statistical analyses were performed by two authors (A.P., G.D.) using Stata/IC (Version 13.1, StataCorp LP, TX, USA).

Quality and risk of bias assessment

The methodological quality of longitudinal cohort and cross-sectional studies was assessed in accordance with a 14-item checklist recommended by the National Heart, Lung, and Blood Institute (National Institutes of Health) [30]. Study quality was classified as follows: poor = 0–4; fair = 5–9; high quality = 10–14.

RESULTS

Search results

The systematic literature search produced a total of 1666 potentially relevant references. Seven additional citations were added by personal search. By titles and abstracts screening, a total of 1457 citations were excluded for various reasons: search overlap (n = 583), study population/problem not pertinent (n = 562) or review articles (n = 312). Amongst the 216 articles selected for full-text evaluation, 92 were excluded because: (i) they dealt with the wrong population, outcome, or the topic was not pertinent (n = 73) or (ii) they were review articles (n = 19). Finally, 124 articles (140 566 participants) were included in the review, of which 5 reported on nocturnal hypoxemia with the pulse oximeter.

A total of 119 articles referring to 107 studies (140 279 participants) provided suitable numerical data to be pooled in cumulative meta-analyses. Figure 1 shows the flow diagram of the studies' selection process.

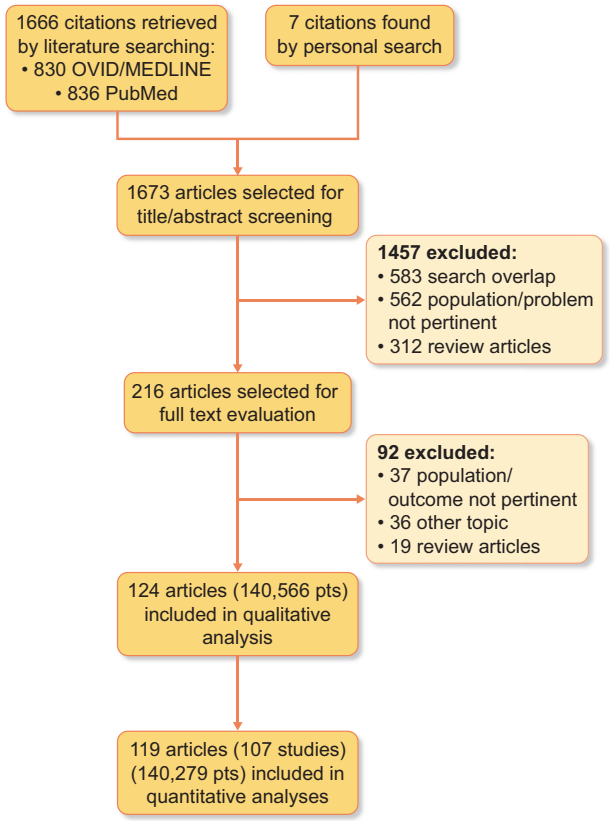

Figure 1: Study selection flow.

Study characteristics

Most studies were cross-sectional (n = 69) [4, 9, 13, 31–96]. Twenty-three studies were prospective observational cohort studies [1, 97–118], while nine had an interventional design [119–127]; only three studies were retrospective [128–130]. Three studies had a case–control design [17, 131, 132].

Twenty-two studies (3870 patients) [4, 32, 34–37, 39–44, 47, 50, 95, 96, 101, 104, 105, 117, 119, 131] focused on non-dialysis CKD population (NFK-KDOQI 1–5ND), while 76 (134 603 patients) were conducted on HD and/or PD patients [1, 9, 31, 33, 52–94, 97, 99, 100, 102, 103, 106–116, 118, 120–128, 130, 132, 133]. Nine studies (1272 patients) provided information on both populations [13, 17, 38, 45, 46, 48, 49, 51, 98].

