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Psychotic depression induced by Obstructive Sleep Apnoea Syndrome (OSAS): a case reported

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Obstructive Sleep Apnea Syndrome (OSAS) is a sleep disorder that is frequently associated with a large variety of conditions, such as hypertension, cardiovascular, neuropsychological or metabolic diseases. The most common and prominent symptoms of apnea is excessive diurnal drowsiness, as well asin addition to alterations of the memory and concentration, irritability, headache, and depression, among others. To date, no known studies have related OSAS with another type of more serious psychiatric disorder, such as psychotic symptoms. The case of a 51-year-old man who was diagnosed of SAOS after presenting psychotic and affective symptoms that did not respond to any medication is presentedreported. The treatment resulted in complete remission of the psychiatric symptoms mentioned.

Key Words:
Sleep apnoea, Psychotic symptoms, Affective disorder

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Depresión psicótica inducida por un Síndrome de Apnea Obstructiva del Sueño (SAOS): a propósito de un caso

El Síndrome de Apnea Obstructiva del Sueño (SAOS) es una alteración del sueño que frecuentemente se asocia a una gran diversidad de patologías, como la hipertensión arterial, enfermedades cardiovasculares, neuropsicológicas o metabólicas. La sintomatología más común y destacada de la apnea es la excesiva somnolencia diurna, así como alteraciones de la memoria y concentración, irritabilidad, cefaleas, y depresión, entre otras. Hasta la fecha no se conocen estudios que hayan relacionado el SAOS con otro tipo de alteraciones psiquiátricas más graves, como por ejemplo, la sintomatología psicótica. A continuación presentamos el caso de un varón de 51 años de edad que, tras presentar sintomatología psicótica y afectiva que no remitía con ningún fármaco, fue diagnosticado de SAOS, cuyo tratamiento logró la remisión completa de la sintomatología psiquiátrica.

Palabras clave:
Apnea del sueño, Síntomas psicóticos, Trastorno afectivo

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INTRODUCTION

Obstructive sleep apnea syndrome (OSAS) is a sleep disorder related with breathing, which is characterized by destructured sleep and defined as an episodic interruption of ventilation for 10 seconds or more. It is believed that the nasopharyngeal airway is smaller than normal in patients with OSAS and becomes wholly or partly obstructed during sleep. This obstruction makes breathing more labored and reduces oxygen saturation and originates sporadic arousals.

The treatment of choice for obstructive sleep apnea syndrome is the use of continuous positive airway pressure (CPAP). This device maintains pharyngeal patency by impeding upper airway collapse.

An association between OSAS and various disorders such as hypertension,[1] cardiovascular disease,[2, 3] neuropsychological, genitourinary and metabolic disorders,[4] etc. has been found in sound epidemiological studies for several years. However, the most common and noteworthy symptoms of apnea are excessive daytime drowsiness, impaired memory and concentration, irritability, headaches, and depression, among others.[3]

It is common for patients with OSAS to experience a strong tendency to fall asleep, a drowsiness that leaves the patient in a state of passivity and lack of initiative. In many cases, the patient is apathetic, with a loss of facial expression and slowed motor and cognitive function.

In the following, aA case of OSAS is reported that exhibited, in addition to some of the symptoms described above, and that arewhich have been reported in many

studies, another together with a type of symptom that has not been previously associated with this syndrome: that is, the psychotic symptoms.

CASE REPORT

The patient was a 51-year-old man from a family of eight children of low socioeconomic and educational status. Since his youth, he had always worked as a farmer and later began to work at a nursing home in janitorial duties. He worked at the nursing home for four years, but when his father became ill he had to stay up with him at night and go to work the next day. At this time That is when he began with a mutism condition began in which his wife observed that he was "listless, motionless, and she had to dress him, feed him and take him to the bathroom ...". He remained that way for a week and was unaware of the death of his father. The wife and husband sought help in a private psychiatry practice. Depressive disorder was diagnosed and treatment with amitriptyline and levomepromazine was begun. He only improved partially with treatment, but was still not participative. He started to have self-referential and prejudice-type delusions. He was diagnosed of depressive disorder with psychotic symptoms and treatment with haloperidol and biperiden was added.

When the patient was seen in the outpatient mental health clinic at the age of 47 years, he continued to be nervous, with feelings of inadequacy and guilt, but his psychotic symptoms had disappeared. He was diagnosed of depressive disorder and a new treatment with venlafaxine 150 mg/day and ketazolam 30 mg/day was started. He improved in subsequent visits, but his wife complained that he was still not very participative. Subsequently, he had several relapses coinciding with his return to work, with reactivation of his depressive symptoms and psychotic symptoms, delusions of reference and auditory hallucinations. He also made two serious suicide attempts. He remained on sick leave until he was granted permanent disability. Antipsychotic treatment with risperidone, up to 4mg/day, was added to antidepressant treatment with venlafaxine. The psychotic symptoms disappeared but the patient's mood was increasingly worse, with loss of initiative, an expressionless face and clumsiness, which is why a neuropsychological study was made: Benton Visual Retention Test. The results point to the possibility of a brain injury that affected both memory and visuo-constructive factors, but suggested that the possibility of below average intellectual quotient cannot be ruled out and may explain the results. In view of this report and the patient's cerebral risk factors, a cranial CT scan was performed and found to be normal, and the patient was referred to the outpatient neurology clinic to rule out frontal or frontotemporal dementia. In the neurological assessment, attention and calculation deficits were found, as well as delayed recall. A cranial MRI was normal and a cerebral SPECT found no pattern of dementia, so the neurology report indicated the presence of cognitive impairment associated with psychiatric disorders.

