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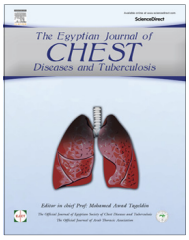

ORIGINAL ARTICLE

Study of sleep – Related breathing disorders in patients admitted to respiratory intensive care unit

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KEYWORDS

Sleep related breathing disorders (SRBD);
Respiratory intensive care unit (RICU);
Bilevel positive airway pressure (BiPAP);
Type II respiratory failure;
Obesity hypoventilation;
Polysomnography

Abstract *Objectives:* The purpose of this study was to assess the prevalence of SRBDs in acutely ill patients admitted to respiratory ICU.

Patients and methods: The study enrolled 72 patients admitted to respiratory ICU. All patients were subjected to full clinical examination, Epworth Sleepiness score, arterial blood gases analysis and clinical apnea score calculation. According to this latter, patients were divided into group I: without clinically suspected SRBDs and group II: with clinically suspected SRBDs. Patients in group II were subjected to polysomnography.

Results: Group I included 21 patients while group II included 51 patients. The BMI, neck circumference and waist/hip ratio were significantly higher in group II. Hypertension was the commonest comorbidity in group II. Type II respiratory failure was the commonest cause of ICU admission in both groups of patients. The mortality rate was higher within the 28 days that followed ICU admission in group I compared to group II. This latter group had a higher mortality rate later on. All patients subjected to polysomnography suffered from OSAHS, 82% of them showed associated sleep hypoventilation (SHV) with significantly elevated bicarbonate level. More than half the patients with SHV fulfilled the criteria of obesity hypoventilation syndrome. The AHI showed a significant direct correlation with neck circumference, systolic blood pressure, snoring index and T85%; and a significant inverse correlation with PaO_2, minimal saturation as well as average saturation.

Conclusion: In ICU patients, SRBDs are common coexistent findings and every physician should systematically search for them. Type II respiratory failure is the main cause of ICU admission in patients with SRBDs. Quality of sleep in ICU is very disturbed. Most ICU patients with SRBDs have concomitant SHVS mostly due to OHS. Important comorbidities coexist in patients with SRBDs; both influence each other and should be identified and managed properly for the wellbeing of the patient. BiPAP therapy is the cardinal mode of ventilation used in patients with respiratory failure and SRBDs.

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Introduction

Sleep related breathing disorders (SRBDs) are common in the general population. They can cause or exacerbate preexisting medical and psychiatric conditions and lead to significant morbidity such as depression, anxiety, impaired daytime functioning, poor occupational performance, motor vehicle accidents, cardiovascular and endocrine disorders [1].

Even though there is a large body of literature on sleep apnea in the outpatient setting, there is a paucity of data about the inpatient setting. An unmanaged SRBD in the hospital may delay recovery from illnesses and prolong hospital stay. Nevertheless, there are no studies on large inpatient populations that have looked into the potential effects of SRBD and acute illness on each other [2]. Critically ill patients, especially in an intensive care unit (ICU) have decreased quality and quantity of sleep attributable to environmental, psychological, and physiologic factors such as noise, pain, and awakenings from nursing care. The presence of SRBD would further disturb their sleep [3]. As most patients with SRBD have not been diagnosed; it is not uncommon for a patient to have this disease first recognized while in the intensive care unit. Sleep apnea by itself is not generally a cause of respiratory failure, and most patients except those with the obesity hypoventilation syndrome will have normal arterial blood gases, at least while awake. However, this disease does decrease respiratory drive, and may worsen respiratory failure from another cause [4].

The purpose of this study was to assess the prevalence of SRBDs in acutely ill patients admitted to respiratory ICU and to identify patients' characteristics that aid in predicting SRBDs in such population.

Patients and methods

The study enrolled 72 patients admitted to respiratory ICU in the Alexandria main university hospital during the period from July 2012 to December 2012. The local ethics committee approved the study and all participants signed an informed consent. Upon admission all patients were subjected to full history taking including symptoms related to SRBDs, Epworth Sleepiness score [5], complete clinical examination, routine laboratory work up, arterial blood gases analysis, plain chest X-ray, and electrocardiogram. Anthropometric measures were taken including: body mass index (BMI); neck [6], waist and hip [7] circumferences; and waist–hip ratio was calculated. When the patient could not move, weight and height measurements were obtained using specific equations [8]. Finally, the APACHE II score was calculated from 13 routine physiological measurements [9].

The clinical apnea score [10] was calculated for all patients. It is a score of 5 items including: loud, habitual snoring; interrupted breathing as reported to the patient by the spouse or family members; excessive daytime sleepiness as evidenced by Epworth Sleepiness score; obesity, defined as a BMI $> 25\ kg/m^2$; essential hypertension identified by the use of antihypertensive medications or blood pressure $\geqslant 140/90$ in two or more separate occasions. Patients were given a score of one for each positive item with a maximum score of five. Then the patients were divided into 2 groups according to their score:

Group I: patients without clinically suspected SRBDs based on a score < 2.
Group II: patients with clinically suspected SRBDs based on a score $\geqslant 2$.

All patients in group II were planned for full polysomnographic study. The latter was carried out using somnoscreen plus RC combi 39, which included the following channels: flow (cannula and/or thermistor), snore (cannula and/or microphone), thoracic movement, abdominal movement, oxygen saturation, plethysmogram, pulse rate, electrocardiogram, periodic leg movement, electrooculogram, electroencephalogram and CPAP/BiPAP pressure. According to the presence of sleep hypoventilation (SHV) as defined by the evidence of sagging of oxygen saturation curve in the polysomnogram, group II patients were further subdivided into two subgroups:

Group IIa: patients without SHV.
Group IIb: patients with SHV.

