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Association between comorbid sleep apnoea–hypopnoea syndrome and prognosis of intensive care patients: a retrospective cohort study

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ABSTRACT

Objective In this study, we investigated the association between comorbid sleep apnoea–hypopnoea syndrome (SAHS) and the prognosis of patients in an intensive care unit (ICU) to determine whether this relationship varies between different disease subgroups.

Methods We conducted a retrospective cohort study using publicly available information from the critical care database Medical Information Mart for Intensive Care III. Adults (≥18 years of age) who attended the ICU for the first time were enrolled. Demographic information and clinical data were obtained from each patient. The primary outcome was 30-day mortality after ICU admission, and the secondary outcomes were in-hospital and ICU mortality. Multivariate logistic regression and Cox regression analyses were used to examine the associations between SAHS comorbidities and the research outcomes. Propensity score matching was used to adjust for potential confounding variables.

Results Of the 32 989 patients enrolled, 1918 (5.81%) were diagnosed with SAHS as a comorbid condition. Patients with SAHS had a significantly lower 30-day mortality rate compared with those without SAHS (5.27% vs 13.65%, respectively; p<0.001). The frequency of chronic obstructive pulmonary disease, cerebral disease, cardiovascular disease, hypertension, diabetes mellitus and renal failure was significantly different between the two groups. Patients with SAHS demonstrated significantly longer survival compared with patients without SAHS. Multivariate Cox proportional hazards regression identified a significant relationship between SAHS and mortality within 30 days (adjusted HR=0.610, 95% CI 0.499 to 0.747, p<0.0001).

Conclusion SAHS as a comorbid condition decreases the risk of 30-day mortality, in-hospital mortality and ICU mortality among ICU patients.

Strengths and limitations of this study

- ► Utilisation of a large sample size from a public data set was helpful in establishing a robust model and hierarchical analysis.
- ► Multivariate Cox regression identified a significant association between sleep apnoea–hypopnoea syndrome (SAHS) comorbidity and 30-day mortality.
- ► We performed a subgroup analysis based on different diagnoses at admission to minimise heterogeneity from the mixed intensive care unit.
- ► Because data on disease severity and continuous positive airway pressure (CPAP) treatment were unavailable, we did not analyse the effects of SAHS severity and CPAP treatment on mortality, both of which might be confounders.

INTRODUCTION

Sleep apnoea–hypopnoea syndrome (SAHS) is a respiratory disease in which apnoea recurs more than 30 times or the apnoea–hypopnoea index increases five times per hour during 7 hours of sleep each night.[1] SAHS is one form of sleep-related breathing disorder, which encompasses obstructive sleep apnoea (OSA),[2 3] mixed sleep apnoea,[4] central sleep apnoea (CSA)[5] and sleep-related hypoventilation.[1] SAHS is a common comorbidity in patients with various diseases and is a risk factor for congestive heart failure,[6 7] myocardial infarction,[8 9] hypertension,[10–12] diabetes mellitus,[13–15] chronic obstructive pulmonary disease (COPD)[16–19] and cerebral disease,[20] which are also reasons for intensive care unit (ICU) admission.

OSA is associated with an increased risk of metabolic and cardiovascular comorbidities, as well as an increased risk of overall mortality.[21–24] However, subjects of most previous studies were not patients in the ICU; thus, current research on the effects of comorbid SAHS on the prognosis and outcomes of critically ill patients is limited. Bolona *et al*[25] showed that OSA is independently associated with lower in-hospital mortality (OR 0.408; 95% CI 0.298 to 0.557), but only disease severity was adjusted in this study. Interestingly, a recent study from Bailly *et al*[26] indicated that OSA status did not affect in-hospital or ICU mortality. As SAHS is often associated with other comorbid conditions, it is important to determine if comorbid SAHS affects the prognosis and outcomes of ICU

