Case report

OBSTRUCTIVE SLEEP APNEA AS A RISK FACTOR FOR WAKE-UP STROKE: A CASE REPORT

Apnéia obstrutiva do sono como fator de risco para *Wake-Up Stroke*: Relato de Caso

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ABSTRACT

Introduction: Wake-up Stroke (WUS) is characterized by the onset of symptoms during sleep, with an unknown exact time of onset, making thrombolytic treatment challenging. Obstructive Sleep Apnea (OSA) is considered an independent risk factor for stroke. This case report discusses a WUS episode in a patient with a history of sleep disorder.
Methods: A retrospective, observational, and analytical study in the form of a case report, using information from the patient and physical medical records, conducted at tertiary hospital in southwest Paraná, Brazil.
Case Description: A 74-year-old female patient with grade I obesity, systemic arterial hypertension, atrial flutter, and obstructive sleep disorder was admitted after waking up with a neurological deficit, having gone to bed asymptomatic. She presented with right-sided hemiplegia, aphasia, and confusion. Upon admission, a brain CT scan showed no evidence of ischemic lesions. The patient was managed clinically, without thrombolytic administration. After seven days, a new CT scan revealed hypodense lesions in the lenticulocapsular region and the left frontal lobe. Four weeks after the episode, there was significant improvement in deficits, with mild right-sided motor weakness and persistence of anomic aphasia.
Conclusion: Recognizing OSA as a risk factor for WUS reinforces the need for early diagnosis and proper management of sleep-related breathing disorders in patients at risk of cerebrovascular events. Extending the thrombolysis window, based on advanced neuroimaging, may redefine the management of WUS and reduce the global impact of stroke.

Keywords: Stroke; Obstructive Sleep Apnea; Wake-up Stroke; Risk Factors; Cerebral Ischemia; Sleep Disorders; Primary Prevention.

RESUMO

Introdução: O Wake-up Stroke (WUS), ou Acidente Vascular Cerebral (AVC) de despertar, caracteriza-se pelo surgimento dos sintomas durante o sono, com hora exata de início desconhecida, dificultando o tratamento trombolítico. A Apneia Obstrutiva do Sono (AOS) é considerada um fator de risco independente para AVC. Este relato aborda um episódio de WUS em paciente com história prévia de distúrbio do sono.
Métodos: Estudo retrospectivo, observacional e analítico, do tipo relato de caso, utilizando informações com a paciente e em prontuário físico, realizado em um hospital terciário no sudoeste do Paraná, Brasil.
Descrição do caso: Paciente feminina, 74 anos, com obesidade grau I, hipertensão arterial sistêmica, flutter atrial e distúrbio obstrutivo do sono, admitida após despertar com déficit neurológico, tendo ido dormir assintomática. Apresentava hemiplegia à direita, afasia e confusão. Na admissão, foi realizada uma Tomografia Computadorizada de Crânio, sem evidências de lesão isquêmica. A paciente foi manejada clinicamente, sem administração de trombolíticos. Após sete dias, uma nova tomografia revelou lesões hipodensas na região lenticulocapsular e no lobo frontal esquerdo. Quatro semanas após o episódio, houve significativa melhora dos déficits, com leve comprometimento da força motora à direita e persistência de afasia anômica.
Conclusão: O reconhecimento da AOS como fator de risco para WUS reforça a necessidade do diagnóstico precoce e manejo adequado de distúrbios respiratórios do sono em pacientes com risco de eventos cerebrovasculares. A ampliação da janela de trombólise, com base em neuroimagem avançada, pode redefinir o manejo do WUS e reduzir o impacto global do AVC.

