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Original article

Obesity-hypoventilation syndrome and associated factors[☆]

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ABSTRACT

Background and objectives: Obesity causes important alterations in the respiratory physiology like sleep obstructive apnoea (SOA) and obesity-hypoventilation syndrome (OHS), both associated with high morbidity and mortality.

Also, these entities are clearly infradiagnosed and in the case of OHS the prevalence is unknown in the general obese population.

To determine the prevalence of OHS in the population of patients with morbid obesity and to know the comorbidity related with OHS, the associated respiratory symptoms and the pulse oximetry alterations.

Patients and method: Descriptive study. Selection of 136 adult patients with morbid obesity (BMI > 40). Collected were anthropometric data, toxic habits, concomitant disease, symptom data, analytic data, dyspnoea grade, sleepiness scale (Epworth Test), electrocardiogram, chest X-ray, spirometry, nocturne ambulatory pulse oximetry and arterial gasometry.

Results: 136 were studied, mean age 60 years old (SD 12.9 years), 73% (98) were women; 6.6% of patients presented diurnal hypercapnia indicative of OHS; 72% presented high blood pressure, 44% dyslipidaemia, 18% presented cardiovascular disease, 83% snored and 46% had apnoea; 30% presented stage II dyspnoea and 10% stage III.

The desaturation/hour index was above 3% ≥ 30 of occasions in 28.6% of patients and the percentage of patients with saturations <90% more than 30% of the time was 23.5%. The results were worse in patients with OHS.

Conclusions: The prevalence of OHS was lower than expected. Noteworthy were the high comorbidity of cardiovascular disease and the high frequency of respiratory symptoms associated with important alterations of pulse oximetry.

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Síndrome de obesidad-hipoventilación y factores asociados

RESUMEN

Fundamento y objetivos: La obesidad ocasiona alteraciones importantes de la fisiología respiratoria como el síndrome de apnea-hipoapnea del sueño (SAHS) y el síndrome de obesidad-hipoventilación (SOH), asociados ambos a elevada morbimortalidad.

Además, estas entidades están claramente infradiagnosticadas y en el caso de SOH se desconoce la prevalencia en población general obesa. Los objetivos del estudio son: determinar la prevalencia del SOH en población de pacientes con obesidad mórbida y conocer la comorbilidad asociada al SOH, la clínica respiratoria y las alteraciones de la pulsioximetría.

Palabras clave:

Obesidad

Hipoventilación

Hipercapnia

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Pacientes y método: Estudio descriptivo. Se seleccionaron 136 pacientes adultos con obesidad mórbida (IMC > 40). Se recogieron datos antropométricos, hábitos tóxicos, enfermedades concomitantes, registro de síntomas, datos analíticos, grado de disnea, escala de somnolencia, electrocardiograma y radiografía de tórax. También se realizó espirometría, pulsioximetría nocturna domiciliaria y gasometría arterial.

Resultados: Se estudiaron 136 pacientes, con una media de edad de 60 años (DE: 12,9 años); el 73% (98) fueron mujeres. El 6,6% presentaban hipercapnia diurna indicativa de SOH. Presentaban hipertensión arterial (HTA) el 72%, dislipidemia el 44% y enfermedad cardiovascular (ECV) el 18%, sin diferencias según presentaran o no SOH. Tenían ronquidos el 83% y apneas el 46%. El 30% presentaban disnea grado II y el 10% grado III.

El índice de desaturaciones/hora (IDH) superior al 3% en ≥ 30 ocasiones estaba presente en el 28,6% de los pacientes y el porcentaje de tiempo con saturaciones de $O_2 < 90\%$ más del 30% del tiempo lo presentaban el 23,5%, resultados peores en pacientes con SOH.

Conclusiones: La prevalencia de SOH es más baja de la esperada. Destacan la elevada comorbilidad cardiovascular y frecuentes síntomas respiratorios, presenten o no SOH, así como alteraciones importantes en la pulsioximetría.

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Introduction

Obesity is one of the main public health problems in today's world. The World Health Organization already speaks of a real epidemic that affects both developed and developing countries.¹ In 2015, 1 of every 3 adults in the world was overweight (body mass index [BMI] ≥ 25 kg/m²) and almost 1 in 10 adults was obese (BMI ≥ 30 kg/m²).² The prevalence figures for obesity according to the Spanish Society for the Study of Obesity (SEEDO),³ defined as a BMI > 30 in adults, are 14.5%, 2% with an BMI > 35 and 0.5% with a BMI > 40. Obesity is a risk factor associated with chronic diseases with a great impact on premature mortality and quality of life and a high economic cost. The prevalence of cardiovascular diseases, diabetes, dyslipidaemia and hypertension is clearly increased in obese patients, as well as osteoarticular diseases.