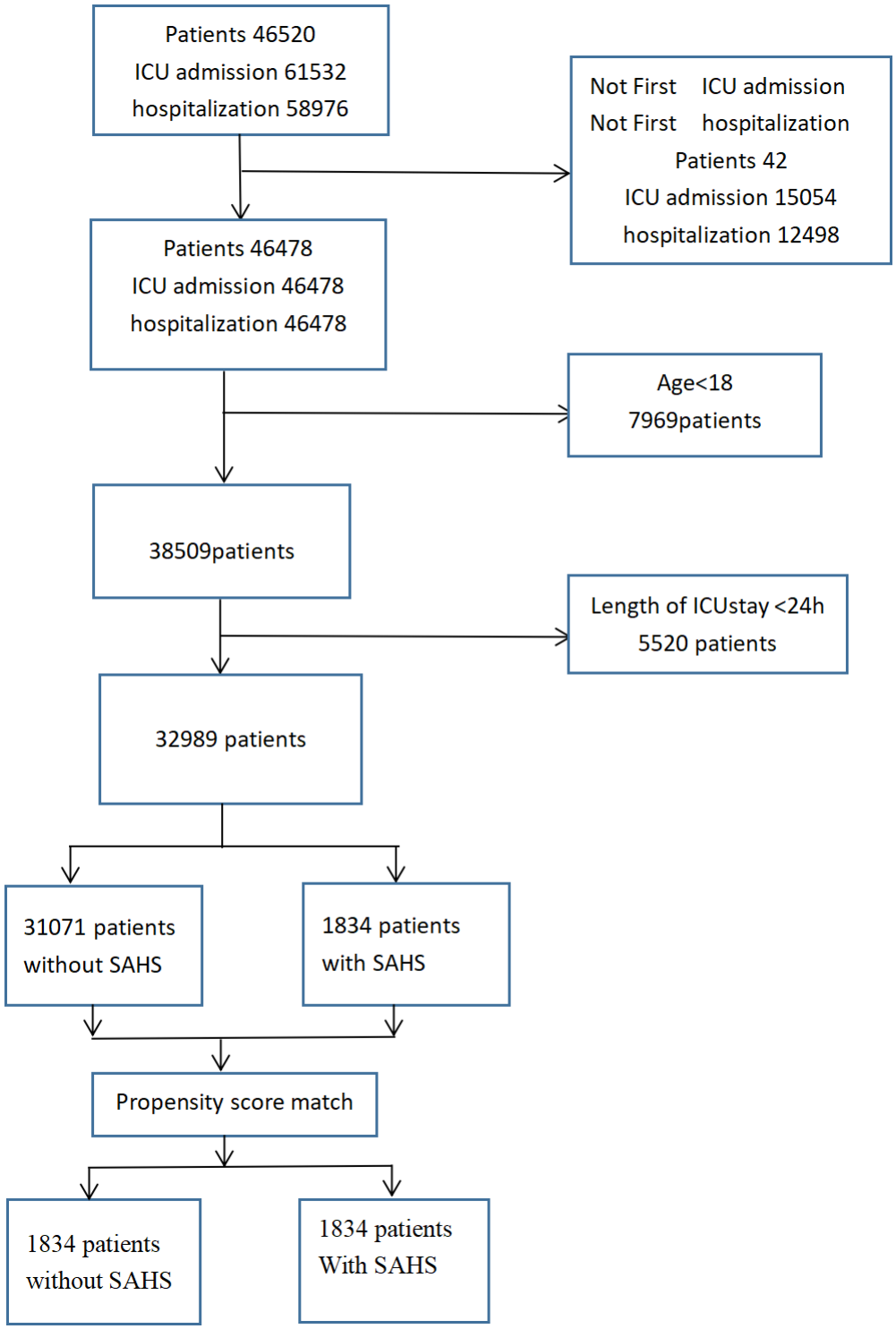

Figure 1 Flow chart depicting patients in this study. ICU, intensive care unit; SAHS, sleep apnoea–hypopnoea syndrome.

patients. In this study, we evaluated the association between comorbid SAHS and prognosis in critically ill patients.

MATERIALS AND METHODS

Patient and public involvement

Neither patients nor the public were involved in the design, conduct, reporting or dissemination of this research.

Database

In this retrospective study, we obtained data from the Medical Information Mart for Intensive Care III (MIMIC-III) database, which contains publicly available deidentified health data from more than 40 000 patients admitted to the ICU of Beth Israel Deaconess Medical Center (Boston, Massachusetts, USA) from 2001 to 2012. All data analysed in this study were obtained by one of

Table 1 Comparison of baseline characteristics and prognosis between patients in the ICU with and without SAHS

	Patients in total (N=32 989)	Without SAHS (n=31 071)	With SAHS (n=1918)	P value
Age (years)	64.18±17.28	64.32±17.49	61.94±13.32	<0.001
Male	18 748 (56.83)	17 479 (56.26)	1269 (66.16)	<0.001
Female	14 241 (43.17)	13 592 (43.74)	649 (33.84)	
BMI	23.79 (19.96–26.49)	23.35 (19.75–26.04)	30.91 (24.74–35.10)	<0.001
Type of admission				
Elective	5413 (16.40)	4971 (16.00)	442 (23.04)	<0.001
Emergency	26 595 (80.62)	25 162 (80.98)	1433 (74.71)	
Urgent	981 (2.98)	938 (3.02)	43 (2.24)	
Type of ICU on admission				
CCU	4809 (14.58)	4532 (14.59)	277 (14.44)	<0.001
CSRU	7069 (21.43)	6637 (21.36)	432 (22.52)	
MICU	11 376 (34.48)	10 636 (34.23)	740 (38.58)	
SICU	5403 (16.38)	5108 (16.44)	295 (15.38)	
TSICU	4332 (13.13)	4158 (13.38)	174 (9.07)	
Scoring systems				
SOFA	4.12 (2–6)	4.12 (2–6)	4.12 (2–6)	0.25
APSIII	42.53 (29–52)	42.62 (29–52)	41.05 (29–50)	0.03
LODS	4.03 (2–6)	4.02 (2–6)	4.16 (2–6)	0.0028
GCS	13.70 (14–15)	13.70 (14–15)	13.80 (14–15)	0.0273
SIRS	2.78 (2–4)	2.79 (2–4)	2.68 (2–3)	<0.001
Sepsis	6071 (18.40)	5669 (18.25)	402 (20.96)	0.003
Mechanical ventilation on first day	18 226 (55.24)	16 977 (54.64)	1249 (65.12)	<0.001
Renal replacement therapy on first day	1346 (4.08)	1239 (3.99)	107 (5.58)	0.001
Outcome				
30-day mortality	4343 (13.16)	4242 (13.65)	101 (5.27)	<0.001
ICU mortality	2556 (7.74)	2500 (8.05)	56 (2.92)	<0.001
Hospital mortality	3419 (10.36)	3341 (10.75)	78 (4.07)	<0.001
Length of ICU stay (days)	4.60 (1.54–4.75)	4.58 (1.58–4.75)	4.78 (1.5–4.79)	0.7345
Length of hospital stay (days)	10.64 (4.62–12.75)	10.65 (4.62–12.75)	10.48 (4.87–12.83)	0.1404
Comorbidities				
Cardiac disease	15 843 (48.02)	14 670 (47.21)	1173 (61.16)	<0.001
COPD	3853 (11.67)	3448 (11.10)	405 (21.12)	<0.001
Diabetes	9156 (27.75)	8251 (26.56)	905 (47.18)	<0.001
Cerebral disease	5080 (15.39)	4813 (15.49)	267 (13.92)	0.065
Hypertension	14 520 (44.01)	13 527 (43.54)	993 (51.77)	<0.001
Renal failure	5534 (16.68)	5161 (16.61)	373 (19.45)	0.001
AIDS	215 (0.65)	201 (0.65)	14 (0.73)	0.661
Anaemia	4489 (13.60)	4151 (13.36)	338 (17.62)	<0.001
CPHD	1673 (5.03)	1407 (4.53)	266 (13.87)	<0.001
Hypothyroid	3494 (10.59)	3201 (10.30)	293 (15.28)	<0.001
Liver disease	1322 (4.0)	1210 (3.89)	112 (5.84)	<0.001
Lymphoma	345 (1.04)	328 (1.06)	17 (0.89)	0.479
Peptic ulcer	269 (0.81)	250 (0.80)	19 (0.99)	0.379
Rheumatoid arthritis	465 (1.40)	428 (1.38)	37 (1.93)	0.047