The polysomnographic analysis was done automatically but was imperatively coupled with manual scoring. The analysis included:

- *Sleep stage analysis*. It identified different sleep stages such as Non-REM (rapid eye movement) and REM sleep. It calculated total sleep time and efficiency. It recorded microarousal index and percent of respiratory related ones.
- *Respiratory event analysis*. It determined apnea hypopnea index (AHI) and respiratory disturbance index (RDI). It identified different types of apneas: obstructive, central or mixed and calculated their absolute values and indices. It estimated respiratory effort related arousal (RERA) index and absolute value. It measured the snoring index.
- *Oxygen saturation analysis*. It determined baseline, average and minimal oxygen saturation. It estimated oxygen desaturation index (ODI) as well as T90% (time of sleep with oxygen saturation below 90%) and T85%.
- *Heart rate analysis*. It measured maximal, minimal and average heart rate. It calculated acceleration, deceleration and arrhythmia indices as well as determined the type of arrhythmia.
- *Diagnosis*. It recognized the presence or absence of obstructive sleep apnea hypopnea syndrome (OSAHS) and its severity, central sleep apnea syndrome (CSAS) as well as sleep hypoventilation syndrome (SHVS).

All the patients were followed up for six months. The lines of treatment in the ICU including the mode of ventilation, the length of ICU stay and the mortality rate as well as recurrence of ICU admission were recorded.

Statistical analysis

Data were analyzed by SPSS 17.0 for Windows (SPSS Inc., Chicago, Illinois, USA). All hypotheses were constructed two-tailed and $p \leqslant 0.05$ was considered significant. Continuous variables were expressed as mean ± standard deviation, ordinal variables as median and minimum–maximum, and frequent variables as rates. Student *t*-test was used to compare data from two samples that were both normally distributed, and had the additional requirement that the standard deviations (SDs) from the two samples were approximately equal. A nonparametric alternative to the unpaired *t*-test was given

by the Wilcoxon rank sum test, which was also known as the Mann–Whitney test. This test was nonparametric because it was based on the ranks of the individual observations rather than on their actual values, which would be used in the *t* test. Chi-square test, a test of association, involved calculating the differences between the observed and expected frequencies. If the differences are large, then this suggests that there is an association between one variable and the other. Fisher's exact test was a statistical significance test used in the analysis of contingency tables. Although in practice it was employed when sample sizes were small, it was valid for all sample sizes. It was one of a class of exact tests, so called because the significance of the deviation from a null hypothesis (e.g.: *p*-value) could be calculated exactly, rather than relying on an approximation that became exact in the limit as the sample size grows to infinity, as with many statistical tests.

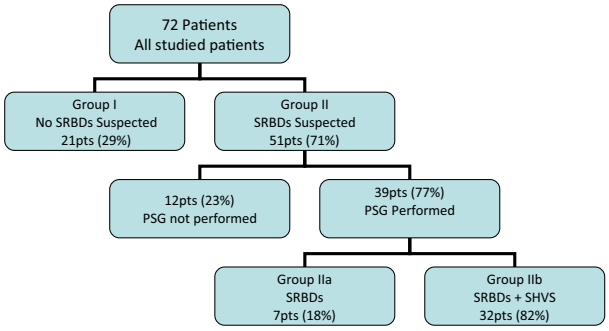

Figure 1 Diagram showing the distribution of all the studied patients.

Results

The patients' distribution is shown in Fig. 1. Group I included 21 patients while group II, those with high clinical probability of SRBDs included 51 patients (Fig. 1). Table 1 shows the characteristics of both groups of patients. The BMI, waist hip and neck circumference as well as the waist/hip ratio were significantly higher in group II than in group I (Fig. 2).

The comorbidities and clinical apnea scores of both groups of patients are shown in Table 2. Hypertension was by far the commonest comorbidity in group II followed by cardiac diseases; while in group I chest diseases were by far the commonest comorbidity. As expected, clinical apnea score was significantly higher in group II than group I.

The cause of ICU admission, lines of management and follow up are shown in Table 3. Type II respiratory failure was by a long way the commonest cause of ICU admission in both groups of patients. The mortality was higher within the 28 days that followed ICU admission in group I compared to group II. This latter had a higher mortality later on.

All patients in group II were scheduled for polysomnography. However, this latter could not be performed in 12 patients due to death in 6 patients, transfer to another care unit in 3 of them and refusal of the test in another 3 patients. The rest of the group, 39 patients was subjected to polysomnography which proved all of them to have SRBDs. The polysomnographic data of those patients revealed a total sleep time of 4.5 ± 1.4 h (range: 1.03–12.43); a sleep efficiency of 67.0 ± 26.0% (range: 16–96); light sleep stage time of 53.9 ± 26.7% of total sleep time (range: 29–93); deep sleep stage time of 23.5 ± 22.5% of total sleep time (range: 0–75) and REM sleep stage of 12.3 ± 12.3% of total sleep time (range: 0–51). The microarousal index was 3.6 ± 6.6 per hour (range: 0–33) and a percent of respiratory related microarousal of

Table 1 Patients' characteristics, laboratory investigations and arterial blood gases.