While the association between obesity and cardiovascular diseases has been the subject of many studies, respiratory morbidity has been relegated to the background, even though obesity determines important alterations in respiratory physiology, such as alterations in ventilatory mechanics, respiratory musculature and the regulation of ventilation during sleep.^{4,5}

Obstructive sleep apnoea/hypopnea syndrome (OSAHS) and obesity-hypoventilation syndrome (OHS) are two of the most representative diseases regarding the consequences of obesity on respiration. They share common pathophysiological mechanisms and similar clinical signs, which can cause diagnostic confusion, although it should be made clear that they are different entities. OHS seems to be associated with increased morbidity and mortality related to severe respiratory and cardiac compromise.⁴

The prevalence of OSAHS in the general population is 2–4%, and increases drastically with obesity's severity, being the main risk factor, reaching 50% in cases of morbid obesity. There are other factors related to OSAHS such as age, male sex and hereditary factors.^{6,7} This syndrome is related to cardiovascular complications that can determine prognosis such as ventricular dysfunction, arterial hypertension, cerebrovascular and pulmonary disease.^{8,9} Nocturnal polysomnography constitutes the diagnostic test.

OHS is less known and was initially described as Pickwick's syndrome. The first studies conducted on this syndrome were made by Auchincloss et al.¹⁰ and Bickelmann et al.,¹¹ describing patients with obesity, hypersomnolence, secondary erythrocytosis, pulmonary hypertension and cor pulmonale. It is characterized by chronic alveolar hypoventilation with diurnal hypercapnia (PaCO₂ > 45 mmHg), in the absence of significant respiratory or muscle disease in patients with obesity (BMI > 30).^{12,13} The polysomnographic characteristic is a significant nocturnal hypoxaemia. The clinical presentation is usually insidious with symptoms

of hypersomnolence, cognitive alterations, headaches, hypertension and heart failure.

The likelihood of hypoventilation increases with BMI, although the pathogenesis of this entity is possibly multifactorial, involving three possible causes: increased respiratory work and respiratory muscle weakness (possibly the main cause), respiratory centre dysfunction and sustained effects of recurrent episodes of obstructive sleep apnoeas. Leptin produced in adipocytes has also been suspected in the pathogenesis of this syndrome.¹³

The majority of patients with this syndrome suffer from OSAHS concomitantly and several studies support the claim that OHS may constitute the final progression of OSAHS, although approximately 10–20% do not have any evidence of nocturnal OSAHS; these patients develop hypoventilation related to sleep, especially during REM sleep,^{14,15} requiring nocturnal polysomnography to determine the pattern of nocturnal hypoventilation (obstructive or non-obstructive).

Although this syndrome is associated with severe cardiovascular disease and early mortality, higher than that observed in OSAHS, the diagnosis is made when the disease is in a highly advanced stage, in the fifth or sixth decade, after a severe respiratory failure episode.^{12,13}

Most studies have been conducted in patients who have been admitted and diagnosed due to severe respiratory failure or patients with OSAHS, and, in these cases, an increase in prevalence, possibly related to the obesity epidemic, has been observed.¹⁶ In a study conducted with healthy obese women and with BMI > 30, 11% had daytime hypercapnia.¹⁷ Another study conducted with obese hospitalized patients found 30% of patients with hypercapnia not attributable to other causes.¹⁴

Given the importance of an early diagnosis and that the prevalence of OHS in the general population with morbid obesity is not known, the present study is carried out with the objective of knowing this prevalence, the comorbidity associated with OHS and nocturnal pulse oximetry changes in patients with morbid obesity.

Materials and method

A descriptive study carried out in an urban area of the province of Barcelona that covers a population of 110,000 inhabitants. The Pneumology Service of the Parc Sanitari Sant Joan de Déu in Sant Boi de Llobregat and the 6 corresponding primary care centres that serve the population of Sant Boi de Llobregat, Sant Vicenç dels Horts and Santa Coloma de Cervelló participated in the study.

Patients older than 18 years of age treated in primary care with a clinical history of a BMI ≥ 40 were included. Patients with terminal illnesses and dementias with dependence for basic activities of daily life were excluded.

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