Data are expressed as mean±SD, median (25th–75th percentile) or n (%).
Kruskal-Wallis or χ^2 (or Fisher's exact) test was used for comparison between groups.
*Statistical significance at p<0.05.
APSIII, Acute Physiology Score III; BMI, body mass index; CCU, coronary care unit; COPD, chronic obstructive pulmonary disease; CPHD, chronic pulmonary heart disease; CSRU, cardiac surgery recovery unit; GCS, Glasgow Coma Scale; ICU, intensive care unit; LODS, Logistic Organ Dysfunction Score; MICU, medical intensive care unit; SAHS, sleep apnoea–hypopnoea syndrome; SICU, surgical intensive care unit; SIRS, systemic inflammatory response syndrome; SOFA, Sequential Organ Failure Assessment; TSICU, trauma/surgical intensive care unit.

the authors, who successfully completed the National Institutes of Health online training course (certification number: 39067458). The PostgreSQL Tools V.4.24 open-source database was used for data extraction.

Study population and data extraction

We selected patients aged >18 years of age when first admitted to hospital and who had an ICU length of stay of >24 hours. For patients with multiple hospital admissions, we analysed data from the first ICU admission only during the first hospitalisation. Detailed procedures for patient selection are presented in figure 1.

SQL was used to extract clinical information, including age, sex, weight, height, type of ICU, reason for ICU admission, Logistic Organ Dysfunction Score (LODS), Sequential Organ Failure Assessment (SOFA) data, Glasgow Coma Scale (GCS) score, Acute Physiology Score III (APSIII) and systemic inflammatory response syndrome (SIRS). Information was also collected on other types of therapy, including mechanical ventilation on the day after admission to the ICU and renal replacement therapy. SAHS was classified using the following International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) codes: 32720, 32721, 32723, 32724, 3275, 32726, 232727 and 78057. We also extracted comorbidity information such as cardiac disease, COPD, diabetes mellitus, cerebral disease, hypertension and renal failure, which were determined by ICD-9-CM codes. In addition, to avoid the impact of missing values, we did not include laboratory measurements.

Study outcomes

The primary outcome was 30-day mortality after admission to the ICU, while the secondary outcomes were ICU mortality and in-hospital mortality. Other outcomes, such as length of ICU hospitalisation and length of overall hospital stay, were extracted for descriptive purposes only.

Statistical analysis and model construction

Data are presented as mean±SD, median (25%–75% quantile) or number (percentage). χ^2 test, Fisher's exact test and Kruskal-Wallis test were used for group comparison. Log-rank test and Kaplan-Meier curve were used to examine survival differences between patients with SAHS and patients without comorbid SAHS. Relationships between comorbid SAHS and the primary outcome and secondary outcomes were determined with Cox proportional hazards regression and logistic regression models, respectively. Three separate analyses, which included the propensity score model, were used to adjust for potential confounding variables. Model 1 included age in years, body mass index (BMI), sex, admission type and ICU type. Model 2 included age in years, BMI, sex, admission type, ICU type, SOFA, APSIII, LODS, GCS, SIRS, sepsis, and renal replacement therapy and/or mechanical ventilation on the day following admission, as well as cardiovascular disease, COPD, cerebral disease, diabetes mellitus, hypertension, AIDS, renal failure, anaemia, chronic pulmonary heart disease, hypothyroidism, liver disease, lymphoma, peptic ulcer and rheumatoid arthritis. The propensity score model included matched sets of intervention patients and control patients who shared similar propensity scores.[27] Propensity score matching is commonly used for one-to-one or matched pairs, in which pairs of intervention and control groups with similar propensity scores are formed.[28] We calculated propensity scores using the variables presented in table 1, the nearest-neighbour matching method and one-to-one matching between groups. The details are presented in figure 2. All statistical tests were two-sided and were conducted using Stata software (V.15.0). A p value <0.05 was considered statistically significant.

