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THE IMPORTANCE OF DIAGNOSING OBSTRUCTIVE SLEEP APNEA IN A PATIENT ADMITTED TO A CORONARY ICU: CASE REPORT

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ABSTRACT

Obstructive sleep apnea is a pathological condition caused by the collapse of the upper airway during sleep, interfering with the respiratory cycle. This pathology has a direct impact on the quality of life of affected patients, since it alters the quality of sleep and, in the long term, is related to systemic arterial hypertension, fatigue and heart failure. The objective of this article is to present a clinical case report of a patient hospitalized for pneumonia who was also diagnosed with obstructive sleep apnea, which allowed the institution of treatment and a visible improvement in his quality of life, demonstrating the effectiveness of positive pressure treatment and the benefits of early diagnosis of this pathology.

Keywords: Apnea. Obstruction. Sleep. CPAP.

INTRODUCTION

Cardiovascular diseases (CVD) are considered the leading cause of death in the world1,2,3. Specifically in Brazil, regardless of the reduction in the mortality rate from these diseases observed in recent years4,5, possibly as a result of successful health policies, the country still concentrates the highest number of deaths from diseases of the circulatory system, especially stroke and coronary artery disease.

In addition to the high mortality rate3, CVDs result in a reduction in quality of life and significantly compromise the world economy6-8. Research related to the economic theme indicates that, in less than 10 years, the costs of CVD have more than doubled, reaching 210 billion euros in 20179. In low- and middle-income countries, such as Brazil, these diseases represent a threat to economic and social development, since the costs related to them were estimated at more than half of the economic burden of non-communicable diseases and, together, represented 2% of the GDP of these countries between 2011 and 201510.

According to Costa et al. (2015)[11], more than 50% of patients under medical follow-up for cardiovascular diseases have a high risk of sleep-disordered breathing. However, most report never having been informed about this possibility, which does not motivate them to seek specialized care and treatment. In the current scenario, most of these individuals with sleep disorders remain undiagnosed for years or even throughout their lives, representing a population at imminent risk of illness and mortality.

In this sense, studies aimed at analyzing the risks of comorbidities related to sleep disorders, as well as the factors associated with the occurrence of these outcomes, are of paramount importance with regard to the search for a better identification of patients at risk, early treatment and prevention of non-fatal and fatal events.

Many studies have shown that sleep disorders cause various damages to the health of individuals, being associated with metabolic alterations and a higher risk of cardiovascular diseases and mortality12-14. In the city of São Paulo, the main cause of hospitalizations or demand for emergency services was due to cardiovascular events and/or arterial hypertension, with the presence of insomnia complaints being an important predictor factor15. These repercussions of sleep disorders generate high costs for the public coffers, adding up to direct expenses of approximately 15.9 billion dollars/year with accidents related to sleep problems16.

OSA is characterized by intermittent obstructions of upper airway airflow during sleep due to episodes of apnea and hypopnea, resulting in sleep impairment17. These obstructive events may occur due to both anatomical and non-anatomical factors.

Anatomical factors in OSA are mostly related to alterations in pharyngeal tissues and in the anatomy of the face and mandible18. On the other hand, non-anatomical factors are related to changes in central breathing control, in addition to lifestyle habits, such as smoking and sedentary lifestyle17-19.

It is estimated that OSA affects around 1 billion individuals worldwide, and Brazil is the 3rd country with the most people with OSA20. In Brazil, the study by Tufik et al. (2010) found that the prevalence of OSA was 26.1% among women and 40.6% among men, with a population average of 32.9%. When the epidemiological characteristics were observed, it was observed that 40% of the population included in the study was at the ideal weight, 38% were overweight and 21% were obese. When evaluated from the perspective of the apnea-hypopnea index, 62.6% of the obese male patients had the result >15; among females, this percentage was 21.8%[21].

The results of the study by Tufik et al. are especially ~~preocupantes~~ alarming when looking at the context of the global obesity epidemic. Recent studies show that the average levels of body mass index (BMI) of the population began to rise in the 1970s, first in rich countries, and the trend spread to underdeveloped countries, such as Brazil, where obesity began to affect the richest layers of the population first, and today it is already concentrated in rural areas and with lower economic power. The United States leads the global obesity ranking, with 38.2% of the population obese, and in the Latin American bloc, there is an accelerated increase in BMI gains by the general population in countries such as Argentina, Uruguay, Chile, Paraguay, and Brazil22.

