Predictors of early diagnosis of obesity hypoventilation syndrome among patients with sleep disordered breathing  
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**Abstract:** Background: Determine predictors of early diagnosis and prevalence of OHS among patient with sleep disordered breathing (SDB) at Mansoura SDB unit.  
**Aim of work:** Determine predictors of early diagnosis and prevalence of OHS among patient with sleep disordered breathing (SDB) diagnosed at Mansoura SDB unit. **Patients and Methods:** The study population consists of 212 patients referred to Mansoura University SDB unit for investigation of possible SDB. All patients were subjected to the following: Clinical evaluation, Pulse oximeter, Erect awake ABG, Spirometry and Polysomnography.  
**Results:** It was found that; The overall prevalence of OHS in studied cases was 13.2% (28 out of 212) and the prevalence of SDB in all studied cases was 83.02% (176 out of 212), 15.91% (28 out of 176) with OHS. The following predictors can be used for early diagnosis of OHS; a cutoff point of 93% of erect awake SpO₂ with sensitivity of 97.1%, Specificity of 100%, p<0.001, a cutoff point of 28 mmol/dL of serum HCO₃ with sensitivity of 85.7%, specificity of 95.6%, p <0.001, and a cutoff point of 46.40 of AHI with sensitivity of 78.6%, specificity of 77.9 p = 0.001.  
**Conclusion:** The prevalence of OHS among patients with SDB diagnosed at Mansoura SDB unit was 15.91% and 16.67% among obese patients with SDB. The following cutoff points can be used as promising predictors for early diagnosis of OHS: Erect awake SpO₂< 93% ,, Serum HCO₃>28 mmol/L and AHI > 46.4.  
**Key words:** obesity, hypoventilation, polysomnography, SDB  

**Background :**  
Obesity hypoventilation syndrome (OHS) is defined as a combination of obesity (ie, body mass index (BMI) >30 kg/m²) and awake chronic hypercapnia (ie, arterial partial pressure of carbon dioxide (PaCO₂) ≥ 45 mmHg) accompanied by sleep disordered breathing (SDB) in the absence of any other reason of hypoventilation. [1].OHS is often unrecognized and treatment is frequently delayed, and can cause secondary erythrocytosis, pulmonary hypertension, and cor pulmonale. The delay in recognizing and treating this condition
increases health care resource use and the likelihood of requiring hospitalization compared with patients who have similar degrees of obesity [2].

The prevalence of OHS in the general population is unknown, however; some reported prevalence of around 10-20% in outpatients presenting to sleep clinics. and a current estimate suggests around 0.37% of the US population may have OHS [1]. In the view of rapidly increasing numbers of individuals joining the ranks of the morbidly obese, and the significantly greater need they have for medical care, OHS needs to be considered as a significant clinical and social problem. Unfortunately it is also one that is frequently underestimated; despite the significant comorbidities and higher hospitalization rates were experienced [3].

An early identification is a key element in managing patients with OHS. Current data however suggest that this disorder is frequently overlooked despite the high rate of hospitalization and medical care interaction received by these individuals in the years prior to a diagnosis being made [2]. Those with chronic stable disease frequently present through sleep clinics, where screening for hypoventilation may not be undertaken. Sleep histories in these patients are usually indistinguishable from simple OSA, with reports of excessive daytime sleepiness, loud snoring and nocturnal choking episodes common. [4]. clinically, it is important to distinguish between those individuals with simple OSA and those with OHS. Although therapy directed towards correcting obstructed breathing at night will be effective in some patients with OHS, but in others persisting or worsening hypoventilation will occur [3].

Several alveolar laboratory findings are supportive of OHS, yet the definitive test for hypoventilation is an arterial blood gas performed on room air. Elevated serum bicarbonate level due to metabolic compensation of respiratory acidosis is common in patients with OHS and points toward the chronic nature of hypercapnia [3]. Therefore, serum bicarbonate from venous blood could be used as a sensitive test to screen for chronic hypercapnia [4] .Additionally, hypoxemia during wakefulness is not common in patients with simple OSA. Therefore, abnormal arterial oxygen saturation detected via finger pulse oximetry (SpO₂) during wakefulness should also lead clinicians to exclude OHS in patients with OSA [5].
Aim of work
Determine predictors of early diagnosis and prevalence of OHS among patient with sleep disordered breathing (SDB) diagnosed at Mansoura SDB unit.