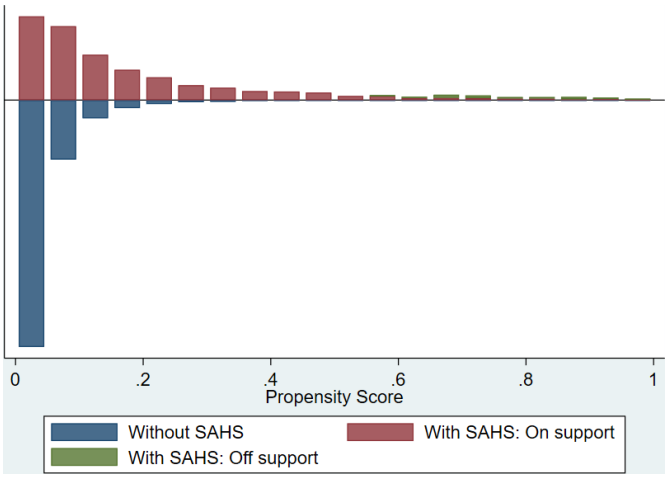

Figure 2 Propensity score matching map. SAHS, sleep apnoea–hypopnoea syndrome.

RESULTS

Baseline characteristics

Of the 32 989 patients enrolled, 1918 (5.81%) had SAHS as a comorbidity. The average age of patients was 64.18±17.28 years. Patients without SAHS comorbidities were significantly older compared with patients with SAHS (64.32±17.49 years vs 61.94±13.32 years, respectively; p<0.001). A greater percentage of patients with SAHS were male, and there was a difference from patients without SAHS (66.16% vs 56.26%; p<0.001). In the scoring system, patients with SAHS had lower scores compared with patients without SAHS, except for SOFA and GCS scores. A higher proportion of patients in the SAHS group were diagnosed with sepsis and required mechanical ventilation and renal replacement therapy on the first day of hospitalisation compared with patients without SAHS. Compared with patients without SAHS, a higher percentage of patients with SAHS had other comorbidities. The demographic characteristics of ICU patients with and without SAHS are presented in table 1.

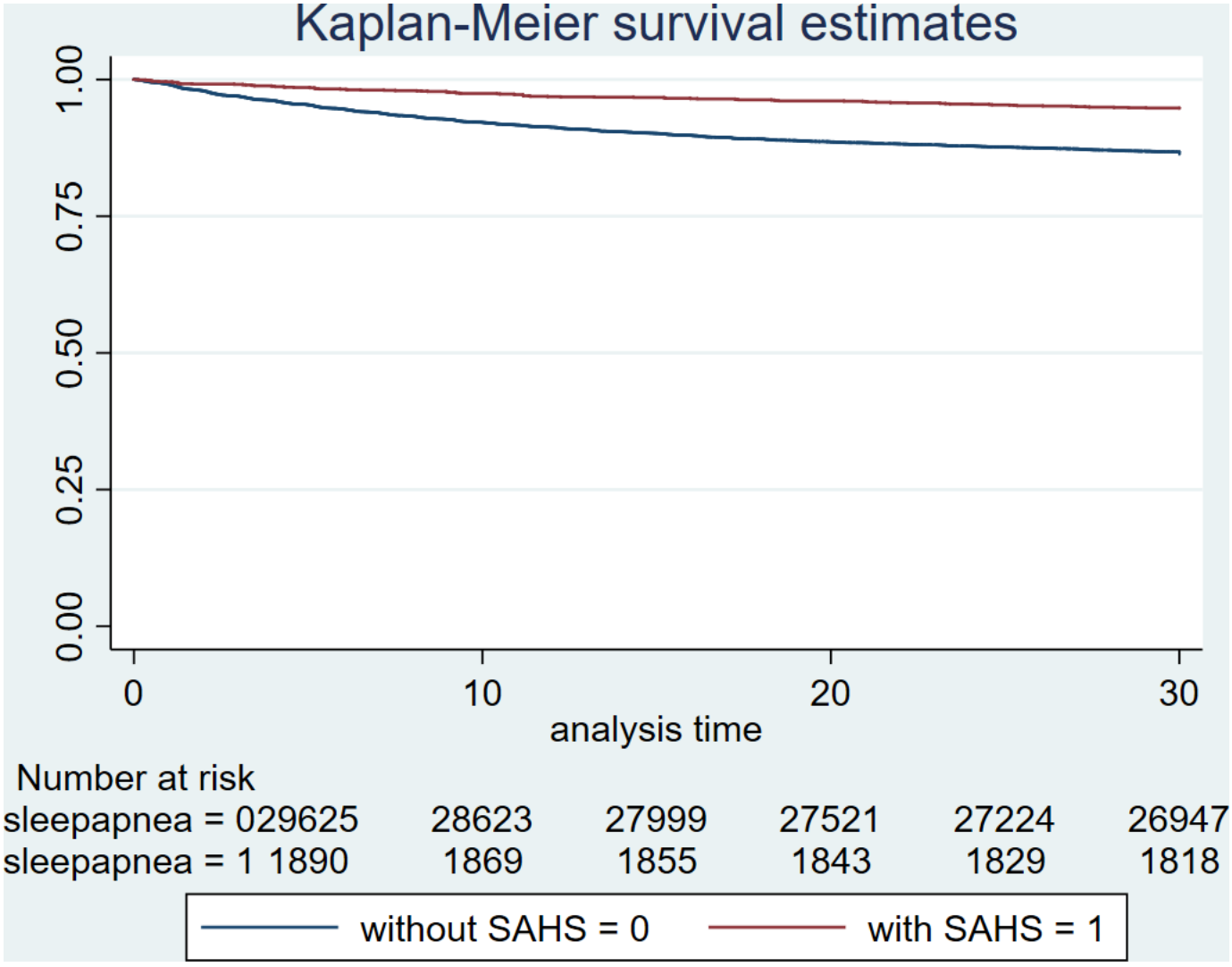

Figure 3 Kaplan-Meier survival curves of patients in this study. ICU, intensive care unit; SAHS, sleep apnoea–hypopnoea syndrome.