According to data published in 2020 by the Brazilian Institute of Geography and Statistics (IBGE), the proportion of obese people in Brazil more than doubled in the period from 2003 to 2019 in the adult population. In women, obesity went from 14.5 to 30.2%, while in men it went from 9.6 to 22.8%. If only patients in the 25 to 39 age group were evaluated, the prevalence is even higher: 58.3% for men and 57% for women23.

Because it is an insidious and progressive disease, sometimes the diagnosis of OSA is delayed, which leads to medium and long-term complications, caused by systemic inflammation resulting from oxidative stress and changes in the cardiopulmonary functioning pattern, such as arterial hypertension, heart failure (HF) and arrhythmias, which together lead, in the long term, to an increase in mortality in patients with untreated OSA25,26.

There is a strong correlation between HF patients and OSA, indicating that up to 36.6% of patients with heart failure have some degree of obstructive sleep apnea, with this rate reaching up to 50% when it comes to heart failure with preserved ejection fraction

(HFpEF)[27,28]. In addition, the epidemiological profile of these patients corresponds to the male gender, with a high body mass index (BMI), with metabolic diseases such as systemic arterial hypertension (SAH) and diabetes mellitus (DM)[27].

The diagnosis of OSA is based on a rigorous clinical evaluation, which begins with the investigation of signs and symptoms; Generally, patients with OSA, or their relatives, complain of loud snoring, non-restorative sleep that causes daytime sleepiness, and fatigue29. The gold standard test for diagnosing obstructive sleep apnea is polysomnography, which measures several patient parameters during sleep, such as electroencephalogram (EEG), electrooculogram, electromyogram, electrocardiogram, position sensor, snoring sensor, positioning sensor, plethysmography straps, pulse oximeter and respiratory flow channels, cannula, and thermistor30.

Early diagnosis favors patients affected by OSA by preventing the insidious progression of the pathology, reducing the incidence of SAH and HF, and significantly improving sleep quality25. In addition, in patients already with HF, early treatment of OSA considerably reduces decompensation, reducing hospitalizations in those patients who adhere to positive pressure therapy25,30.

The objective of this article is to report the clinical case of a 37-year-old patient diagnosed with heart failure with preserved ejection fraction, who was hospitalized due to respiratory failure caused by bacterial pneumonia. During hospitalization, the patient was diagnosed with OSA, which allowed early treatment and, after resolution of the infection, improved quality of life through treatment with positive pressure ventilation.

METHODS

This article is a clinical case report with a literature review. Data related to patient hospitalization, test results, and clinical evolution were obtained through a search of medical records at the hospital where the care was performed. In parallel, a brief literature review was carried out in databases such as PubMed and Lilacs, where recent and relevant articles on the subject were obtained. The research in question followed all the guidelines recommended by the National Council for Ethics in Research (CONEP).

CASE REPORT

A 37-year-old male patient, obese grade III, was admitted to the emergency room in the morning complaining of dyspnea and fatigue for 4 days, with significant worsening in the last 12 hours, associated with broken speech and tiredness on minimal exertion. The patient was promptly monitored, with room air oxygen saturation of 77%. On physical

examination, there were diffuse rales in the chest. Oxygen support in a high-flow mask was initiated, which reduced patient fatigue.

Due to a history of previous pulmonary thromboembolism (PTE), the main suspicion was of a new condition of the disease, given the significant exacerbation of the patient within a few hours. Laboratory and imaging tests were requested, such as transthoracic ultrasound (ECOtt) and ultrasound (USG) with lower limb doppler, and heparin was started in an infusion pump. Due to the patient's obesity, a CT angiography cannot be performed, given the limitations of the device available at the service.

The ECOtt revealed no signs suggestive of right ventricular (RV) overload, in addition to delivering normal left ventricular (LV) function parameters. Lower limb Doppler ultrasound also ruled out any evidence of ongoing thromboembolic phenomenon; the combined ECOtt and USG results ruled out the diagnosis of PTE, although confirmation could be made by CT angiography, which could not be performed.

During the intensive care unit stay, the patient was maintained on high-flow, noninvasive ventilatory support, with improvement of the initial symptoms, complaining only of mild dyspnea on exertion in bed. Febrile peaks and deterioration of cardiac auscultation were recorded, which led to the initiation of antibiotic therapy for the treatment of pneumonia.