Patients and Methods: This prospective study was conducted at Mansoura SDB unit, Mansoura University Hospital, Dakahlia, Egypt during the period from March 2013 to September 2014. The study population consists of 212 patients were investigated for possible SDB. Those patients were referred either from the respiratory clinics, or other specialties (such as cardiology, ENT and endocrinology or from outside private clinics). Inclusion criteria; Patients with symptoms suggestive of sleep disordered breathing. Exclusion criteria; Patients younger than 18 years.

All patients were subjected to the following: Clinical evaluation: A detailed history which included; demographic data (age, gender and smoking status), presenting symptoms (daytime sleepiness by Epworth Sleepiness Scale (ESS), snoring, witnessed apnea, fatigue), Berlin questionnaire and STOP-Bang questionnaire. Medical history taken (respiratory, metabolic and cardiovascular comorbidities). Anthropometric measurements such as height, weight, BMI, neck circumference (NC), and Mallampati score were performed. Laboratory work up: Complete metabolic profile including CBC, liver function, serum creatinine, sodium and potassium. Thyroid function tests (freeT3, freeT4, TSH). Lipogram ECG at rest. Radiological study: Chest x ray was performed to exclude other causes of hypoventilation e.g. COPD, bronchiectasis. Pulmonary function tests: Pulse oximeter in sitting position to measure erect awake SpO2, Erect awake ABG. Spirometry was performed by (smart PFT CO) manufactured by medical equipment Europe-Hammet burg- Germany. Full night attended PSG: Using in-lab attended PSG to monitor brain activity electroencephalogram, muscle tone (electromyogram of both the legs and chin), eye movements (electrooculogram), electrocardiogram, oxygen saturation (finger pulse Oximeter), chest and abdominal wall movements (thoracic and abdominal belts), airflow (thermistor and nasal prong pressure transducer), and snoring (microphone). The PSG recording was performed using (SOMNO-screen, SOMNO medics, Germany). According to AASM, 2012 (6) AHI, ODI, basal oxygen saturation, minimum nocturnal SpO2 and % of time with SpO2 < 90% were recorded on PSG.
Cases will be considered to have OSA, CSA or OHS based on:-

i. **OSA if present A+B+D or C+D**

   A- At least one of the following applies: a) Complaints of excessive day time sleepiness, b) Awakening with chocking, c) Loud snoring and/or interruption of sleep.

   B- AHI ≥ 5/h and evidence of respiratory effort during all or portion of the event.

   C- AHI ≥ 15/h and evidence of respiratory effort during all or portion of the event.

   D- The disorder is not explained by another current sleep disorder, medical or neurologic disorder, medication use, or substance use disorder.

ii. **CSA if have; 1) AHI ≥ 5/h, 2) Central apneas / hypopneas >50% of total apneas / hypopneas, 3) Central apneas / hypopneas ≥5/h and 4) Symptoms of either excessive sleepiness or disrupted sleep.**

iii. **OHS if have** BMI > 30kg/m², awake erect PaCO₂ ≥ 45 mm Hg and exclusion of diseases that can cause hypoventilation e.g. neuromuscular or chest wall diseases, or airway diseases like COPD and bronchiectasis.

**Statistical analysis:** The statistical analysis data was done by using excel and SPSS programs, statistical package for social science version 17. Qualitative data were presented as number and percent, chi square and student t- test were used to compare between two groups. The analysis of data was done to Test statistically significant difference between groups. P < 0.05 was considered statistically significant.

**Results:** It was found that; among the 212 patients studied, the mean age was 46.6 ± 11.2, 112 patients (52.8%) were men and 100 (47.2%) were women. The mean value of BMI was 41.74 ± 10.60 kg/m². Also it was found that; 128 (60.4%) patients were nonsmokers, 28 (13.2%) patients were Ex-smokers and 56 (26.4%) patients were current smokers.

![Figure (1): Flow diagram (A) of studied cases classification according to SDB. OHS (obesity hypoventilation syndrome), OSA (obstructive sleep apnea), CSA (central sleep apnea)](image-url)
This flow diagram (A) shows that the prevalence of SDB in studied cases was 83.02% (176 out of 212), 81.82% (144 out of 176) with OSA, 15.91% (28 out of 176) with OHS, and 2.27% (4 out of 176) with CSA. While 16.98% (36 out of 212) were without SDB.

