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Short Communication Open Access

Reflections on Obesity Hypoventilation Syndrome

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Obesity hypoventilation syndrome (OHS) is defined as the combined presence of obesity (BMI ≥ 30 kg/m^2) with chronic daytime hypercapnia ($PaCO_2$>45 mmHg) in the absence of other pathologies that cause hypoventilation (neuromuscular, metabolic, lung and chest diseases) [1]. Untreated OHS patients have lower quality of life, higher health-care utilization, significantly worse cardiovascular morbidity, and greater risk of hospitalization and death compared with eucapnic obese patients [2,3]. Despite of its significant morbidity and mortality, there is still insufficient data about the prevalence, the clinical characteristics and the predictors of OHS. The impact of ethnicity was not studied either. It seems also, that OHS is frequently underestimated by physicians which lead to more complications as a result of delayed diagnosis. The prevalence of OHS in general populations is insufficiently documented, as data about OHS are mostly described in patients with suspected or confirmed obstructive apnea hypopnea syndrome (OAHS). Previous studies reported the prevalence of OHS among patients with OSAHS that ranged from 9-20% [4]. A retrospective analysis of data on 525 sleep clinic patients suspected of having sleep disordered breathing showed that hypercapnia was present in 20.6% obese and 22.1% OAHS patients [5]. In studies that required patients with high BMIs (≥ 40 kg/m^2), OHS prevalence tends to be higher. It was the case for an American study that revealed a prevalence of 20% in a sample of obese OAHS patients with a mean BMI of 43 kg/m^2 [6].

Regarding the OHS prevalence in the general population, Mokhlesi estimated that 0.37% of general adult United States (US) population (1 in 270 adults) may have OHS [7]. This estimation was based on the fact that approximately 5% of the general US population has severe obesity while half of the patients with severe obesity have OSAHS, and 15% of severely obese OSAHS patients have OHS.

Overall, we can conclude that less than 1% of the general population may have OHS, versus 10-20% in obese as well as patients with OAHS. However, OHS is likely to increase due to increasing obesity rates worldwide.

Worldwide, obesity prevalence is increasing, with future projections predicting that over one billion people, or approximately 20% of the entire adult population of the world, will be obese by 2030 [8]. In Tunisia today, obesity is recognized as a major public health problem with a high prevalence that has almost tripled over the last three decades, according to the latest statistics of the world health organization. It has increased from 8.7%% in 1980 [9] to 28% in 2010 among adults >30 years, which means that over than three million Tunisian adults are obese [10]. To our knowledge, there are no data on the OHS prevalence in the Tunisian obese population. As obesity is of growing concern in this population, an accurate assessment of the OHS prevalence is definitely needed in order to plan health services and make provisions for this condition.

Despite its significant morbidity and mortality, OHS is frequently under diagnosed because arterial blood gases (ABD) is not routinely performed in obese patients, due to its invasive nature [11]. OHS also remains under recognized by physicians with only the third of patients receiving the right diagnosis when admitted with acute-on-chronic hypercapnia respiratory failure [12]. Moreover, even if OHS is diagnosed during systemic in-hospital screening, the majority of identified OHS patients are discharged from the hospital without any form of therapy for hypoventilation, as was reported by investigators from the University of Colorado at Denver [13].

These data show that the identification of OHS clinical predictors in obese subjects and of the clinical pattern of the disease is more than needed in order to help physicians understand and diagnose the disorder at an early stage. Previous studies have been conducted to assess the clinical characteristics and the predictors of hypercapnia in obese subjects, but most of them were in cohort of patients with OSAH and reported discordant data.

Kaw et al. found that the severity of obesity, according to the BMI, were a significant determinant of hypercapnia [14]. Similarly, a more recent report from United Kingdom showed that the prevalence of daytime hypercapnia increased with BMI while Harada from Japan [15] implied that abdominal obesity, as assessed by waist circumference, was a risk factor of chronic hypoventilation as was previously reported [16]. In contrast of these findings, others reports did not find BMI to be a predictor of OHHS.

In a sample of American patients with OSAHS, two sleep parameters have been identified as independent predictors of OHS: apnea hyponea index (AHI), and lowest oxygen saturation during sleep. In the same study, an AHI of 100/h was defined as a highly specific threshold for the detection of patients with OHS. A Similar conclusion has been recently drawn by Pıhtılı [17] about 419 Turkish with OSAHS. However, the contribution of AHI in OHS prediction remains widely controversial as was mentioned by Bahammam (14) In his study including a large sample of Saudi patients with OSAHS, it was showed that not AHI but sleep time of nocturnal arterial oxygen saturation less than 90% ($nSaO_2$<90%) was a predictor of OHS.

Some biological parameters were also described as a significant determinant of hypercapnia in such obese subjects such. A calculated

HCO_3- cutoff >27 mmol/L was found to have a 85.7% sensitivity and a 89.5% specificity for the diagnosis of OHS in an English sleep cohort of 525 patients suspected of having sleep disordered breathing [5].

Therefore, it has been suggested that a high calculated HCO_3- level could be a sensitive and a specific screening tool for the diagnosis of OHS. Similar findings were also reported by others research studies.

The same suggestion was taken up for the elevated haemoglobin concentration, according to a recent Japanese study the high level of C-reactive protein (CRP) [18] as well as the increased blood levels of leptin. Leptin, which is a human protein secreted by adipocytes, acts on the central respiratory drive to stimulate ventilation. High serum leptin levels in patients with OHS are noted in comparison to those without such sleep-disordered breathing. Curiously, increased serum level of leptin was found to be associated with a reduced stimulatory effect on respiratory drive which leads to a higher risk of hypoventilation and consequent hypercapnia [19].

Elsewhere, several studies showed that patients with OHS are characterized by a lower total pulmonary capacity (LPC), first-second-forced-expiratory-volume (FEV_1), forced vital capacity (FVC) and FEV_1/FVC in comparison with eucapnic obese patients [14]. In contrast, there was no significant difference in residual volume (RV). This restrictive ventilator defect identified in patients with OHS would be independent from BMI as was revealed by a Japanese multicentric study. In this study, OSAHS patients with OHS were compared to markedly obese OSAHS patients without hypercapnia and it has found that FVC, and FEV_1 would be rigorous determinants of hypercapnia in obese subjects [20]. It was the same for carbon monoxide diffusing capacity/ alveolar volume according to another Japanese study. Authors explained this correlation by a high cardiac output, a higher total and central blood volume, and a low lung volume in patients with OHS which was previously reported

In contrast to these findings, other researchers did not find any correlation between plethysmographic parameters and $PaCO_2$, and therefore concluded that only bicarbonate serum level is a predictor of hypercapnia.

For all the reasons mentioned above, authors conclude that large sample studies would be required to determine the prevalence of OHS in the population at large. It would be also interesting to identify useful screening tools for routinely diagnosis of OHS among obese patients.

Competing Interests

The authors declare that they have no competing interests.

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