Due to the maintenance of the pattern of tachypnea and respiratory distress during hospitalization, obesity and other risk factors, the hypothesis of obstructive sleep apnea was raised. The hypothesis was discussed with the physiotherapy team, which opted for a type IV polygraph to screen for the disease. The test result suggested severe apnea. A physiotherapy evaluation showed a cervical circumference of 50cm and Malampatti II.

It was decided to start positive pressure therapy with automatic pressure between 10 and 14cmH2O per nasal route and the ResMed equipment from the Airsense 10 AutoSet line was used. There was a technical problem with the operation of the first CPAP, but it was promptly replaced. With the end of antibiotic therapy for pneumonia and improvement of the patient's clinical appearance, he was discharged from the ICU and went to the ward and, later, was discharged from the hospital, with follow-up with the physiotherapy team and guidance on treatment and recommendations for lifestyle changes, especially weight loss.

During the 30-night interval of evaluation, it was possible to observe an excellent therapeutic use, as the average time of daily use was more than 4 hours. The leakage remained at 4.8 L/min, staying below the threshold value stimulated by the manufacturer. The global Apnea and Hypopnea Index (AHI) presented values of 0.8 events per hour, and values below 5 events/hour were considered normal.

After this adaptation period, the pressure was set at 10 cmH2O for 5 days and humidification was set at level 5. During 5 nights, it was possible to observe excellent therapeutic use, as the average time of use was more than 4 hours. The leakage of 4.8L/min was again below the threshold value stimulated by the manufacturer. The AHI was 0.1 events/hours.

During this period of use of positive pressure therapy at home, there was a need for small adjustments, such as the position and number of pillows used during the patient's sleep, as leaks were detected, but which were resolved after the suggestions made by the team.

The patient reported that he adapted well to the nasal interface with no associated complaints. She did not present significant complaints associated with dryness of the mouth and throat. He reported improved sleep quality, significant improvement in daytime sleepiness, tiredness, and amounts of awakenings. Currently, he is being monitored with specialized physiotherapy via telemonitoring.

DISCUSSION

The patient in the case presented here has several relevant diagnoses and, in fact, it cannot be inferred how, individually, they can influence the emergence and progression of the others. However, there is a well-established and elucidated relationship between OSA and HF progression, due to several mechanisms such as intermittent hypoxia, sympathetic activation, which leads to increased blood pressure and heart rate, and arrhythmias31.

It has been proven that patients with HFpEF, such as the one in the case reported here, benefit from positive pressure therapies, when correctly performed, as there is a relationship between their use and the reduction of cases of decompensation; a randomized clinical trial showed that the in-hospital use of continuous positive pressure therapy for at least 3 hours per night was sufficient to increase the ejection fraction of patients with HFpEF and also reduce readmissions at 6 months, when compared to patients with the same diagnosis but who did not use the same therapy for a minimum of 3 hours32.

The diagnosis of OSA depends on the performance of complementary tests that evaluate parameters related to the functioning of the respiratory system during sleep. Currently, the gold standard testis type I polysomnography, with high specificity and sensitivity for detecting the pathology. In this examination, body position, electroencephalography, electro-oculography, electromyography, air flow, respiratory effort, pulse oximetry, electrocardiogram, and body posture are monitored33.

In the case described, a type IV polygraphy was used, which monitors a smaller number of parameters, such as high-resolution oximetry and air flow, however, given the characteristics of the patient and the result of the exam, the diagnosis was possible without the need to perform a more complex variation of the exam.

Once the diagnosis of obstructive sleep apnea is made, there are several resources with different levels of intervention that can be used and recommended for patients. Weight loss is usually the first treatment recommendation, since there is a positive relationship between BMI and the apnea-hyponea index, and weight reduction helps to reduce the symptoms caused by OSA30,34.

The therapy of choice for the treatment of OSA is, without a doubt, continuous positive pressure, performed through CPAP. This device, as its name suggests, produces intermittent pressure in the airway, preventing it from collapsing during the inspiratory act, which keeps the flow of air constant to the lungs. This therapy has been proven to reduce mortality from long-term complications of OSA, in addition to improving quality of life and sleep in patients with good treatment adherence30,32,34.