Characteristics	Group I (n = 21)	Group II (n = 51)	Significance
Anthropometric measures			
Age (years)	50.8 ± 14.8	59.1 ± 12.5	$t = 2.408$ $p = 0.019$*
Body mass index (kg/m^2)	24.9 ± 4.4	40.37 ± 9.76	$t = 8.666$ $p < 0.0001$*
Waist/hip ratio	0.9 ± 0.1	1.1 ± 0.1	$Z = 5.002$ $p < 0.0001$*
Neck circumference (cm)	36.0 ± 2.0	42.9 ± 3.9	$t = 9.767$ $p < 0.0001$*
Laboratory investigations			
Hematocrit (%)	39.8 ± 9.6	42.1 ± 11.2	$t = 0.836$ $p = 0.406$
WBC count	15.1 ± 8.6	11.4 ± 6.3	$t = 2.038$ $p = 0.045$*
Fasting blood sugar (mg/dl)	111.4 ± 38.3	130.2 ± 48.9	$t = 1.443$ $p = 0.155$
Post-prandial blood sugar (mg/dl)	149.5 ± 37.1	202.1 ± 90.2	$t = 3.2$ $p = 0.002$*
Total Protein (gm/dl)	5.5 ± 1.1	6.2 ± 1.1	$t = 2.402$ $p = 0.019$*
SGPT (IU/dl)	168.7 ± 467.8	46.3 ± 39.2	$Z = 2.649$ $p = 0.008$*
Cholesterol (mg/dl)	143.5 ± 54.5	194.7 ± 64.2	$t = 2.442$ $p = 0.018$*
Triglyceride (mg/dl)	81.9 ± 16.3	102.0 ± 32.7	$t = 2.905$ $p = 0.007$*
Serum uric acid (mg/dl)	6.4 ± 4.3	8.7 ± 2.7	$t = 2.363$ $p = 0.022$*
Arterial blood gases			
PaO_2 (mmHg)	64.6 ± 29.7	57.5 ± 17.0	$t = 1.278$ $p = 0.206$
$PaCO_2$ (mmHg)	59.1 ± 24.4	62.1 ± 20.7	$t = 0.536$ $p = 0.594$
pH	7.35 ± 0.13	7.31 ± 0.08	$t = 0.781$ $p = 0.175$
Bicarbonate (mmol/L)	29.0 ± 7.8	30.7 ± 8.4	$t = 0.79$ $p = 0.432$

t: *t*-test; *Z*: Mann Whitney test.
* Significant at $p \leqslant 0.05$.

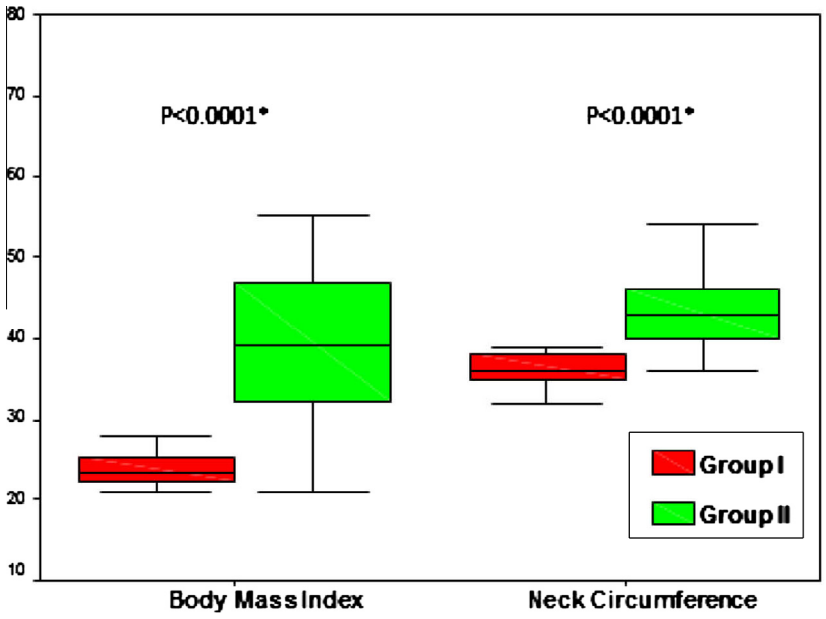

Figure 2 Body mass index and neck circumference in group I and group II.

Table 2 Patients' comorbidities and clinical apnea score.

	Group I (n = 21)	Group II (n = 51)	Significance
Co-morbidities			
Diabetes mellitus	5 (23.8%)	21 (41.2%)	$X^2 = 1.945$ $p = 0.163$
Hypertension	2 (9.5%)	38 (74.5%)	$X^2 = 25.442$ $p < 0.0001$*
Chest diseases	20 (95.2%)	27 (52.9%)	$X^2 = 11.74$ $p < 0.0001$*
COPD	10 (47.6%)	22 (43.1%)	
Pneumonia	1 (4.8%)	1 (2.0%)	
Bronchiectasis	3 (14.3%)	0 (0.0%)	
Pleural disease	1 (4.8%)	0 (0.0%)	
Interstitial lung diseases	3 (14.3%)	2 (3.9%)	
Pulmonary embolism	2 (9.5%)	2 (3.9%)	
Cardiac diseases	11 (52.4%)	30 (58.8%)	$X^2 = 0.25$ $p = 0.616$
Arrhythmia	9 (42.8%)	9 (17.6%)	
Ischemic heart diseases	1 (4.8%)	4 (7.8%)	
Decompensated heart failure	1 (4.8%)	4 (7.8%)	
Corpulmonale	8 (38.1%)	17 (33.3%)	
Hypothyroidism	0 (0.0%)	6 (11.8%)	$^{FE}p = 0.171$
Renal impairment	1 (4.8%)	14 (27.5%)	$^{FE}p = 0.052$
Malignancy	4 (19.0%)	1 (2.0%)	$^{FE}p = 0.023$*
Cerebrovascular stroke	1 (4.8%)	4 (7.8%)	$^{FE}p = 1.0$
Previous usage of CPAP or oxygen	0 (0.0%)	8 (15.7%)	$^{FE}p = 0.095$
CPAP	0 (0.0%)	4 (7.8%)	
Oxygen	0 (0.0%)	1 (2.0%)	
Oxygen and CPAP	0 (0.0%)	3 (5.9%)	
Clinical apnea score	0.3 ± 0.5	3.4 ± 1.1	$Z = 6.766$ $p < 0.0001$*

^{FE}p: Fisher's exact test; X^2: Chi-square test; Z: Mann–Whitney test.
* Significant at $p \leqslant 0.05$.