Eighty studies included a no-selected or random selected population [1, 4, 9, 17, 22, 32, 33, 35, 36, 38–41, 43, 45, 46, 49, 52–54, 56–71, 73–77, 80–86, 88, 89, 91, 93, 95–105, 107–113, 115, 117, 119, 122, 123, 125, 130–133], while 27 studies enrolled pre-selected individuals with a potential risk for SA [31, 34, 37, 42, 44, 47, 48, 50, 51, 55, 72, 78, 79, 87, 90, 92, 94, 106, 114, 116, 118, 120, 121, 124, 126–128]. The study sample size was variable, spanning from 6 [51] to 1452 [104] for CKD and from 7 [127] to 81 538 [128] for dialysis populations.

The vast majority of studies (n = 78) [4, 9, 13, 17, 32, 35–44, 46–51, 53–55, 57, 60, 67–71, 73, 74, 77–80, 82–85, 87–89, 91–94, 97–103, 105–108, 110–114, 116–127, 130, 131] assessed prevalence and severity of SA with different sleep monitoring devices (such as PSG, cardiopulmonary/cardiorespiratory monitoring, type III portable monitors or other diagnostic tools). Specifically, PSG was employed in 65 studies [4, 9, 17, 32, 37–40, 42–44, 46–51, 54, 55, 57, 60, 67–71, 73, 77–80, 82, 87–89, 91, 92, 94, 97–103, 105–107, 111–114, 116–127, 130]. Thirty studies had available data on

SA risk based on different sleep-related questionnaires, such as Berlin, STOP-Bang or other sleep and health questionnaires [1, 31, 33, 34, 38, 45, 52, 56, 58, 59, 61–66, 75, 76, 81, 86, 90, 93, 95, 96, 103, 104, 115, 128, 132, 133]. Five studies (323 patients) [18, 19, 52, 134, 135] documented arterial O_2 desaturation with pulse oximeter, recording hypoxia episodes [4% oxygen desaturation index (ODI) >5 events/h] during sleep.

The main characteristics of the studies reviewed are summarized in Supplementary data, Table S2.

Study quality and risk of bias

The vast majority of the studies ($n = 83$) had a fair quality score (mean of 7.4 points; range 5–9) implying that they were susceptible to some bias which was deemed insufficient to invalidate the results. Fourteen studies had a good quality score (mean of 10.4 points; range 10–12); these studies had the least risk of bias, and results are considered to be valid. Only seven studies had a poor rating score (mean of 3.9 points; range 3–4) indicating significant risk of bias. Study quality judgement is reported in Supplementary data, Table S2.

SA definition

Forty-four studies assessed SA by sleep monitoring devices applying an AHI or RDI or Disordered Breathing Events cut-off value ≥5 events/h [4, 17, 35–38, 41, 42, 44, 47, 50, 52, 53, 67–69, 71, 74, 78–80, 82–85, 88, 91, 93, 98, 101–103, 105, 106, 112, 113, 117, 119, 122, 125–127, 130, 131]. Eight [9, 39, 70, 77, 94, 110, 114, 116] and 20 studies [13, 32, 40, 43, 48, 54, 55, 57, 60, 73, 89, 97, 99, 100, 108, 111, 120, 121, 123, 124] confirmed SA with an AHI ≥10 and ≥15 events/h, respectively. Five studies assessed SA using an AHI/RDI cut-off of ≥20 events/h [49, 51, 87, 92, 107].

The Berlin questionnaire placed the individual at high risk of SA if a positive score in at least two out of the three symptom domains (categories) was reported and a total score of three or more out of eight in STOP-Bang questionnaire. Thirty studies reported SA prevalence using questionnaires [1, 31, 33, 34, 38, 45, 52, 56, 58, 59, 61–66, 75, 76, 81, 86, 90, 93, 95, 96, 103, 104, 115, 128, 132, 133].