In the patient in the case above, a change in diet that would allow weight loss was advised and CPAP was initiated. The use of positive pressure therapy was very well tolerated and, in this case, brought very positive results, with improvement in the apnea-hypopnea indices and reduction in nocturnal awakenings, which brought quality of life to the patient.

However, in some cases, patients cannot tolerate the use of these devices, and other measures need to be used, such as mandibular advancement devices or, in more severe cases refractory to CPAP, surgeries that correct anatomical defects that trigger symptoms. In any case, the treatment is individualized and the anatomical characteristics and preferences of the patients in question must be taken into account32,34,35.

It can be concluded, based on the case reported and what is presente in the literature, that the diagnosis and institution of the treatment of obstructive sleep apnea in this patient not only provided an improvement in the quality of his sleep and, directly, his quality of life, but also avoided, in the medium and long term, his hospitalizations due to HFpEF decompensation; this reinforces the need to diagnose and treat OSA early, in an individualized and assertive way. In addition, it is necessary to be aware of obesity in patients like this, given the strong correlation between both pathologies and a lower chance of success in the treatment of OSA without a decrease in BMI.

CONFLICTS OF INTEREST

The authors declare that there are no possible conflicts of interest that could compromise the impartiality of this scientific work.

REFERENCES

1. World Health Organization. (2008). *WHO guidelines approved by the Guidelines Review Committee*. PubMed. Disponível em: https://www.ncbi.nlm.nih.gov/books/NBK132015/. Acesso em: 24 dez. 2024.

2. Mozaffarian, D., et al. (2016). Heart disease and stroke statistics-2016 update: A report from the American Heart Association. *Circulation, 133*(4), e38-e360.

3. Perk, J., et al. (2012). European guidelines on cardiovascular disease prevention in clinical practice (version 2012): The Fifth Joint Task Force of the European Society of Cardiology and other societies on cardiovascular disease prevention in clinical practice. *European Heart Journal*.

4. Ribeiro, A. L., et al. (2016). Cardiovascular health in Brazil: Trends and perspectives. *Circulation, 133*(4), 422-433.

5. Cortez, A. A., et al. (2005). Reabilitação cardiopulmonar e metabólica: Aspectos práticos e responsabilidades. *Revista Brasileira de Medicina do Esporte, 11*, 313-318.

6. Rohde, L. E. P., et al. (2018). Diretriz brasileira de insuficiência cardíaca crônica e aguda. *Arquivos Brasileiros de Cardiologia, 111*(3), 436-539.

7. Thomas, R. J., et al. (2010). AACVPR/ACCF/AHA 2010 update: Performance measures on cardiac rehabilitation for referral to cardiac rehabilitation/secondary prevention services. *Circulation, 122*(13), 1342-1350.

8. Dalal, H. M., Doherty, P., & Taylor, R. S. (2015). Cardiac rehabilitation. *BMJ, 351*, h5000.

9. Price, K. J., et al. (2016). A review of guidelines for cardiac rehabilitation exercise programmes: Is there an international consensus? *European Journal of Preventive Cardiology, 23*(16), 1715-1733.

10. World Health Organization. (2011). *From burden to “best buys”: Reducing the economic impact of non-communicable diseases in low- and middle-income countries*. Geneva: WHO.

11. Costa, L. E., et al. (2015). Potential underdiagnosis of obstructive sleep apnoea in the cardiology outpatient setting. *Heart, 101*(16), 1288-1292.

12. Mullington, J. M., et al. (2009). Cardiovascular, inflammatory, and metabolic consequences of sleep deprivation. *Progress in Cardiovascular Diseases, 51*(4), 294-302.

13. Stone, K. L., et al. (2016). Sleep disordered breathing and risk of stroke in older community-dwelling men. *Sleep, 39*(3), 531-540.

14. Xie, C., et al. (2017). Association of obstructive sleep apnoea with the risk of vascular outcomes and all-cause mortality: A meta-analysis. *BMJ Open, 7*(12), e013983.

15. Santos-Silva, R., et al. (2012). Sleep disorders and demand for medical services: Evidence from a population-based longitudinal study. *PLoS One, 7*(2), e30085.