Figure (2): Flow diagram (B) of studied cases

This flow diagram (B) shows that 94.3% (200 out of 212) of studied cases were obese, 84% (168 out of 200) of them had SDB, 80.95% (136 out of 168) with OSA, 16.67% (28 out of 168) with OHS, and 2.38% (4 out of 168) with CSA. While non-obese were 5.7% (12 out of 212), 66.7% (8 out of 12) had SDB and all of them (100%) were OSA. OHS patients were divided into 2 groups, 26 cases out of 28 (92.9%) with OSA, and 2 cases out of 28 (7.1%) without OSA.
<table>
<thead>
<tr>
<th></th>
<th>Obese OSA patients (n = 136)</th>
<th>OHS patients (n= 28)</th>
<th>(\chi^2)</th>
<th>(P)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male gender</td>
<td>74 54.4%</td>
<td>12 42.9%</td>
<td>0.764</td>
<td>0.430</td>
</tr>
<tr>
<td>Female gender</td>
<td>62 45.6%</td>
<td>16 57.1%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Snoring</td>
<td>136 100%</td>
<td>26 92.9%</td>
<td>4.917</td>
<td>0.027*</td>
</tr>
<tr>
<td>Witnessed apnea</td>
<td>66 48.5%</td>
<td>24 85.7%</td>
<td>6.483</td>
<td>0.011*</td>
</tr>
<tr>
<td>Early awakening headache</td>
<td>74 54.4%</td>
<td>26 92.9%</td>
<td>7.211</td>
<td>0.007*</td>
</tr>
<tr>
<td>Choking during sleep</td>
<td>58 42.6%</td>
<td>24 85.7%</td>
<td>8.613</td>
<td>0.003*</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age(year)</td>
<td>46.32 ± 10.92</td>
<td>49.14 ± 11.15</td>
<td>0.879</td>
<td>0.382</td>
</tr>
<tr>
<td>BMI(kg/m(^2))</td>
<td>41.98 ± 9.49</td>
<td>51.13 ± 11.23</td>
<td>3.183</td>
<td>0.002*</td>
</tr>
<tr>
<td>Neck Circumference(cm)</td>
<td>43.31 ± 4.27</td>
<td>44.57 ± 2.41</td>
<td>1.529</td>
<td>0.136</td>
</tr>
<tr>
<td>Hemoglobin %</td>
<td>12.59 ± 1.83</td>
<td>13.43 ± 2.57</td>
<td>1.444</td>
<td>0.153</td>
</tr>
<tr>
<td>Berlin score</td>
<td>2.69 ± 0.60</td>
<td>2.93 ± 0.27</td>
<td>2.319</td>
<td>0.025*</td>
</tr>
<tr>
<td>STOP-Bang score</td>
<td>5.00 ± 1.22</td>
<td>6.14 ± 0.86</td>
<td>3.325</td>
<td>0.001*</td>
</tr>
<tr>
<td>Epworth sleepiness scale (ESS)</td>
<td>12.99 ± 5.33</td>
<td>15.79 ± 6.84</td>
<td>1.702</td>
<td>0.093</td>
</tr>
</tbody>
</table>