SA prevalence in CKD population

Amongst all studies reporting on non-dialysed CKD patients ($n = 31$), the prevalence of SA ranged from 6.4% [104], based on a self-reported home interview, to 96% [38] assessed by PSG. A pooled prevalence of 57% (95% CI 42%–71%) was observed from 32 single CKD study data (4607 patients) [4, 13, 17, 32, 34–51, 95, 96, 98, 101, 104, 105, 117, 119, 131], with high heterogeneity among studies ($I^2 = 99.4\%$). Analyses according to different diagnostic methods were conducted. The prevalence of SA using monitoring devices, including classical PSG and type III portable sleep monitors (26 studies, 2630 patients) was 62% (95% CI 52%–72%) ($I^2 = 97.5\%$) (Fig. 2).

In a meta-regression model including patients' selection, CKD stages, age, male gender, body mass index and AHI cut-off, the residual heterogeneity (i.e. the proportion of residual between-studies variation due to heterogeneity) was 90%, with 74% of the between-studies variance explained by the covariates mentioned above. The remaining between-studies variance appears small at 0.01225.

In a meta-analysis including 18 studies, applying AHI/RDI cut-offs of ≥5 or ≥10 events/h, the pooled prevalence was 72% (95% CI 64%–81%) ($I^2 = 95.5\%$), while pooled data from 7 studies, using a cut-off of ≥15 events/h, the prevalence was 39% (95% CI 32%–47%) ($I^2 = 80.5\%$).

Additionally, the cumulative prevalence amongst different CKD strata was calculated. Studies were classified into early (stage 1–2) (9 study groups), intermediate (stage 3–4) (15 study groups) and advanced CKD stage (stage >4) (11 study groups). The pooled prevalence amongst CKD strata was 56% (95% CI 41%–71%, $I^2 = 93\%$), 60% (95% CI 46%–73%, $I^2 = 96.7\%$) and 57% (95% CI 39%–75%, $I^2 = 97.5\%$), respectively. All analyses had high level of heterogeneity.

In studies based on sleep questionnaires (six studies, 1977 patients) the pooled prevalence was 33% (95% CI 16%–49%, $I^2 = 98\%$) (Fig. 2).

SA prevalence in ESKD population

Eighty-five studies [1, 9, 13, 17, 31, 33, 38, 45, 46, 48, 49, 51–94, 97–100, 102, 103, 106–116, 118, 120–128, 130, 132, 133] had available data on SA prevalence. Single study prevalence of SA, assessed by PSG, ranged from 0.33% [130] to 100% [126], this last one reporting on a selected high-risk population. A pooled prevalence of 49% (95% CI 47%–52%) was observed, with high heterogeneity among 91 single-study data (135 138 patients) ($I^2 = 99.9\%$).

Pooled analyses according to different diagnostic methods were conducted. In 63 studies (48 572 patients) employing sleep monitoring devices, a pooled prevalence of 56% (95% CI 42–69%) was observed. High heterogeneity among studies was observed ($I^2 = 99.5\%$) (Fig. 3a).

In a meta-regression model including patients' selection, dialysis modality, age, male gender, body mass index and AHI cut-off, the residual heterogeneity (i.e. the proportion of residual between-studies variation due to heterogeneity) was 90.11%, with 23% of the between-studies variance explained by the covariates mentioned above. The remaining between-studies variance appears small at 0.03021.

In analyses stratified according to AHI/RDI cut-off values, the pooled prevalence was 57% (95% CI 38%–75%) ($I^2 = 99.64\%$) from 38 studies using an AHI/RDI cut-off of ≥5 or ≥10 events/h and 56% (95% CI 50%–62%) ($I^2 = 72.5\%$) from 22 studies using a cut-off of ≥15 events/h.

We made also separate estimates of SA prevalence in HD and PD patients.

In studies applying sleep monitoring devices, the pooled prevalence was 55% (95% CI 47%–63%) ($I^2 = 92.9\%$) in the HD population (42 studies), 57% (95% CI 41%–72%) in PD individuals (8 studies) ($I^2 = 90.8\%$) and 65% (95% CI 46%–83%) in studies performed on a mixed population, HD and PD (10 studies) ($I^2 = 97.6\%$).