31.9 ± 29.4 (range: 0–100). The respiratory and oxygen saturation data as well as the heart rate analysis as scored from the sleep study are shown in Table 4, Figs. 3 and 4. The snoring analysis showed a snoring index of 153.9 ± 149.4 (range: 5–551.3) and a snore percent of total sleep time of 11.5 ± 12.9% (range: 0.0–47.0). The arrhythmia index was 104.6 ± 174.4. As regards the types of arrhythmia detected during polysomnogram, premature atrial contractions were the commonest encountered in 21 patients (53.8%) followed by bradytachyarrhythmia encountered in 18 patients (46.2%). Premature ventricular contractions were met in 16 patients (41%), sinus tachycardia in 14 patients (35.9%) and atrial fibrillation in 4 patients (10.3%).

The polysomnographic studies revealed that all 39 patients suffered from OSAHS. The latter was mild in 10.2% of patients (n = 4), moderate in 25.6% (n = 10) and severe in

Table 3 Patients' diagnosis, management and follow up.

	Group I (n = 21)	Group II (n = 51)	Significance
Diagnosis on admission			
Type II respiratory failure	15 (71.4%)	41 (80.4%)	$^{MC}p = 0.022^{*}$
Type I respiratory failure	5 (23.8%)	2 (3.9%)	
Pulmonary edema	0 (0.0%)	3 (5.9%)	
Shock	0 (0.0%)	4 (7.8%)	
Cerebrovascular stroke	1 (4.8%)	1 (2.0%)	
Total APACHE II score	20.7 ± 7.9	19.1 ± 6.8	$t = 0.858$ $Z = 0.394$
Predicted death rate	39.8 ± 24.6	34.3 ± 20.3	$Z = 0.54$ $p = 0.589$
Assisted ventilation and oxygen therapy			
Oxygen therapy	6 (28.6%)	4 (7.8%)	$^{MC}p < 0.0001^{*}$
CPAP	0 (0.0%)	10 (19.7%)	
BIPAP	4 (19.0%)	28 (54.9%)	
Invasive mechanical ventilation	11 (52.4%)	9 (17.6%)	
Length of ICU stay (days)	8.8 ± 6.1	9.5 ± 11.5	$Z = 0.597$ $p = 0.551$
Number of ICU admission over 6 months			
Once	14 (66.7%)	46 (90.2%)	$^{FE}p = 0.032^{*}$
Twice	7 (33.3%)	5 (9.8%)	
Mortality			
Deceased within 28 days	9 (42.8%)	11 (21.6%)	$^{MC}p = 0.091$
Deceased from 28 days – 6 months	0 (0.0%)	5 (9.8%)	
Alive after 6 months	12 (57.1%)	35 (68.6%)	

^{FE}p: Fisher's exact test; t: t-test; ^{MC}p: Monte Carlo test; Z: Mann Whitney test.
* Significant at $p \leqslant 0.05$.

Table 4 Respiratory, oxygen saturation and heart rate data at polysomnography.

Studied patients with SRBDs (n = 39)					
	Min–Max	Mean ± SD		Min–Max	Mean ± SD
AHI	6.5–135.2	50.5 ± 37.3	ODI 3%	1.0–125.7	42.9 ± 35.5
RDI	6.5–135.7	51.0 ± 37.2	ODI 4%	1.0–122.0	39.67 ± 35.35
Hypopnea index	0.7–120.5	28.4 ± 23.7	Average minimal saturation	48–93	77.9 ± 10.5
Obstructive apnea index	0.0–90.5	20.4 ± 23.2	Minimal saturation	37–92	64.8 ± 13.1
Mixed apneas index	0.0–14.6	0.9 ± 2.8	Baseline saturation	63–96	85.5 ± 7.4
Central apnea index	0.0–14.5	0.8 ± 2.6	Average saturation	54.0–96.0	81.41 ± 9.79
RERA	0.0–2.9	0.4 ± 0.7	T90%	0.0–100.0	66.0 ± 35.6
Average circulatory delay	13.5–25.7	19.6 ± 2.9	T85%	0.0–100.0	52.47 ± 37.58
Acceleration index	0.0–110.0	24.1 ± 29.9	Deceleration index	0.0–124.3	9.1 ± 23.3
Arrhythmia index	0.0–732.0	104.6 ± 174.4	Average heart rate	58–150	90.8 ± 20.6
Minimal heart rate	49–110	73.8 ± 18.1	Maximum heart rate	84–207	125.0 ± 29.4

AHI: apnea/hypopnea index; RDI: respiratory disturbance index; RERA: respiratory effort related arousal index; ODI 3%: oxygen desaturation more than 3% index; ODI 4%: oxygen desaturation more than 4% index; T90%: sleep time with oxygen saturation < 90%; T85%: sleep time with oxygen saturation < 85%.

64.1% (n = 25) of patients. Concomitant significant CSAS was encountered in 2 patients. The OSAHS patients were divided to two groups according to the associated SHVS: group IIa: those without SHVS which constituted 18% (n = 7) of patients; and group IIb: those with SHVS which constituted 82% (n = 32) of patients.