Clinical outcomes of the study and subgroups

A total of 4343 patients (13.16%) died within 30 days of admission to the ICU. A higher 30-day mortality rate was observed in patients without SAHS compared with those with SAHS (13.65% vs 5.27%, respectively; p<0.001). Similarly, ICU mortality rate (8.05% vs 2.92%, respectively; p<0.001) and in-hospital mortality rate (10.75% vs 4.07%, respectively; p<0.001) were higher in patients without SAHS compared with those with SAHS. The median length of ICU stay and hospital stay did not differ significantly between the two groups (table 1). Kaplan-Meier survival curves depict the survival of patients diagnosed with SAHS and patients without SAHS within 30 days of admission to the ICU (figure 3). The 30-day survival rate of patients with SAHS (p<0.0001 in a log-rank test) was significantly higher compared with patients without SAHS. Moreover, patients with SAHS in different disease subgroups, including cardiac disease, COPD, diabetes mellitus, hypertension and renal failure, were associated with lower mortality compared with patients without SAHS (figure 4 and table 2). In the BMI subgroups, most patients with comorbid SAHS had lower 30-day mortality, ICU mortality and in-hospital mortality rates compared with patients without SAHS (table 3).

Association between SAHS and clinical outcomes

Multivariate Cox regression and logistic regression analyses indicate that patients with comorbid SAHS had a lower risk of 30-day mortality, ICU mortality and in-hospital mortality, with crude HR of 0.36 (95% CI 0.30 to 0.44), 0.343 (95% CI 0.262 to 0.449) and 0.351 (95% CI 0.279 to 0.442), respectively. These relationships remained significant and coincident after adjustment in two models (table 4). To further explore the association between comorbid SAHS and clinical outcomes, we performed propensity score matching between the two patient groups using the variables presented in table 1 and one-to-one matching. Patients with comorbid SAHS still had a lower risk of 30-day mortality, ICU mortality and in-hospital mortality. The results of the propensity score model are presented in table 5.

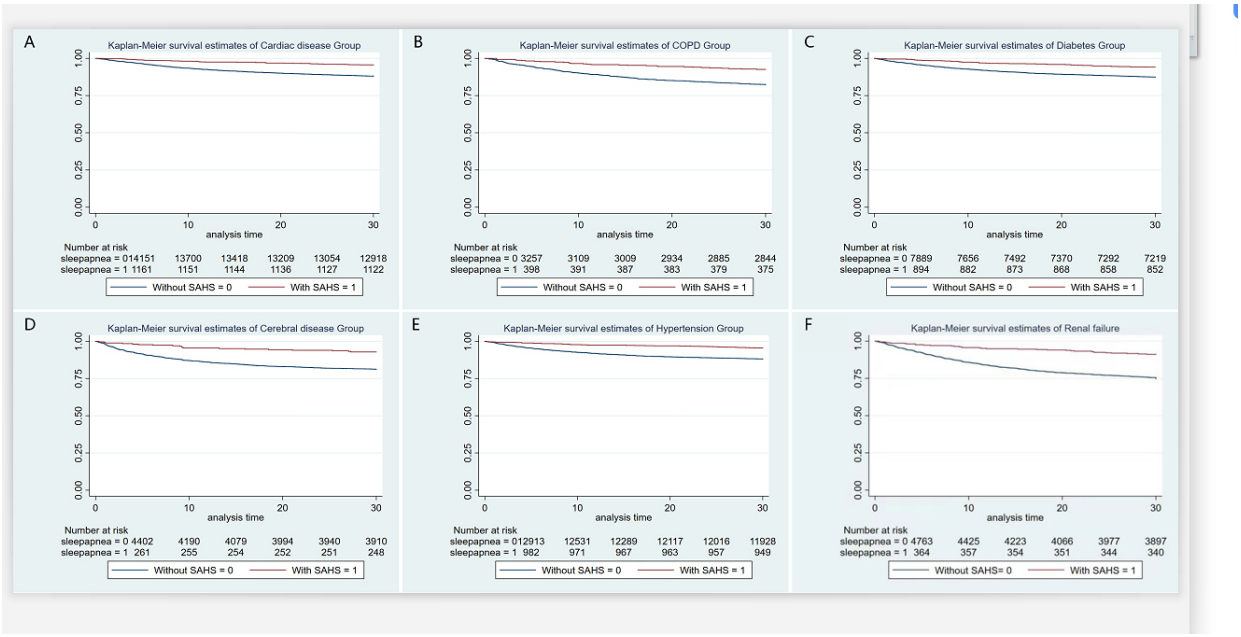

Figure 4 Kaplan-Meier survival curves for different disease subgroups: (A) cardiovascular disease, (B) COPD, (C) diabetes mellitus, (D) cerebral disease, (E) hypertension and (F) renal failure. COPD, chronic obstructive pulmonary disease; SAHS, sleep apnoea–hypopnoea syndrome.