In a routine follow-up visit, the wife remarked that during the night her husband had always snored a lot, but now he "stopped snoring" as if he had "stopped breathing." She also said that he fell asleep during the day, while watching TV and at any time. Suspecting a possible obstructive sleep apnea syndrome (OSAS), the patient was referred to outpatient pulmonology, where the polygraphic study was consistent with moderate OSAS (AHI 32), which was corrected with CPAP at 9 cm H2O.

After a month of treatment with CPAP, the patient was asymptomatic, so the antipsychotic and antidepressant medication was gradually withdrawn. After one year in which he was completely asymptomatic and discharged from care at the mental health clinic.

DISCUSSION

As we mentioned above, many studies link OSAS with numerous complications that significantly impair the quality of life of these patients.[5, 6] They also have a high rate of psychiatric comorbidity, especially in relation to anxiety disorders and depression.[3, 7, 8]

However, no studies found in the current literature have reported severe psychotic symptoms induced by sleep apnea, as described in this clinical case. Numerous organic conditions that cause psychotic symptoms, both positive and negative, have been described. Among them are various types of dementia, neoplasms, vascular lesions, epilepsy and infections, etc. In our case, the appearance of psychotic and affective symptoms was subordinate to the sleep disorder and resolved completely when the apnea was treated.

It is difficult to explain the pathophysiological mechanism in this case. One possible explanation is linked to disorders resulting from sleep deprivation. Some studies[9] indicate that in situations of total sleep deprivation for several days, some individuals may experience paranoid symptoms. In addition, subjects with no history of mental illness may experience perceptual distortions and even hallucinations in situations of sleep deprivation.[10] Although the subjects recognize that the distortions and hallucinations are not real, this illustrates the effects that sleep deprivation can have on brain function.[11]

In psychiatric patients, total sleep deprivation can trigger manic episodes in bipolar patients.[12] On the other hand, other authors[13] have reported a relation between partial deprivation of certain stages of sleep and changes in the symptoms of patients with severe depression or psychosis, resulting in a seemingly paradoxical phenomenon: symptoms

diminish after the suppression of paradoxical sleep (or REM sleep).

The fact that sleep deprivation can alter mood and induce alterations in sensory perception indicates the importance that sleep can have on all these aspects. At present, hardly any studies have attempted to explain the mechanisms involved in causing these phenomena, thus making it necessary to delve into future lines of research to explain this question.

REFERENCES

1. Kario K. Obstructive sleep apnea syndrome and hypertension: ambulatory blood pressure. Hypertens Res. 2009;32(6):428-32.
2. Jain V. Clinical perspective of obstructive sleep apnea-induced cardiovascular complications. Antioxid Redox Signal. 2007;9(6):701-10.
3. Culebras A. Repercusión de la apnea del sueño en neurología. Rev Neurol. 2003;36(1):67-70.
4. Gibson GJ. Obstructive sleep apnoea syndrome: underestimated and undertreated. Brit Med Bull. 2004;72:49-64.
5. Jenkinson C, Strandling J, Petersen S. Comparison of three measures of quality of life outcome in the evaluation of continuous positive airways pressure therapy for sleep apnoea. J Sleep Res. 1997;6:199-204.
6. Flemons WW, Reimer MA. Development of disease-specific helth-related quality of life questionnaire foe sleep apnoea. Am J Respir Crit Care Med. 1998;158:494-503.
7. Saunamäki T, Jehkonen M. Depression and anxiety in obstructive sleep apnea syndrome: a review. Acta Neurol Scand. 2007;116(5):277-88.
8. Douglas NJ, Engleman HM. Effects of CPAP on vigilance and related functions in patients with the sleep apnea/hypopnea syndrome. Sleep. 2000;23(4):S147-9.
9. Freeman D, Brugha T, Meltzer H, Jenkins R, Stahl D, Bebbington P. Persecutory ideation and insomnia: findings from the second British National Survey of Psychiatric Morbidity. J Psychiatr Res. 2010;44(15):1021-6.
10. Ohayon MM. Prevalence of hallucinations and their pathological associations in the general population. Psychiatry Res. 2000;97(2-3):153-64.
11. El-Ad B, Lavie P. Effect of sleep apnea on cognition and mood. Int Rev Psychiatry. 2005;17(4):277-82.
12. Plante DT, Winkelman JW. Sleep disturbance in bipolar disorder: therapeutic implications. Am J Psychiatry. 2008;165(7):830-43.
13. Carlson NR. Fisiología de la conducta. Barcelona: Ariel, 2005.