In studies based on sleep questionnaires (28 studies) (86 566 patients) the pooled prevalence was 39% (95% CI 30%–49%) ($I^2 = 99.2\%$) (Fig. 3b).

Nocturnal hypoxemia

Seven studies reported information on nocturnal hypoxemia [13, 18, 19, 37, 52, 134, 135]. The arterial oxygen saturation (SaO_2) during sleep of each subject was monitored using a pulse oximeter in five [18, 19, 52, 134, 135] out of seven studies. Desaturation was defined as a >4% drop in the SaO_2 level from baseline (4% ODI). Two [37, 134] and four studies [18, 19, 52, 135] reported, respectively, on CKD and ESKD patients, while Nicholl *et al.* [13] reported on both populations.

Hussein *et al.* [37], employing PSG to assess SA, found an overall nocturnal hypoxemia (NH) prevalence of 17.8% among

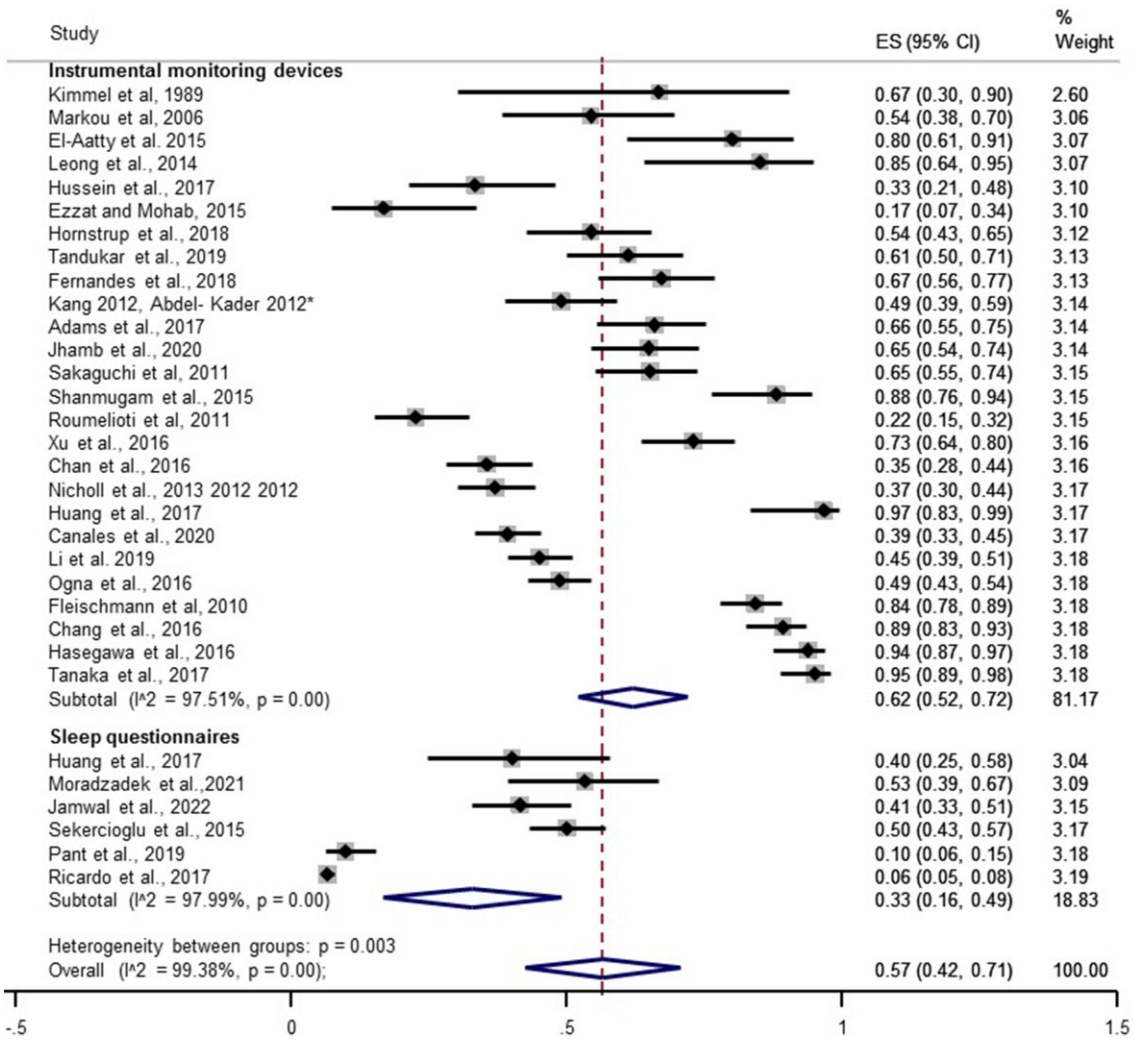

Figure 2: Pooled prevalence of SA in CKD according to different diagnostic criteria.

45 stages 2–5 CKD patients. NH prevalence increased as kidney function declined: stage 3–4 and stage 5 CKD groups had a higher occurrence (27.3% and 16.7%, respectively) than stage 2 (10%).

Similarly, in a study by Nicholl *et al.*, 179 stages 1–5 CKD and 75 HD patients completed an overnight cardiopulmonary monitoring, reporting an increase in NH prevalence as estimated glomerular filtration rate (eGFR) decreased [16% (eGFR ≥60) vs 47% (eGFR <60) vs 48% (ESKD), $P < .001$] [13].

In a retrospective cohort study by Sakaguchi *et al.* [134], 161 stages 3–4 CKD individuals had an overall NH prevalence of 50%. Nocturnal oxygen desaturation revealed an increased severity of NH as kidney function declined. eGFR (mL/min/1.73 m^2) declined 3- to 4-fold faster in the moderate–severe NH group (–8.59 ± 12.37 per year) than mild NH (–3.02 ± 6.86) and no-NH (–2.14 ± 4.86) groups, $P = .003$, analysis of variance).