DISCUSSION

We conducted a large retrospective cohort study using data on ICU patients from the MIMIC-III database. We found that patients with comorbid SAHS had a lower risk of 30-day mortality, ICU mortality and in-hospital mortality compared with patients without SAHS. Moreover, patients with SAHS in different disease subgroups, including cardiac disease, COPD, diabetes mellitus, cerebral disease, hypertension and renal failure, also demonstrated significantly better survival.

OSA is a common disorder in middle-aged individuals, with a prevalence of >20%.[29 30] According to polysomnographic assessment in ICU survivors, some studies have suggested that the prevalence of OSA is considerably high.[31–33] However, there are no reliable epidemiological studies on the prevalence of SAHS in ICU patients. Only a few similar studies reported a prevalence ranging from 5.6% to 7.8%,[25 26] which is similar to the prevalence of 5.8% observed in our study.

Table 2 Comparison of mortality between patients in different disease subgroups in the ICU with and without SAHS

Disease subgroups	Groups	30-day mortality	P value	ICU mortality	P value	In-hospital mortality	P value
Cardiac disease (n=15 843)	Without SAHS	1814 (12.37)	<0.001	1031 (7.03)	<0.001	1417 (9.66)	<0.001
	With SAHS	52 (4.43)		26 (2.22)		40 (3.41)	
COPD (n=3853)	Without SAHS	617 (17.89)	<0.001	331 (9.60)	<0.001	443 (12.85)	<0.001
	With SAHS	30 (7.41)		14 (3.46)		20 (4.94)	
Diabetes (n=9156)	Without SAHS	1057 (12.81)	<0.001	613 (7.43)	<0.001	820 (9.94)	<0.001
	With SAHS	53 (5.86)		27 (2.98)		42 (4.64)	
Cerebral disease (n=5080)	Without SAHS	914 (18.99)	<0.001	550 (11.43)	<0.001	727 (15.10)	<0.001
	With SAHS	19 (7.12)		7 (2.62)		14 (5.24)	
Hypertension (n=14 520)	Without SAHS	1638 (12.11)	<0.001	923 (6.82)	<0.001	1234 (9.12)	<0.001
	With SAHS	44 (4.43)		21 (2.11)		33 (3.32)	
Renal failure (n=5534)	Without SAHS	1309 (25.36)	<0.001	770 (14.92)	<0.001	1079 (20.91)	<0.001
	With SAHS	33 (8.85)		19 (5.09)		28 (7.51)	

Data are expressed as n (%).
χ^2 (or Fisher's exact) test was used for comparison between groups.
*Statistical significance at p<0.05.
COPD, chronic obstructive pulmonary disease; ICU, intensive care unit; SAHS, sleep apnoea–hypopnoea syndrome.

Table 3 Comparison of mortality between patients in different BMI subgroups in the ICU with and without SAHS

Disease subgroups	Groups	30-day mortality	P value	ICU mortality	P value	In-hospital mortality	P value
Group 1: BMI <18.5 (n=5246)	Without SAHS	1068 (20.56)	0.058	574 (11.05)	0.039	786 (15.13)	0.066
	With SAHS	5 (9.80)		1 (1.96)		3 (5.88)	
Group 2: 18.5≤BMI<23.9 (n=14 103)	Without SAHS	1787 (12.99)	<0.001	1037 (7.54)	0.001	1404 (10.21)	0.001
	With SAHS	22 (6.30)		10 (2.87)		16 (4.58)	
Group 3: 24≤BMI<28 (n=7554)	Without SAHS	825 (11.54)	<0.001	495 (6.92)	<0.001	664 (9.29)	<0.001
	With SAHS	19 (4.69)		9 (2.22)		13 (3.21)	
Group 4: 28≤BMI<32 (n=3434)	Without SAHS	347 (11.41)	<0.001	243 (7.99)	<0.001	293 (9.63)	<0.001
	With SAHS	12 (3.06)		8 (2.04)		10 (2.55)	
Group 5: 32≤BMI<40 (n=2092)	Without SAHS	165 (10.19)	0.036	111 (6.86)	0.057	147 (9.08)	0.013
	With SAHS	33 (6.98)		21 (4.44)		26 (5.50)	
Group 6: BMI ≥40 (n=560)	Without SAHS	50 (16.03)	<0.001	40 (12.82)	<0.001	47 (15.06)	<0.001
	With SAHS	10 (4.03)		7 (2.82)		10 (4.03)	

Data are expressed as n (%).
χ^2 (or Fisher's exact) test was used for comparison between groups.
*Statistical significance at p<0.05.
BMI, body mass index; ICU, intensive care unit; SAHS, sleep apnoea–hypopnoea syndrome.

Compared with a study by Bolona *et al*,[25] which used a large patient population to examine the effects of comorbid SAHS on the prognosis and outcomes of patients admitted to the ICU, there are several differences in our research. We enrolled a larger sample size, which is more advantageous for stratified analyses. We also used different methods of analysis, such as propensity score matching, to adjust for potential confounders. However, the results of both these studies are generally consistent with regard to the association between comorbid SAHS and clinical outcomes. Interestingly, a recent study from Bailly *et al*,[26] which used an appropriate exposed/unexposed matched design, reported the opposite conclusion. The sample size in that study was smaller, and the study did not consider the effects of sepsis, mechanical ventilation or renal replacement therapy, which may have led to poor results.