In table (1); it was noticed that, OHS patients having SDB symptoms (witnessed apnea, early awakening headache and choking during sleep) more than obese OSA patients with significant differences (\(p < 0.05\)). While obese OSA patients show significantly higher snoring than OHS patients (100% vs 92%) \(P < 0.05\). Also; OHS patients had significantly higher BMI, Berlin and STOP-Bang score than obese OSA patients. However; there were no significant differences as regard age, gender, ESS (\(P = 0.093\)), hemoglobin %, and neck circumference (NC).
Table (2): Comparisons between obese OSA patients and OHS patients as regards pulse oximeter, ABG and Polysomnographic parameters.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Obese OSA patients (n = 136)</th>
<th>OHS patients (n= 28)</th>
<th>t</th>
<th>P</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>mean ± SD</td>
<td>mean ± SD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Erect awake SpO2 %</td>
<td>96.40 ± 1.93</td>
<td>87.71 ± 4.23</td>
<td>7.526</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>PH</td>
<td>7.38 ± 0.03</td>
<td>7.36 ± 0.03</td>
<td>2.219</td>
<td>0.029*</td>
</tr>
<tr>
<td>PaO2 (mmHg)</td>
<td>83.83 ± 8.95</td>
<td>57.58 ± 6.69</td>
<td>10.374</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>PaCO2 (mmHg)</td>
<td>39.94 ± 4.05</td>
<td>53.69 ± 2.88</td>
<td>12.070</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>HCO3 (mmol/L)</td>
<td>24.71 ± 2.03</td>
<td>28.74 ± 3.60</td>
<td>4.055</td>
<td>0.001*</td>
</tr>
<tr>
<td>Basal SPO2 % (awake supine)</td>
<td>93.27 ± 2.97</td>
<td>87.79 ± 3.09</td>
<td>6.242</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>AHI (event/hour)</td>
<td>29.74 ± 21.85</td>
<td>58.89 ± 30.45</td>
<td>4.233</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>% of total sleep time of SpO2&lt;90%</td>
<td>18.92 ± 26.11</td>
<td>84.62 ± 17.18</td>
<td>8.997</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Minimum SpO2 %</td>
<td>78.35 ± 9.51</td>
<td>60.50 ± 13.97</td>
<td>4.570</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>Oxygen Desaturation Index (ODI)</td>
<td>31.44 ± 27.02</td>
<td>77.39 ± 31.10</td>
<td>5.647</td>
<td>&lt;0.001*</td>
</tr>
</tbody>
</table>

Table (2), showed that; OHS patients had significantly lower erect awake SpO2, PaO2 and PH values, but significantly higher PaCO2 and HCO3 values (p<0.001) in comparison to obese OSA patient. Also; highlights a number of statistically significant differences according to PSG data. In comparison between OHS versus obese OSA patients we found that OHS patients had significantly higher AHI (58.89 ± 30.45 versus 29.74 ± 21.85), (p <0.001) and significantly higher ODI (77.39 ± 31.10 versus 31.44 ± 27.02), (p <0.001). Also OHS patients had significantly lower basal (awake supine) SPO2% (87.79 ± 3.09 vs 93.27 ± 2.97), (p < 0.001), spent significantly longer durations of SpO2<90% (84.62 ± 17.18 vs 18.92 ± 26.11), (p <0.001), and they exhibited significantly lower minimum SpO2 (60.50 ± 13.97 vs 78.35 ± 9.51), (p <0.001).
As regard predictors of early diagnosis of OHS related to pulse oximeter, ABG and polysmonographic parameters; a cutoff point of 93% of erect awake SpO2 with sensitivity of 97.1%, Specificity of 100%, PPV of 100%, NPV of 87.5% , and p<0.001, a cutoff point of 28 mmol/dL of HCO3 from ABGs with sensitivity of 85.7%, specificity of 95.6%, PPV of 80%, NPV of 97%, and p <0.001, a cutoff point of 91.15% of basal (awake supine) with sensitivity of 85.3%, specificity of 100% and, PPV of 100%, NPV of 58.3%, and p <0.001, a cutoff point of 46.40 of AHI with sensitivity of 78.6%, specificity of 77.9%, PPV of 42.3%, NPV of 94.6%, and p = 0.001, a cutoff point of 45.6 of % of total sleep time of SpO2<90% with sensitivity of 100%, specificity of 82.35%, PPV of 53.85%, NPV of 100% and p < 0.001, and a cutoff point of 75.5% of minimum SpO2 with sensitivity of 70.6%, specificity of 85.7%, PPV of 96%, NPV of 37.5% and p < 0.001 can be used as a predictors for diagnosis of OHS.

**Figure (3):** Receiver operating curve (ROC) analysis for erect awake SpO2 (A), Serum HCO3 (B), basal (supine awake) SpO2 (C), and AHI (D) as a predictors for diagnosis of OHS.
Discussion:

In this study, it was found that the prevalence of SDB (AHI ≥5) in studied cases was 83.02% (176 out of 212), the majority of them had OSA (81.8% (144 out of 176) and 15.91% (28 out of 176) had OHS, however; The prevalence of OHS in all studied cases was 13.2% (28 out of 212). With respect to obesity; SDB was present in 84% (168 out of 200) of obese patients and 66.7% (8 out of 12) of non-obese patients. Our results approach to what reported by BaHammam [7], Goring and Collop [8], who reported a prevalence of 85.8%, 77% respectively of SDB in all cases referred to their SDB units and also approach to what reported by Prudon and West, [9], who reported a prevalence of 88% of SDB among 288 obese out patients referred to sleep clinic.