In a prospective study by Zoccali *et al.* [18], 50 uraemic patients on dialysis, undergoing pulse oximetry monitoring, were followed up to 32 months. Nocturnal SaO_2 was significantly lower ($P = .006$) in patients who had fatal and non-fatal CV events during follow-up (94.7 ± 2.9%) than in event-free individuals (97.1 ± 1.3%). Furthermore, Zoccali *et al.* found an NH prevalence of 47% in a cohort of 38 HD patients undergoing continuous monitoring of SaO_2 during nighttime, revealing NH as the stronger independent predictor of relative wall thickness, mean wall thickness and left ventricular mass index, suggesting NH as an independent predictor of left ventricular hypertrophy.

In a multivariate analysis performed by Chu *et al.*, presence of large neck circumference ($P = .02$) and haemoglobin ($P = .003$) were independently associated with nocturnal oximetry in a relatively large number of patients with ESKD [52].

DISCUSSION

This systematic review and meta-analysis highlights that SA is a common comorbidity in patients with CKD and ESKD as well, with a prevalence almost 2- to 3-fold higher than that in the general population. Since SA is a consistent risk factor for mortality and CV complications in pre-dialysis CKD patients [136] and ESKD patients [1], findings in this meta-analysis have public health implications.

We observed an overall prevalence of SA of 53%, higher than that reported by a recent meta-analysis by Hansrivijit *et al.* (47.5%) [137] almost exclusively based on ESKD patients (97.8% ESKD and 2.2% CKD). A limitation of this meta-analysis was that the diagnosis was based on a hybrid definition, including International Classification of Diseases, Ninth Revision