16. Leger, D., et al. (2012). Impact of sleep apnea on economics. *Sleep Medicine Reviews, 16*(5), 455-462.

17. Dempsey, J. A., et al. (2010). Pathophysiology of sleep apnea. *Physiological Reviews, 90*(1), 47-112.

18. Chen, H., et al. (2016). Three-dimensional imaging of the upper airway anatomy in obstructive sleep apnea: A systematic review. *Sleep Medicine, 21*, 19-27.

19. Heiser, C., & Eckert, D. (2019). [Pathophysiology of obstructive sleep apnea]. *HNO, 67*(9), 654-662.

20. Benjafield, A. V., et al. (2019). Estimation of the global prevalence and burden of obstructive sleep apnoea: A literature-based analysis. *Lancet Respiratory Medicine, 7*(8), 687-698. https://doi.org/10.1016/S2213-2600(19)30198-5.

21. Tufik, S., et al. (2010). Obstructive sleep apnea syndrome in the São Paulo Epidemiologic Sleep Study. *Sleep Medicine, 11*(5), 441-446.

22. Blüher, M. (2019). Obesity: Global epidemiology and pathogenesis. *Nature Reviews Endocrinology*. Disponível em: https://pubmed.ncbi.nlm.nih.gov/30814686/. Acesso em: 24 dez. 2024.

23. Brasil. (2020). Pesquisa do IBGE mostra aumento da obesidade entre adultos. Governo do Brasil. Disponível em: https://www.gov.br/pt-br/noticias/saude-e-vigilancia-sanitaria/2020/10/pesquisa-do-ibge-mostra-aumento-da-obesidade-entre-adultos. Acesso em: 24 dez. 2024.

24. Heinzer, R., et al. (2015). Prevalence of sleep-disordered breathing in the general population: The HypnoLaus study. *Lancet Respiratory Medicine, 3*(4), 310-318.

25. European Heart Journal. (2012). European guidelines on cardiovascular disease prevention in clinical practice (version 2012). *European Heart Journal, 33*(13), 1635-1701. Disponível em: https://academic.oup.com/eurheartj/article/33/13/1635/488083. Acesso em: 24 dez. 2024.

26. Rundo, J. V. (2019). Obstructive sleep apnea basics. *Cleveland Clinic Journal of Medicine, 86*(9 Suppl 1), 2-9. Disponível em: https://www.ccjm.org/content/86/9_suppl_1/2.long. Acesso em: 24 dez. 2024.

27. Baguet, J. P., et al. (2012). Mechanisms of cardiac dysfunction in obstructive sleep apnea. *Nature Reviews Cardiology, 9*(12), 679-688.

28. Abdullah, A., et al. (2018). Relation of obstructive sleep apnea to risk of hospitalization in patients with heart failure and preserved ejection fraction from the National Inpatient Sample. *The American Journal of Cardiology, 122*(4), 612-615. Disponível em: https://pubmed.ncbi.nlm.nih.gov/30205888/. Acesso em: 9 set. 2024.

29. Bozkurt, B., et al. (2016). Contributory risk and management of comorbidities of hypertension, obesity, diabetes mellitus, hyperlipidemia, and metabolic syndrome in chronic heart failure: A scientific statement from the American Heart Association. *Circulation, 134*(23), 6 dez. 2016.

30. Kimoff, R. J. (2015). When to suspect sleep apnea and what to do about it. *Canadian Journal of Cardiology, 31*(7), 945-948.

31. Qaseem, A., et al. (2014). Diagnosis of obstructive sleep apnea in adults: A clinical practice guideline from the American College of Physicians. *Annals of Internal Medicine, 161*(3), 210.

32. Kasai, T., & Bradley, T. D. (2011). Obstructive sleep apnea and heart failure. *Journal of the American College of Cardiology, 57*(2), 119-127.

33. Khayat, R. N., et al. (2020). In-hospital management of sleep apnea during heart failure hospitalization: A randomized controlled trial. *Journal of Cardiac Failure*.

34. Soltis, M. E., & Spector, A. R. (2022). A concise review of polysomnography and obstructive sleep apnea for the neurophysiologist. *Journal of Clinical Neurophysiology, 40*(3), 191-197. Disponível em: https://pubmed.ncbi.nlm.nih.gov/36872497/. Acesso em: 14 set. 2024.

35. Qaseem, A. (2013). Management of obstructive sleep apnea in adults: A clinical practice guideline from the American College of Physicians. *Annals of Internal Medicine, 24 set.*