Goring and Collop [8], also found that SDB was detected in 60% of patients with normal weight, which was comparable to our result of 66.7% of patients with normal weight had SDB, but it was difficult to draw statistically significant difference between obese and non-obese because of the small numbers of subjects in non-obese category.

In our study, it was found that; the prevalence of OHS among patients with SDB diagnosed at Mansoura SDB unit was 15.91% (28 out of 176), 16.67% (28 out of 168) among obese patients with SDB, this is within the range reported in previous studies about the prevalence of OHS among patients with OSA that ranged from 9%-20%. In a large French retrospective study (n=1141), the prevalence of OHS was 11% among OSA patients and in a subgroup analysis of subjects with a BMI >40 kg/m² the prevalence of OHS was 24% [11]. Studies that reported a high prevalence of OHS recruited patients with high BMI (≥40 kg/m²), as in Mokhlesi et al [12] study that reported a prevalence of 20% in a model of obese OSA patients with a mean BMI of 43 kg/m². Our result showed that 20.6% among obese patients with OSA with mean BMI of 41.98 kg/m². To some extent low prevalence of 8.5% was seen among Saudi OSA patients in BaHammam study as the mean BMI in the studied group was 36.3 kg/m² but he found that the prevalence of OHS among OSA patients with a BMI ≥ 40 kg/m² was 21% [7].

Our study showed that; 7.1% (2 out of 28) of our patients had hypoventilation without obstructive events (pure OHS), while 92.9% had concomitant OSA. These are supported by the previous studies that showed the majority of patients with OHS have concomitant OSA (90%). The residual 10% of patients have non-obstructive sleep
hypoventilation (characterized by an AHI <5/hour) [1], BaHammam [7] also found that prevalence of pure OHS was 6.25% and this result approach our result.

Previous studies that made an effort to determine risk issues or predictors of OHS in cohorts of patients with OSA reported mixed results [6]. The majority of patients have the typical symptoms of OSA, as loud snoring, episodes of choking during sleep with witnessed apneas, excessive daytime sleepiness, and early awakening headache [13]. In our study it was found that OHS patients having significantly higher symptoms of (witnessed apnea, early awakening headache and choking during sleep) than obese OSA patients (p < 0.05). While obese OSA patients show significantly higher snoring than OHS patients. This higher snoring in OSA Vs OHS patients can be explained by the presence of some cases of OHS without OSA (pure OHS), while the significantly higher other symptoms of OSA (witnessed apnea, and choking during sleep) can be explained by significantly higher AHI in OHS Vs OSA patients, while higher early awakening headache in OHS Vs OSA patients can be explained by the more sleep hypoventilation in OHS Vs OSA patients as early awakening headache is caused by nocturnal CO₂ accumulation that lead to vasodilatation of cerebral vessels that is relieved by awakening with increase of ventilation by return of wakefulness drive.

We also found that OHS patients had significantly higher Berlin and STOP Bang score Versus obese OSA patients (2.93 ±0.27 Vs 2.69 ± 0.60, P =0.025) and (6.14 ± 0.86 Vs 5.00 ± 1.22, P = 0.001) respectively. These can be explained by higher severity of OSA in OHS patients Versus those with pure OSA patients.

In our study it was found that there is no significant differences in OSA Versus OHS patients as regard hemoglobin percentage and this can be explained by the small number of OHS patients and presence of four patients in OHS group that had co-morbid renal impairment which affects erythropoiesis and leads to anemia. Also other causes of decrease of hemoglobin can occur like parasitic infestations which were not searched for.