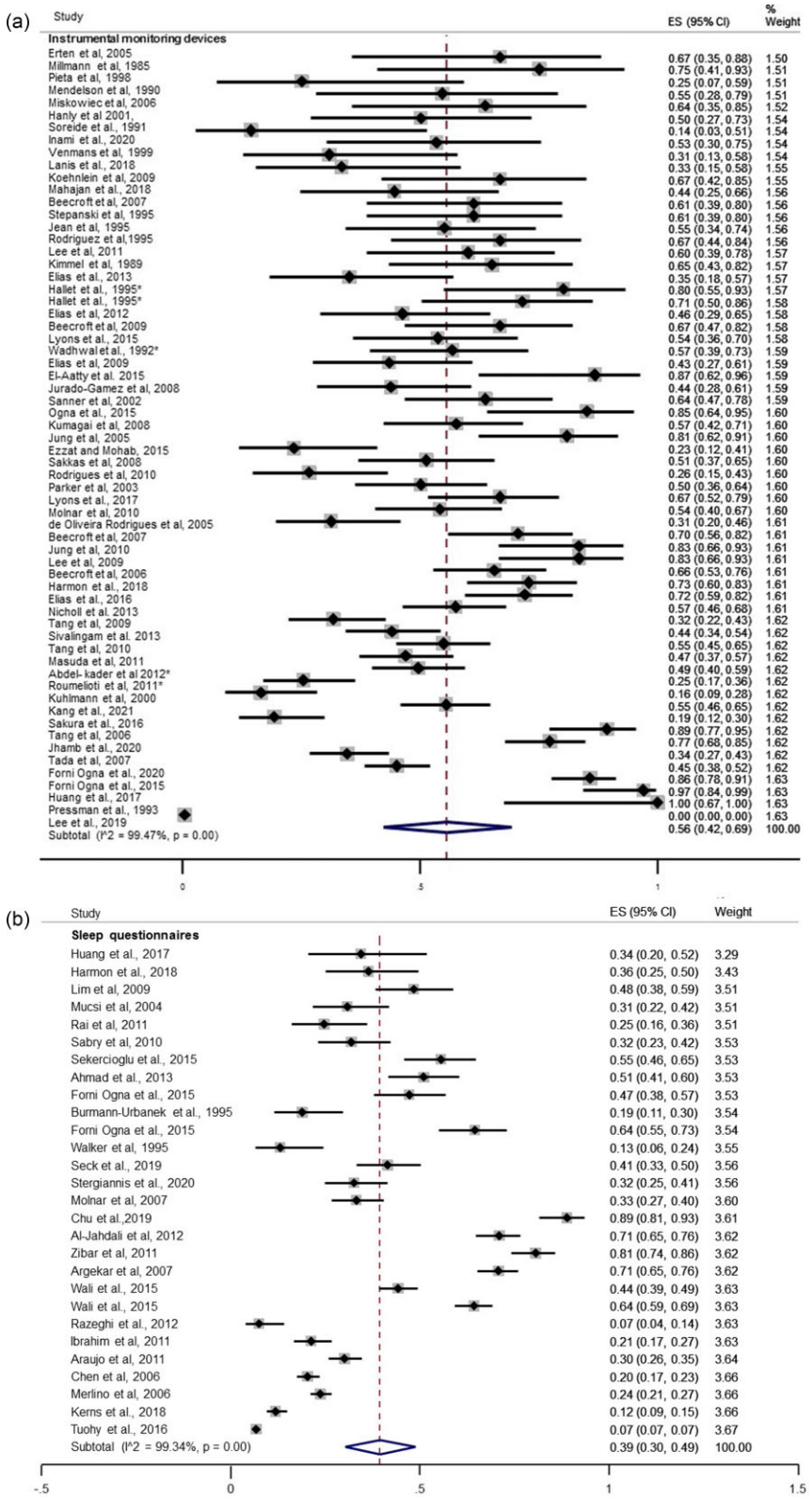

Figure 3: (a) Pooled prevalence of SA in ESKD according to instrumental monitoring devices. (b) Pooled prevalence of SA in ESKD according to sleep questionnaires.

coding for SA and sleep monitoring methods. A previous meta-analysis by Huang *et al*. [138], analysing only pre-dialysis CKD patients, reported an overall SA prevalence of 38% (26 studies) and a pooled prevalence of 56% in 21 studies (2430 individuals) based on sleep monitoring devices, figures that are lower than those in the present meta-analysis (57% and 62%, respectively) which included, respectively, 30 and 26 studies involving 4450 pre-dialysis CKD patients.