As regards association between COPD and SRBDs, we found fifteen patients in group II (representing 20.8% of all patients) to have AHI > 5/h and COPD with or without SHVS i.e. overlap syndrome. Since we had thirty patients in this study suffering from COPD, it means that 50% of them proved to have overlap syndrome.

The comparison between the characteristics of group IIa and group IIb is shown in Table 5. Among all the arterial blood gases analysis, only the serum bicarbonate level showed a statistically significant difference being higher in group IIb (Fig. 5). All anthropometric measures did not show any significant difference between the two groups. The pattern of obesity showed a central obesity in all patients (100%) in group IIa and in 29 patients (90.6%) in group IIb. There was no statistically significant difference between the two groups (p = 1.000).

Seventeen patients (53.1%) of group IIb, who represented 43.6% of patients with SRBDs and 23.6% of all the patients,

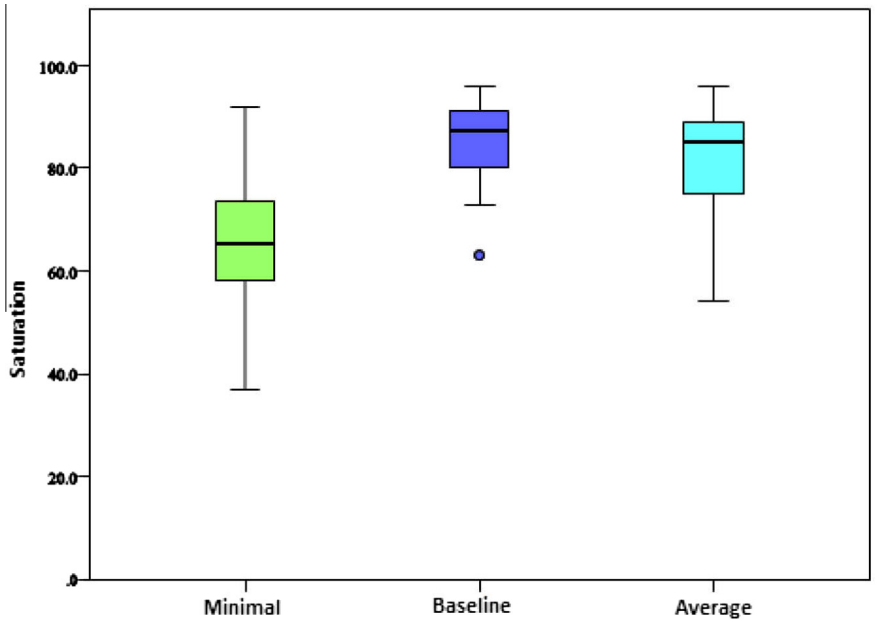

Figure 3 Oximetric values in the polysomnogram of the studied patients.

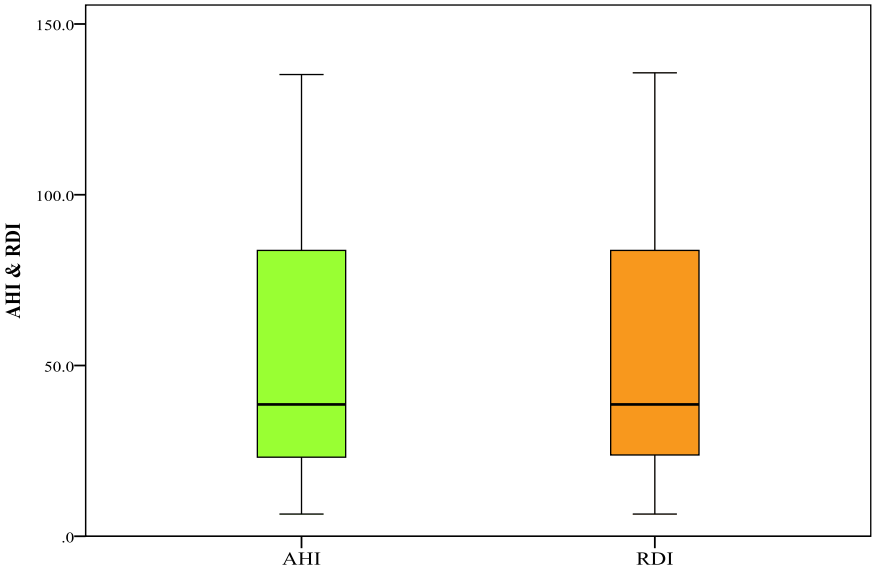

Figure 4 Apnea hypopnea and respiratory disturbance indices in the studied patients.

Table 5 Characteristics of group IIa and group IIb.

	Group IIa (n = 7)	Group IIb (n = 32)	Significance
Age (years)	59.29 ± 12.22	57.31 ± 12.48	^{t}p = 0.709
Body mass index (kg/m^2)	39.11 ± 5.19	41.70 ± 10.29	^{t}p = 0.524
Waist circumference (cm)	126.14 ± 8.99	127.47 ± 22.67	^{t}p = 0.803
Hip circumference (cm)	119.14 ± 6.62	118.88 ± 18.45	^{t}p = 0.949
Waist/hip ratio	1.06 ± 0.03	1.07 ± 0.07	^{t}p = 0.539
Neck circumference (cm)	41.43 ± 3.31	44.0 ± 3.75	^{t}p = 0.103
Hematocrit (%)	37.71 ± 10.20	45.09 ± 10.64	^{t}p = 0.103
PaO_2 (mmHg)	76.86 ± 32.86	53.03 ± 15.76	^{t}p = 0.106
$PaCO_2$ (mmHg)	51.14 ± 25.18	68.56 ± 11.97	^{t}p = 0.120
pH	7.31 ± 0.08	7.32 ± 0.06	^{t}p = 0.633
Bicarbonate (mmol/L)	23.36 ± 7.90	33.87 ± 5.26	^{t}p = 0.012*

^{t}p: p value for Student t-test.
* Statistically significant at $p \leqslant 0.05$.

