

Obesity and apnea: is it always the same?

Volume 10 Issue 1 - 2018

Keywords: obesity, apnea, hypercapnia, bmi, pco2, osa**Abbreviations:** OHS, obesity-hypoventilation syndrome; OSA, obstructive sleep apnea; BMI, body mass index; PAP, positive pressure therapy

Introduction

The Obesity-Hypoventilation Syndrome (OHS) is defined as the combined presence of hypercapnia ($p\text{CO}_2 > 45$ mmHg) and obesity ($\text{BMI} \geq 30$) in a patient with sleep respiratory disorder and in the absence of others causes of hypoventilation.³⁻⁹ It is an important sleep disorder that can lead to hospitalization in acute cases.⁸ The prevalence in general population is 1,1% and ranges from 10 to 20 % in obese patients and with obstructive sleep apnea (OSA).⁴

The sleep disorder which is present in 90% of the cases is OSA, and the other 10% are represented by sleep hypoventilation.³

Case report

A 40-year-old-male driver, was referred to the Sleep Ambulatory of the Federal Hospital of Lagoa, in Rio de Janeiro - Brazil, with the following complaints: snoring since childhood, nasal blockage, hypersomnolence, memory loss, morning headache, sleep fragmentation with 5 to 6 times going to the bathroom during the night of sleep and cognitive impairment. Presented with high blood pressure and chronic venous insufficiency in the use of Hydrochlorothiazide, Allopurinol and Acetylsalicylic Acid. The patient's had a 40kg weight gain in the past three years; with worsen of the snoring and sleep apnea episodes.

Physical exam revealed obesity (Body Mass Index-BMI: 45); Cervical circumference: 49cm (>40cm considered to be most likely to have OSA); Oroscopy: Modified Mallampati IV, palatine tonsils II (Brodsky), soft palate web; Nasal endoscopy revealed signs of chronic rhinopathy with pale mucosa and inferior turbinates hypertrophy and non-obstructive septal deviation on the left side.

Daytime sleepiness according to the Epworth 5 sleepiness scale (24 pts - Normal Range <10) and high risk of sleep apnea, according to the prediction of the STOP-BANG10 scale (7 pts - High Risk >5).

Polisomnography resulted in severe sleep apnea with the following parameters: Apnea and Hypopnea Index (AHI)/h: 80,4 (<5: normal; 5-15: mild; 15-30: moderate; >30: severe); Apnea Index: 79,8; Hypopnea Index: 96; Sat O₂ % med: 74% / Sat O₂ % min 61% / Sat O₂ <90%: 74,2. Gasometry revealed hypercapnia ($p\text{CO}_2$: 45,8 mmHg / $p\text{O}_2$: 66,5 mmHg). Spirometry was normal and bronchodilator test showed negative to Salbutamol. We've had also performed a chest X-ray that showed no alterations. The thyroid-stimulating hormone and thyroxine (T₄) were also normal.

Due to the high cost of BIPAP in our country, CPAP was recommended and follow-up to assess its effectiveness. Besides that, the patient was also referred to a nutritionist as well as to a physical education teacher, to start a cardiovascular and weight loss program. Information about bariatric surgery was also explained to the patient.

Luciane F Mello Jose Eduardo Esposito
Almeida, Marianna Pires Cordeiro Caiana,
Patrícia Ciminelli Linhares, Rubem Brito
University Hospital Federal da Lagoa, Brazil

Correspondence: Luciane F Mello Jose Eduardo Esposito, MD
Resident, Rua Jardim Botânico, 501. Rio de Janeiro-RJ, Brazil, Tel
55-21-982831166, Email contato@dralucianemello.com.br

Received: July 13, 2017 | **Published:** January 30, 2018

Discussion

OHS is an exclusion diagnosis and the patients must be evaluated if they have other pathologies as an interstitial or obstructive pulmonary disease; chest wall pathologies; severe hypothyroidism; neuromuscular disease and central hypoventilation 3. Therefore, the investigation must include arterial gasometry, spirometry, pulmonary image exams and thyroid hormones.^{3,9}