We also used propensity score matching in our study. Patients with comorbid SAHS still had a lower risk of 30-day mortality, ICU mortality and in-hospital mortality. These results indicate that SAHS may be independently associated with lower mortality rates.

Protective effects attributed to obesity in Bolona *et al*'s[25] research may explain these findings. The relationship between ICU prognosis and obesity has been extensively studied. Most studies found that obesity is independently associated with improved ICU outcomes,[34–36] but there are some controversies. Specifically, some studies have shown no advantage or increased ICU mortality in patients with obesity.[37 38] It is difficult to explain the results with obesity in our study because, after adjusting for BMI in different models and propensity score matching, patients without SAHS still had a lower risk of 30-day mortality, ICU mortality and in-hospital mortality compared with those with SAHS. The reason SAHS provides protection in ICU patients is unclear; however, we hypothesise that it is related to the complex pathophysiology of the disease. CSA and OSA may disrupt breathing through different physiological mechanisms and may impose qualitatively similar inflammatory burden on the cardiovascular system. The primary differences between OSA and CSA are the greater negative intrathoracic pressure and the lower average awake partial pressure of carbon dioxide (PaCO2) caused by obstructive events and greater sympathoexcitation.[39 40] With continued apnoea, OSA may transform into a pattern that resembles CSA, and if treatment improves ventricular filling pressure CSA may revert to OSA.[41] Archetypical cycles of increasing and decreasing tidal volume that are characteristic of CSA, termed Cheyne-Stokes respiration,[42–44] have compensatory and beneficial effects in patients with heart failure.[45] It is possible that the same benefits exist in critically ill patients. In addition, a landmark feature of sleep apnoea is intermittent hypoxia, which may make critically ill patients more tolerant to hypoxia and circulatory damage. The likelihood that these conjectures will be proven by research is remote. However, some researchers believe that sleep-related breathing disorders may confer underappreciated benefits that may be offset by current methods of targeted therapy.[46]

Interestingly, during the COVID-19 pandemic, increasing evidence suggests that OSA is an independent risk factor for severe COVID-19.[47–49] A multicentre, prospective study from Peker *et al*[47] showed that adults with high-risk OSA in a COVID-19 cohort had poorer clinical outcomes compared with patients with low-risk OSA, but their study focused on clinical improvement

Table 4 Association between comorbid COPD and prognosis

Outcomes	Group	HR (or OR)	95% CI	P value
30-day mortality				
Crude	Without SAHS	Ref	0.30 to 0.44	<0.001
	With SAHS	0.36		
Model 1	Without SAHS	Ref	0.405 to 0.606	<0.001
	With SAHS	0.495		
Model 2	Without SAHS	Ref	0.499 to 0.747	<0.001
	With SAHS	0.610		
ICU mortality				
Crude	Without SAHS	Ref	0.262 to 0.449	0.0047
	With SAHS	0.343		
Model 1	Without SAHS	Ref	0.301 to 0.517	0.0390
	With SAHS	0.349		
Model 2	Without SAHS	Ref	0.361 to 0.646	<0.001
	With SAHS	0.483		
In-hospital mortality				
Crude	Without SAHS	Ref	0.279 to 0.442	
	With SAHS	0.351		
Model 1	Without SAHS	Ref	0.346 to 0.554	<0.001
	With SAHS	0.438		
Model 2	Without SAHS	Ref	0.417 to 0.691	<0.001
	With SAHS	0.537		

The association between comorbid SAHS and 30-day mortality was analysed using Cox regression analysis, and in-hospital mortality and ICU mortality were analysed using logistic regression models.
Model 1 was adjusted by age, sex, BMI, type of ICU and type of admission; model 2 was adjusted by age, sex, BMI, type of ICU, type of admission, SOFA, APSIII, SIRS, LODS, GCS, sepsis, mechanical ventilation on the first day, renal replacement therapy on the first day, cardiac disease, COPD, cerebral disease, hypertension, diabetes mellitus, renal failure, AIDS, anaemia, CPHD, hypothyroidism, liver disease, lymphoma, peptic ulcer and rheumatoid arthritis.
Statistical significance at p<0.05.
APSIII, Acute Physiology Score III; BMI, body mass index; COPD, chronic obstructive pulmonary disease; CPHD, chronic pulmonary heart disease; GCS, Glasgow Coma Scale; ICU, intensive care unit; LODS, Logistic Organ Dysfunction Score; Ref, reference; SAHS, sleep apnoea–hypopnoea syndrome; SIRS, systemic inflammatory response syndrome; SOFA, Sequential Organ Failure Assessment.

without death analysis and patients who died were excluded. A study by Strausz *et al*[48] demonstrated the same trend, and the authors emphasised that OSA was associated with a higher risk of hospitalisation. Cade *et al*'s[49] research revealed that patients with COVID-19 and sleep apnoea had an increased all-cause mortality rate (11.7%) compared with control patients with sleep apnoea only (6.0%) (p<0.001), with an OR of 1.79 (95% CI 1.31 to 2.45) before adjustment. However, after full adjustment, there was no significant difference in OR (1.16; 95% CI 0.8 to 1.68). In addition, they found no significant difference between patients undergoing continuous positive airway pressure (CPAP) treatment in the year prior and those without CPAP treatment. Unfortunately, our research lacked CPAP treatment records and also lacked data from patients with COVID-19. However, these studies suggest that OSA may play different roles in different populations; thus, further studies are needed.