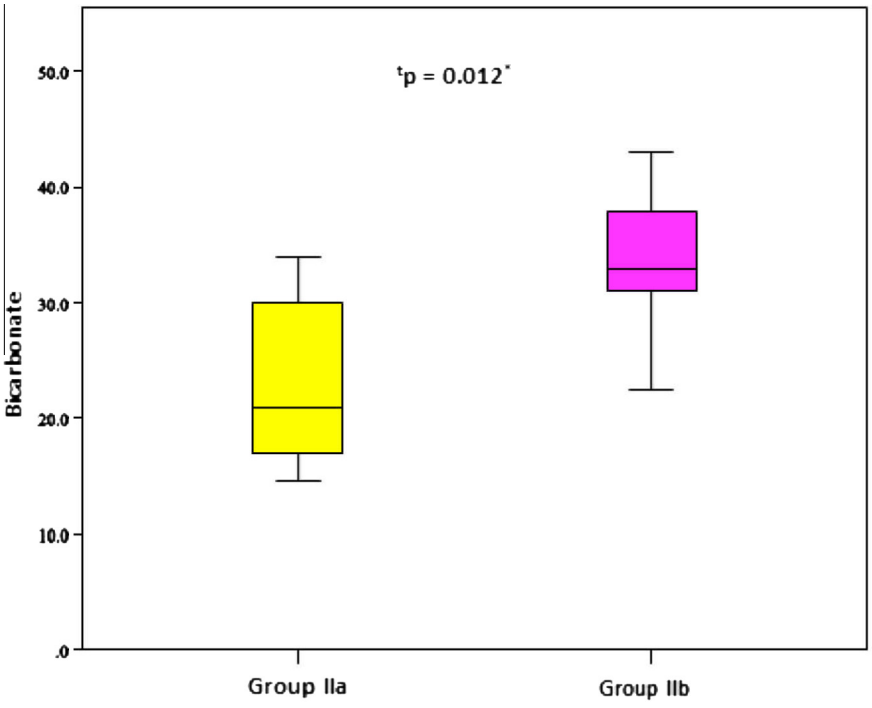

Figure 5 Bicarbonate level in group IIa and group IIb.

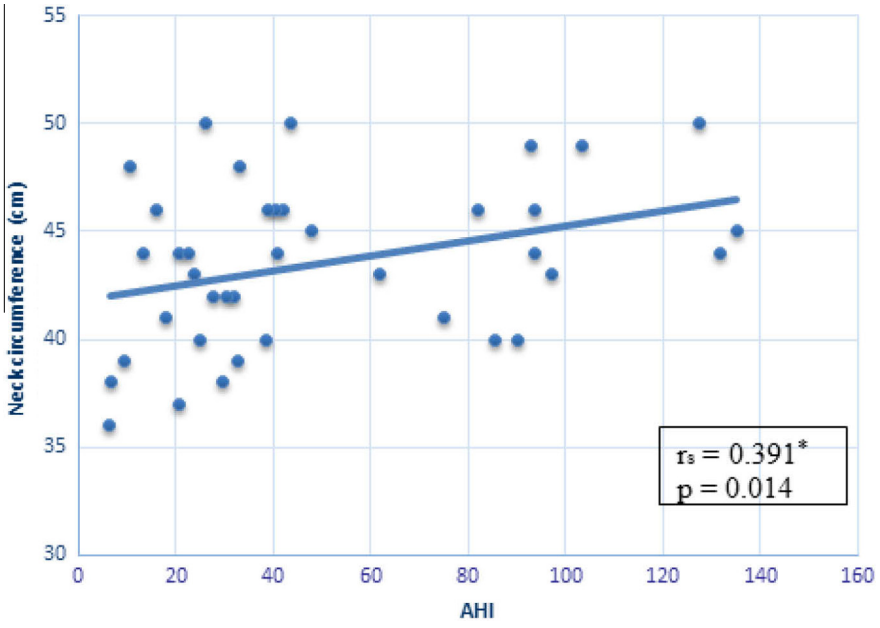

Figure 6 Correlation between apnea/hypopnea index (AHI) and neck circumference.

fulfilled the criteria of obesity hypoventilation syndrome (OHS): awake $PaCO_2 \geqslant 45$ mmHg, BMI $\geqslant 30$ kg/m^2 and absence of other causes of alveolar hypoventilation due to intrinsic lung pathology [11]. Seven of those 17 patients (9.7% of all admission) had BMI $\geqslant 40$ kg/m^2 and concomitant metabolic syndrome. This is a combination newly called malignant obesity hypoventilation syndrome (MOHS) [12]. Five of them had elevated liver enzymes maybe due to developing of nonalcoholic steatohepatitis. The remaining 15 (46.9%) out of 32 patients in group IIb, who represented 38.4% of patients with SRBDs, showed SHV attributed to other cause than OHS: COPD in 14 patients and congenital central hypoventilation in one patient.