The diagnosis may occur as one of the following: acute exacerbation with acute respiratory insufficiency, respiratory acidemia and the necessity of admission in an intensive unit care or during a routine examination, with a specialist.³ The symptoms include snoring, choking attacks during sleep, hyper somnolence and morning headache.^{3,9} Furthermore, the patients may develop dyspnea and present clinic signs of cor pulmonale.¹

The detection of this pathology is important because of the high hospital cost due to exacerbations and serious complications such as pulmonary hypertension and right ventricular failure.^{1,2}

Obesity causes an impact in ventilation in many ways. During sleep, chronic alveolar hypoventilation leads to hypercapnia and nocturnal hypoxemia and, consequently, a ventilatory drive alteration that becomes more tolerant to high levels of CO₂.^{2,9} Furthermore, the overweight patient presents a leptin resistance, hormone responsible for satiety and for stimulate the ventilation process. At last, the fat accumulation may overload the respiratory system with the reduction of the lung volume, elasticity of the chest wall and increase of the airway pressure.^{2,9}

The aims of the treatment are to correct the basic cause through the reversion of the respiratory disorder by positive pressure therapy (PAP) and weight loss.^{7,8} There are two modalities of PAP: CPAP and BIPAP.⁶⁻⁸ The latter, being the initial option of treatment since the bilevel device offers more comfort and efficacy in a scenery that requires higher levels of air pressure.^{2,6-8}

During the use of BIPAP, the patient must present saturation higher or at least 90%, with the absence of snoring, apnea, paradoxical

breathing. The treatment efficacy can be measured by somnolence scales, clinical condition and arterial gasometry.⁶⁻⁸ If the saturation of O₂ does not reach 90%, O₂ can be added to the therapy. Other options include procedures for weight loss and, as a last resource, tracheostomy.^{2,7,8}

In our country, due the financial restrictions on the public health system it's quite difficult to diagnose and treat those patients. Both the polisomnography exam and PAP therapy are expensive and rare in public hospitals.

Acknowledgements

None.

Conflicts of interest

Author declares there are no conflicts of interest.

Funding

None.

References

1. Almeneessier AS, Nashwan SZ, Al-Shamiri MQ, et al. The prevalence of pulmonary hypertension in patients with obesity hypoventilation syndrome: a prospective observational study. *J Thorac Dis.* 2017;9(3):779–788.
2. Bahammam A. Acute ventilatory failure complicating obesity hypoventilation: update on a 'critical care syndrome. *Curr Opin Pulm Med.* 2010;16(6):543–551.
3. Balachandran JS, Masa JF, Mokhlesi B. Obesity Hypoventilation Syndrome Epidemiology and Diagnosis. *Sleep Medicine Clinics* . 2014;9(3):341–347.
4. Borel JC, Guerber F, Jullian-Desayes I, et al. Prevalence of obesity hypoventilation syndrome in ambulatory obese patients attending pathology laboratories. *Respirology.* 2017;22(6):1190–1198.
5. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep.* 1991;14(6):540–545.
6. Orfanos S, Jaffuel D, Perrin C, et al. Switch of noninvasive ventilation (NIV) to continuous positive airway pressure (CPAP) in patients with obesity hypoventilation syndrome: a pilot study. *BMC Pulm Med.* 2017;17(1):50.
7. Piper A. Obesity hypoventilation syndrome: weighing in on therapy options. *Chest.* 2016;149(3):856–868.
8. Priou P, Hamel JF, Person C, et al. Long-Term Outcome of Noninvasive Positive Pressure Ventilation for Obesity Hypoventilation Syndrome. *Chest.* 2010;138(1):84–90.
9. Shetty S, Parthasarathy S. Obesity Hypoventilation Syndrome. *Curr Pulmonol Rep.* 2015; 4(1):42–55.
10. Reis R, Teixeira F, Martins V, et al. Validation of a Portuguese Version of the STOP-Bang Questionnaire as a Screening Tool for Obstructive Sleep Apnea: Analysis in a Sleep Clinic. *Rev Port Pneumol.* 2015; (2006)21(2):61–68.