There are some limitations to the present study that should be highlighted. First, a subgroup analysis based on different admission diagnoses was performed to alleviate heterogeneity from the mixed ICU. Second, due to unavailability of data on disease severity and CPAP treatment, we did not analyse the effects of SAHS severity and CPAP treatment on mortality, both of which might be confounders. These limitations led to a decline in the accuracy of our conclusions. Therefore, prospective studies, particularly studies assessing disease severity and CPAP treatment, are needed to further confirm our results.

CONCLUSION

We conclude that comorbid SAHS decreases 30-day mortality, ICU mortality and in-hospital mortality rates in ICU patients.

Table 5 Comparison of baseline characteristics and prognosis between patients in the ICU with and without SAHS: postmatching

	Patients in total (n=3668)	Without SAHS (n=1834)	With SAHS (n=1834)	P value
Age (years)	62.11±14.37	62.07±15.24	62.15±13.43	0.87
Male	2426 (66.13)	1212 (66.09)	1214 (66.19)	0.944
Female	1242 (33.87)	622 (33.91)	620 (33.81)	
BMI	30.06 (24.38–34.16)	30.06 (24.21–34.20)	30.06 (24.60–34.15)	0.2003
Type of admission				
Elective	850 (23.17)	421 (22.96)	429 (23.39)	0.068
Emergency	2754 (75.08)	1390 (75.79)	1364 (74.37)	
Urgent	64 (1.74)	23 (1.25)	41 (2.24)	
Type of ICU on admission				
CCU	530 (14.45)	266 (14.50)	264 (14.39)	0.003
CSRU	902 (24.59)	478 (26.06)	432 (22.52)	
MICU	1294 (35.28)	601 (32.78)	740 (38.58)	
SICU	553 (15.07)	270 (14.72)	295 (15.38)	
TSICU	389 (10.60)	219 (11.94)	174 (9.07)	
Scoring systems				
SOFA	4.12 (2–6)	4.13 (2–6)	4.11 (2–6)	0.8992
APSIII	40.92 (28–50)	40.95 (28–51)	40.89 (29–51)	0.6163
LODS	4.09 (2–6)	4.05 (2–6)	4.14 (2–6)	0.2185
GCS	13.8 (14–15)	13.79 (14–15)	13.81 (14–15)	0.5135
SIRS	2.67 (2–4)	2.66 (2–3)	2.68 (2–3)	0.7464
Sepsis	727 (19.8)	358 (19.52)	369 (20.12)	0.7464
Mechanical ventilation on first day	2362 (64.39)	1173 (63.96)	1189 (64.83)	0.581
Renal replacement therapy on first day	216 (5.88)	115 (6.27)	101 (5.51)	0.326
Outcome				
30-day mortality	272 (7.41)	171 (9.32)	101 (5.51)	<0.001
ICU mortality	150 (4.08)	94 (5.13)	56 (3.05)	0.002
Hospital mortality	216 (5.88)	138 (7.52)	78 (4.25)	<0.001
Length of ICU stay (days)	4.672 (1.48–4.91)	4.58 (1.5–5.08)	4.65 (1.45–4.58)	0.3615
Length of hospital stay (days)	10.80 (4.91–12.91)	11.27 (5.04–13.37)	10.34 (4.83–12.75)	0.0568
Comorbidities				
Cardiac disease	2194 (59.81)	1085 (59.16)	1109 (60.47)	0.419
COPD	745 (20.31)	379 (20.67)	366 (19.96)	0.594
Diabetes	1689 (46.04)	844 (46.02)	845 (46.07)	0.974
Cerebral disease	5080 (15.39)	269 (14.67)	264 (14.39)	0.815
Hypertension	1918 (52.29)	967 (52.73)	951 (51.85)	0.597
Renal failure	714 (19.46)	368 (20.07)	346 (18.87)	0.359
AIDS	26 (0.70)	14 (0.76)	12 (0.65)	0.694
Anaemia	641 (17.47)	316 (17.23)	325 (17.72)	0.696
CPHD	467 (12.73)	232 (12.65)	266 (12.81)	0.882
Hypothyroid	554 (15.10)	281 (15.32)	273 (14.89)	0.712
Liver disease	217 (5.91)	114 (6.22)	103 (5.62)	0.441
Lymphoma	31 (0.84)	14 (0.76)	17 (0.93)	0.588
Peptic ulcer	38 (1.03)	19 (1.4)	19 (1.4)	1.000
Rheumatoid arthritis	61 (1.66)	29 (1.58)	32 (1.74)	0.047

Data are expressed as mean±SD, median (25th–75th percentile) or n (%).
Kruskal-Wallis or χ^2 (or Fisher's exact) test was used for comparison between groups.
*Statistical significance at p<0.05.
APSIII, Acute Physiology Score III; BMI, body mass index; CCU, coronary care unit; COPD, chronic obstructive pulmonary disease; CPHD, chronic pulmonary heart disease; CSRU, cardiac surgery recovery unit; GCS, Glasgow Coma Scale; ICU, intensive care unit; LODS, Logistic Organ Dysfunction Score; MICU, medical intensive care unit; SAHS, sleep apnoea–hypopnoea syndrome; SICU, surgical intensive care unit; SIRS, systemic inflammatory response syndrome; SOFA, Sequential Organ Failure Assessment; TSICU, trauma/surgical intensive care unit.

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