Kaw et al [14] , discovered that severity of obesity, severity of OSA and pulmonary restrictive impairment were risk factors for hypercapnia , this support our results of significantly higher BMI and AHI of OHS patients in comparison to obese OSA patients (51.13 ± 11.23 Vs 41.98 ± 9.49, P= 0.002 and 58.89 ± 30.45 Vs 29.74 ± 21.85, p <0.001) respectively and a restrictive spirometric pattern reflected by lower FEV1% (56.36 ± 20.47 Vs 79.96 ± 21.90, p <0.001) and lower FVC% (52.00 ± 22.35 Vs 79.96 ± 20.14, p <0.001) and higher
FEV1/FVC% (93.21 ± 10.06 Vs 85.41 ± 13.73, P = 0.047) values with statistically significant differences. These are comparable to what reported by Macaviet al, [15], who registered that BMI of OHS patients Vs obese OSA patients was (41.6 ± 7.7 Vs 37.9 ± 6.5) and prevalence of daytime hypercapnia increased with BMI increase (10.9% in class 1 obesity, 20.5% in class 2 obesity, and 30.4% in morbidly obese patients), and comparable to what reported by BaHammam who found that AHI in OHS patient was significantly higher than OSA patients (68.2 ± 47.1 Vs 46.5 ± 34.1, p < 0.001), and a restrictive spirometric pattern reflected by lower FEV1% (56.8 ± 26.1 Vs 85.3 ± 18, P < 0.001) and lower FVC% (57.4 ± 24.6 Vs 86.6 ± 19.3, P < 0.001) and higher FEV1/FVC% (85.5 ± 10.1 Vs 84.5 ± 11.2, P = 0.4) [7]. The increase in BMI can explain the higher degree of restrictive ventilatory defect in OHS Vs OSA [16]. The higher AHI in OHS Vs OSA can support the hypothesis of daytime hypercapnia can be caused by higher severity of AHI that lead to severe hypoxemia with hypoventilation with subsequent rise of cerebrospinal fluid HCO₃ that lead to suppression of respiratory center with subsequent hypoventilation. The returns of HCO₃ to normal value take few hours after awakening [17].

In current study it was found that the erect awake SpO₂ and PaO₂ was significantly lower in OHS than obese OSA patient (p<0.001) and a level ≤ 93% of erect awake SpO₂ can be used as a predictor for diagnosis of OHS with sensitivity of 97.1%, Specificity of 100%, PPV of 100%, NPV of 87.5%, and p <0.001. Basoglu and Tasbakan, [18] in their study established that daytime SpO₂ ≤ 95% had a sensitivity of 64.4% and specificity of 73.9%, (P=0.003) for recognizing OHS. So our result had more sensitivity and specificity at lower cutoff point. This was supported by Balachandran et al, who reported that Patients with OHS are unlikely to have a PaO₂ >70 mmHg so a SpO₂ < 93% should suggest the need for additional testing [19].

In our study it was found that OHS patients had significantly higher serum HCO₃ values than obese OSA patients (28.74 ± 3.60Vs 24.71 ± 2.03, P = 0.001) and serum HCO₃ was identified as a predictor of OHS with a cutoff point of ≥ 28 mmol/L with sensitivity of 95.6%, specificity of 85.7%, PPV of 80%, NPV of 97%, and p <0.001. In previous studies Mokhlesi et al[12], in their sector of OSA patients found that a serum HCO₃ threshold of ≥ 27 mmol/L had a sensitivity of 92% and specificity of 50% in predicting OHS, Basoglu and Tasbakan [20], reported that HCO₃ level ≥ 27 mmol/L had a sensitivity of 88.1% and specificity of 73.1%, and Pingol et al [21] found that a serum HCO₃ level of ≥ 27 mmol/L as the cutoff gives an acceptable decision for the diagnosis of OHS (sensitivity of 76.6%, specificity of 74.6%, PPV of 54.5%, NPV of 88.9%). Our cutoff point is slightly higher than
reported by Mokhlesi et al, Basoglu and Tasbakan, and Pingol et al but with higher sensitivity and specificity. While gold standard for diagnosis of OHS still elevated arterial PaCO₂, a rise in the HCO₃ level due to metabolic compensation of respiratory acidosis has been found to be a sensitive screening tool for daytime hypercapnia [12]. Treger et al in their study found that the mean arterial minus venous difference for HCO₃ was −0.80 mmol/L, so ABG can be replaced by peripheral venous sample as a puncture of the artery is very painful and harmful [23].

Our study shows that OHS patients had significantly lower basal (awake supine) SPO₂ % than obese OSA patients (87.79 ± 3.09 Vs 93.27 ± 2.97, p < 0.001), and a cutoff point of 91.15% of basal (awake supine) SpO₂ can be used as a predictor for diagnosis of OHS with sensitivity of 85.3%, specificity of 100% and, PPV of 100%, NPV of 58.3%, and p <0.001. So this parameter in polysomnographic report can raise the possibility of OHS, so needing subsequent confirmation by ABG.