Finally, we studied the correlations of the AHI with all the other parameters of the study. It showed a significant direct correlation with neck circumference ($r = 0.333$, $p = 0.014$)

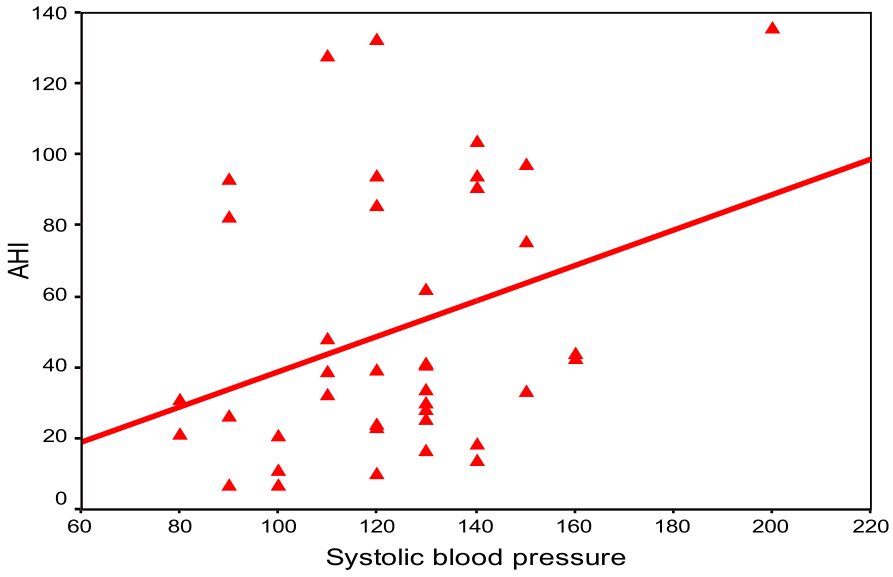

Figure 7 Correlation between apnea/hypopnea index (AHI) and systolic blood pressure.

(Fig. 6), systolic blood pressure ($r = 0.329$, $p = 0.041$) (Fig. 7), snoring index ($r = 0.488$, $p = 0.002$) and T85% ($r = 0.333$, $p = 0.038$). Moreover, it was found to be significantly higher in patients with diabetes mellitus than in those without ($Z = 2.325$, $p = 0.048$). It had a significant inverse correlation with PaO_2 ($r = -0.395$, $p = 0.013$), minimal saturation ($r = -0.477$, $p = 0.002$) as well as average saturation ($r = -338$, $p = 0.036$). Lastly, it did not show any significant correlation with age ($r = -0.044$, $p = 0.791$), BMI ($r = 0.239$, $p = 0.074$) nor clinical apnea score ($r = 0.233$, $p = 0.153$).

Discussion

In our study, 71% of patients admitted to ICU had a high clinical probability of SRBDs; and actually all of them who underwent sleep study were proven to have at least one sleep disorder, as all of them suffered from OSAHS. In concordance, Goring et al. [13] who evaluated 94 patients found that 77% of patients suffered from SRBDs proved by polysomnogram and the vast majority of those (95%) had OSAHS. Similarly, Adly et al. [14] in his study of the prevalence of SRBDs in patients with acute coronary syndrome admitted to ICU, found that 70% of his patients suffered from SRBDs including OSAHS (50%) and central sleep apnea – Cheyne–Stokes breathing (20%). Richards et al. [15] reported a lower prevalence of SRBDs in coronary care unit being 47% including both obstructive and central etiologies.

We found that 82% of patients with SRBDs suffered also from SHVS; and 53% of those fulfilled criteria of OHS. Nowbar et al. [16] prospectively evaluated obese patients with BMI $\geqslant$ 35 admitted to hospital in an attempt to determine the percentage of patients meeting the criteria for OHS versus simple obesity. They reported 6% to be obese patients and 31% of those suffered from OHS. We found 7 patients of those with OHS, who constituted 9.7% of all admission, suffering from MOHS and 71.4% of those, were females. Likewise, Marik et al. [12] reported a prevalence of MOHS in all patients admitted over an eight month period, of 8% of all admissions and 77% of them were females. Forty-one percent of our patients with OSAHS had SHVS caused by COPD: overlap syndrome. Those represented 21% of all admissions. Chaouat et al. [17] found the prevalence of sleep apnea in COPD patients to be 10–15%. Zammaron et al. [18] stated that overlap syndrome population tends to be older than the simple OSA population, with more frequent hypoxemia and hypercapnia, higher mean pulmonary artery pressures, but similar BMI.

The age of our patients with SRBDs was significantly higher than that of patients without SRBDs; a finding in agreement with the fact that SRBDs increase with age. The BMI and neck circumference of our patients with SRBDs were also significantly higher than that without SRBDs. Mortimore et al. [19] showed that neck circumference is the anthropometric measurement most closely associated with OSA, even in those with a normal BMI. We also found a significant positive correlation between AHI and neck circumference but there was no correlation between AHI and BMI. Caffo et al. [20] found neck circumference to be the most important predictor of OSAHS severity and to a lesser extent BMI, waist circumference, age and frequency of snoring. When we compared the anthropometric data of patients with SHVS and those without, there were no significant differences between the two groups. Shimura et al. [21] reported that leptin level was the only variable predictive of hypoventilation in Japanese men with OSA; whereas BMI, measures of OSA severity and visceral or subcutaneous fat measurements were not predictive of hypercapnia.