The prevalence of SA depends on the diagnostic method employed. Although screening for SA using sleep-related questionnaires is more cost-effective, they have only 50%–70% specificity (compared with PSG) in CKD/ESKD patients [13]. Validated questionnaires like STOP-Bang and Berlin significantly underestimate the prevalence; only 36% of patients included in this meta-analysis were at high risk of SA when screened by such methods. This finding confirms the low efficiency of sleep questionnaires in ascertaining SA in the kidney disease population, compared with respiration monitoring equipment. As mentioned, pooling data from studies employing instrumental sleep monitoring devices, including PSG, the prevalence of SA rises to 59%. Since SA is clinically relevant in CKD, PSG or other reliable instrumental sleep monitoring is recommendable.

Several mechanisms have been proposed to explain the association between SA in patients with kidney disease, such as accumulation of uraemic toxins and fluid throughout the body with consequent migration to the upper airway during sleep, leading to obstruction [139], all exacerbated by advanced kidney failure. As CKD worsens, the AHI can increase. However, our subgroup analysis did not reveal a higher prevalence of SA in patients undergoing regular dialysis than in non-dialysis CKD patients, and revealed no significant difference in the SA prevalence of HD and PD patients.

We observed an overall prevalence of SA of 57% in non-dialysis CKD population; this occurrence increased to 62% pooling studies employing sleep monitoring devices. In meta-regression analysis, the prevalence of SA in CKD and dialysis patients was clearly linked to classic risk factors, such as age, gender and obesity, but not to the severity of CKD. Stratifying according to CKD stages, we found a similar occurrence of SA in all strata (56% in early, 60% in intermediate and 57% in advanced CKD stages), with a slight increase from early to intermediate stages.

Our meta-analysis has several limitations. First, the sample size of included studies varied greatly from 6 [51] to 81 538 [128], which could be partially responsible for considerable heterogeneity observed. Second, studies were mainly cross-sectional; their quality would greatly influence the results. Third, the diagnostic methods and AHI cut-off values for ascertaining SA were not uniform across studies. Patients with mild SA ($5 \leq \text{AHI} < 10$) would be misdiagnosed if the AHI cut-off score applied by studies was $\geq$15 events/h. This high heterogeneity limits the value of the cumulative estimates of SA in the CKD population. A high heterogeneity was detected also in previous meta-analyses by Huang *et al*. [138] and Hansrivijit *et al*. [137], focusing on the prevalence of SA in pre-dialysis CKD and ESKD patients, respectively. In general, heterogeneity is more pronounced in small than in large studies but also in these studies a degree of heterogeneity persists [140]. However, we believe that our study is still meaningful in pointing out the necessity of SA screening in CKD/ESKD patients.

Our study reveals that SA is commonly seen in all strata of CKD, including ESKD patients; more than 50% of individuals are positive for SA. These findings suggest that screening for SA and monitoring sleep status in high-risk patients is a worthy undertaking in these populations. Sleep-related questionnaires underestimated the presence of SA and therefore they should not be used to diagnose SA in the same populations.

SUPPLEMENTARY DATA

Supplementary data are available at *ckj* online.

CONFLICT OF INTEREST STATEMENT

C.Z. and F.M. are members of the *CKJ* editorial board. The results presented in this article have not been published previously in whole or part. Authors declare no conflicts of interest related to the present work.

AUTHORS' CONTRIBUTIONS

All authors participated in the critical revision of the article for important intellectual content.

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DATA AVAILABILITY STATEMENT

The data underlying this article will be shared on reasonable request to the corresponding author.

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