In the current study, OHS patients had higher AHI scores than those for obese OSA patients (58.89 Vs 29.74 events/hour, p <0.001), and a cutoff point of 46.40 of AHI can be used as a predictor for diagnosis of OHS with sensitivity of 78.6%, specificity of 77.9%, PPV of 42.3%, NPV of 94.6%, and p = 0.001. In a previous study, Mokhlesi et al [12].defined an AHI of 100/h has a specificity of 85% and sensitivity of 45% for the presence of OHS . But the severity of AHI is not generally accepted as a good predictor of OHS, because the use of AHI severity as a predictor of OHS may cause significant under diagnosis of critical medical problem especially pure OHS [23].

A useful tool used for the screening of OHS when interpreting sleep studies, appears to be the amount of time spent with SpO₂ < 90% [6]. In our study OHS patients spent significantly higher % of total sleep time of SpO₂< 90% than obese OSA patients (84.62 ±17.18 Vs 18.92 ± 26.11, p <0.001), and was known as a predictor of OHS with a cutoff point of ≥ 45.6 % of total sleep time and can be used as a predictor for early diagnosis of OHS with sensitivity of 100%, specificity of 82.35%, PPV of 53.85%, NPV of 100% and p < 0.001.Our results coincide with those of previous studies as in BaHammam and Mokhlesi et al, studies that showed that % of total sleep time of SpO₂ < 90% in OHS patients is higher than OSA patients (71±34.3 Vs 10.5±20.5, p <0.001), (56% Vs 19% , p <0.001) respectively, but they didn't report cutoff point for their studies [7, 12].
In our study we got that OHS patients exhibited significantly lower minimum SpO$_2$ than obese OSA patients ($60.50 \pm 13.97$ Vs $78.35 \pm 9.51$, $p < 0.001$), and a minimum SpO$_2$ of $< 75.5\%$ as the cutoff point can be used as a predictor for diagnosis of OHS with sensitivity of 70.6\%, specificity of 85.7\%, PPV of 96\%, NPV of 37.5\% and $p < 0.001$, this is approach what reported by Bingol et al (21), who found that a minimum Spo$_2$ of $< 80\%$ as the cutoff point gives an acceptable discrimination for the diagnosis of OHS (sensitivity of 82.8\%, specificity of 54.5\%, PPV of 56.9\%, NPV of 81.4\%) .

Our study had some limitations. First, it was done at a single center in Egypt so multi center studies are needed in our country to confirm these results. Second, the number of all patients and patients with OHS was small; it would have been interesting to compare patients with OHS with OSA to pure OHS patients. Unfortunately we found only two patients with pure OHS, so statistical comparisons were not possible. Third, we cannot asses sleep hypoventilation in patients with OSA as there is no available non-invasive monitoring for PaCO$_2$ in our department like end tidal CO$_2$ or trans-cutaneous Co2 (PtcCO$_2$). If it is available, it can detect those OSA with sleep hypoventilation that later on after some times will develop OHS as it is well known that sleep hypoventilation precedes day time hypoventilation. So early detection of sleep hypoventilation with subsequent treatment can prevent the development of more cases of OHS with its higher comorbidities.

**Conclusion**

1. The overall prevalence of OHS in Mansoura SDB unit is 13.2\% and the prevalence among patients with SDB is 15.91\% while 16.67\% among obese patients with SDB.
2. The following cutoff points are a promising predictors that can be used in early diagnosis of OHS:
   - Erect awake SpO$_2$< 93\% with sensitivity of 97.1\% and Specificity of 100\%.
   - Serum HCO$_3$>28 mmol/L with sensitivity of 85.7\% and specificity of 95.6\%.
   - Basal (awake supine) SpO$_2$ %< 91.15\% with sensitivity of 85.3\% and specificity of 100\%.
   - AHI > 46.4 with sensitivity of 78.6\% and specificity of 77.9\%.
   - The percentage of total sleep time of SpO$_2$<90\% >45.6\% with sensitivity of 100\% and specificity of 82.35\%.
   - Minimum SpO$_2$% <75.5\% with sensitivity of 70.6\% and specificity of 85.7\%. 
Recommendations

- Multi-center studies are recommended to determine the prevalence of OHS among Egyptians.
- Screening for OHS among patients with SDB using the above cutoff points might be helpful in early diagnosis, prevention, and better management of this serious disease.

Conflict of interest

No conflict of interest.

References