Systemic comorbidities are known to be higher in patients with SRBDs. In our study, the incidence of DM was much higher, almost double, in patients with SRBDs than in patients without SRBDs. The Wisconsin Sleep Cohort study [22] reported a 2-fold increase in risk of DM in 51387 subjects with OSA (defined by AHI $\geqslant$ 15) after adjustment for confounders. Tamura et al. [23] reported a prevalence of DM as high as 30% in 129 Japanese middle-aged adults with OSAHS. We also found that the AHI was significantly higher in diabetic patients than in non-diabetic ones and the post-prandial blood sugar was significantly higher in patients with SRBDs. Aurora

et al. [24] found the AHI and average oxygen saturation during sleep to be associated with elevated fasting and post-prandial glucose levels. Our patients with SRBDs had a significantly higher incidence of systemic hypertension compared to patients without SRBDs. In accordance, Silverberg et al. [25] found that about 50–60% of patients with SRBDs had systemic hypertension in comparison to only about 20% of general population; showing about 3 times more risk. We also found that AHI was positively correlated with systolic blood pressure value. Nieto et al. [26] previously reported this finding. Pulmonary hypertension was encountered in about a third of all of our patients, but it was not associated with the presence of SRBDs. Such lack of association was reported before [13]. We attributed the high prevalence of pulmonary hypertension to the high prevalence of COPD in both groups and OHS in patients with SRBDs. Indeed, COPD was by far the commonest concomitant intrinsic lung disease in our patients being significantly higher in patients without SRBDs. COPD, being a disease associated with systemic inflammation and many comorbidities, could alter the usual milieu of comorbidities allied with SRBDs.

Our laboratory investigations showed that serum uric acid was significantly higher in patients with SRBDs than in those without SRBDs. Garcia et al. [27] found 36% of patients with SRBDs had serum uric acid above established normal values. Also Marinchev et al. [28] reported that patients with gout and hyperuricemia have more severe OSAHS than subjects with osteoarthritis and OSAHS. The serum cholesterol and triglycerides were significantly higher in our patients with SRBDs. Togeiro et al. [29] studied a total of 1042 volunteers polysomnography, found subjects with moderate to severe OSA to have hither triglycerides levels than did the mild and non-OSA subjects. In fact, it was reported that nocturnal intermittent hypoxemia is independently associated with metabolic dyslipidemia [30].

Type II respiratory failure was by far the commonest cause of ICU admission of all of our patients; and it was a significantly higher cause in patients with SRBDs. Confalonieri et al. [31] who conducted a cohort study in 26 respiratory ICU, found that 581 patients (68%) had type II respiratory failure. BaHammam et al. [32] who studied respiratory failure in obese patients with SRBDs found that the reason for ICU admission for all subjects was type II respiratory failure. However, El-Solh et al. [33] found that most morbid obese patients including those with SRBDs were hospitalized most frequently because of type I respiratory failure. Noninvasive ventilation (NIV) precisely BiPAP was the cardinal mode of ventilation used in our patients with SRBDs. Bi-level NIV is the first line of management of acute hypercapnic respiratory failure in patients with OHS and OSAS. Carrillo et al. [34] studied 716 patients with type II respiratory failure (173 with OHS and 543 with COPD) and treated with a similar protocol of NIV. They concluded that patients with OHS were treated with similar efficacy but better outcomes than patients with COPD. We used invasive mechanical ventilation initially in patients with absolute contraindication to NIV or patients who failed to improve by it. Confalonieri et al. [31] in their study, found 56% of ICU patients needed NIV as first line of management where 21% of them needed invasive mechanical ventilation. Our patients with SRBDs had significantly less frequent repeated admission than those without SRBDs. This could be due to the fact that patients without SRBDs had significantly higher existence of an intrinsic pulmonary disease mostly irreversible leading to difficult and worse prognosis. In addition, SRBDs are treatable disorders once properly diagnosed.

The daytime arterial oxygen tension of our patients showed a significant inverse correlation with AHI; a finding which could be explained by the fact that most of our patients had severe OSAHS and hypoventilation syndrome. As we compared the arterial blood gases parameters of patients with SRBDs associated with SHVS and those without SHVS, we found only serum bicarbonate level to be significantly higher in patients with SHVS. Macavei et al. [35] confirmed a high prevalence of OHS in obese patients with OSAHS and reported that a high bicarbonate level (> 27 mmol/L) to be one of the most sensitive and specific predictors for the diagnosis of OHS.

Regarding sleep stages, light sleep stage time was encroaching on deep sleep stage time and REM sleep stage with reduced total sleep time. Such findings were comparable to those reported by Goring et al. [13] Sleep disruption has been recognized as a complication of acute illness. It is characterized by reduced nocturnal sleep efficiency and altered sleep architecture with increased wakefulness and stage 1 Non-REM sleep, together with reduced deep sleep and REM sleep [4]. Freedman et al. [36] studied 22 patients admitted to medical ICU (20 of whom were mechanically ventilated) to determine the effect of environmental noise on sleep disruption. Studied patients were found to be qualitatively and not necessarily quantitatively sleep deprived and environmental noise was not responsible for the majority of sleep fragmentation.

Limitations of the study

The sleep study should have been performed in every patient admitted to ICU and not only the high clinically predicted, as long as it is feasible to test the specificity of the clinical apnea score in such critically ill patients. Also the diagnosis of COPD in most patients was clinically and radiologically based and not by spirometry.

Conclusion

In ICU patients, SRBDs are common coexistent findings and every physician should systematically search for them. Type II respiratory failure is the main cause of ICU admission in patients with SRBDs. Quality of sleep in ICU is very disturbed. Most ICU patients with SRBDs have concomitant SHVS mostly due to OHS. Important comorbidities coexist in patients with SRBDs; both influence each other and should be identified and managed properly for the wellbeing of the patient. BiPAP therapy is the cardinal mode of ventilation used in patients with respiratory failure and SRBDs.

Conflict of interest

The authors have no conflict of interest to declare.

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