Palavras chaves: Acidente Vascular Cerebral; Apneia Obstrutiva do Sono; Wake Up Stroke; Fatores de Risco; Isquemia Cerebral; Distúrbios do Sono; Prevenção Primária

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INTRODUCTION

Wake-up Stroke (WUS) refers to a stroke in which symptoms are noticed upon waking, making it impossible to determine the exact moment of onset[1]. It represents approximately 20% of acute ischemic strokes (AIS) and poses therapeutic challenges, as it prevents the safe administration of time-based intravenous thrombolysis[1,2]. Among the risk factors, Obstructive Sleep Apnea (OSA) has gained attention due to its impact on cerebrovascular pathophysiology[3].

OSA is characterized by recurrent episodes of partial or complete upper airway collapse during sleep, resulting in intermittent hypoxia, blood pressure fluctuations, and endothelial dysfunction. These mechanisms promote conditions that increase cardiovascular risk and may predispose individuals to WUS[4,5]. Recognizing OSA as a risk factor for WUS highlights the need for early diagnostic approaches and personalized preventive strategies.

This case report illustrates the correlation between these conditions, emphasizing the importance of screening for sleep-disordered breathing and the potential impact of targeted therapies on secondary stroke prevention. The disclosure of patient information was approved by the local Research Ethics Committee under CAAE No. 86398225.9.0000.0107.

CASE DESCRIPTION

A 74-year-old female patient with a history of hypertension, atrial flutter, and heart failure, under regular treatment, reported symptoms consistent with OSA since early adulthood, including fatigue, daytime sleepiness, and progressive snoring, without prior treatment. At 3:00 AM, she was found with right-sided hemiplegia, aphasia, and confusion, with facial asymmetry and loss of sphincter control, and was immediately taken to the emergency unit. The patient reported going to sleep asymptomatic and being seen without changes approximately two hours before the event.

Upon hospital admission, a cranial computed tomography (CT) scan showed no acute lesions (Figure 1A), and clinical management was initiated without reperfusion therapy using thrombolytics. After seven days, a follow-up CT scan revealed a lesion suggestive of ischemic insult (Figure 1B-C).

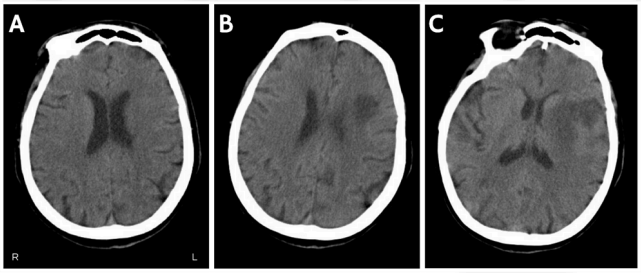

Figure 1. Axial Cranial Computed Tomography (CT) scan showing the evolution of an ischemic insult area. **A:** Initial CT scan at admission, with no evidence of ischemic injury. **B** and **C:** Follow-up CT scans seven days later, revealing a poorly defined hypodense lesion in the external lenticulocapsular region and the left frontal lobe.

Upon hospital discharge, the patient showed progressive motor strength recovery, with significant improvement in functional independence and communication.

Five months later, at an outpatient follow-up, she was in good general condition, walking unassisted, with mild disability (Rankin Scale 2), persisting anomic aphasia, and mild dysarthria. On physical examination, she weighed 79 kg, was 158 cm tall, and had a BMI of 31.65 kg/m², with no cardiovascular abnormalities. Motor strength and range of motion were reduced in the right upper and lower limbs (proximal 4/5, distal 3/5), preserved on the left. Anthropometric measurements showed waist and abdominal circumferences of 102 cm and 117 cm, respectively. The Mallampati score was 3, with an oral opening of 3 cm and a neck circumference of 39 cm.

After the stroke, sleep - related symptoms worsened, including frequent awakenings, respiratory pauses, and excessive daytime sleepiness, as assessed by the Epworth Sleepiness Scale (12 points) and STOP-BANG Questionnaire (6 points). Polysomnography (Figure 2) revealed a severe apnea-hypopnea index (52.3 episodes/hour), with significant oxyhemoglobin desaturation (54%), occurring approximately 331 times during the evaluation. Based on these findings, CPAP titration was recommended for managing severe apnea.

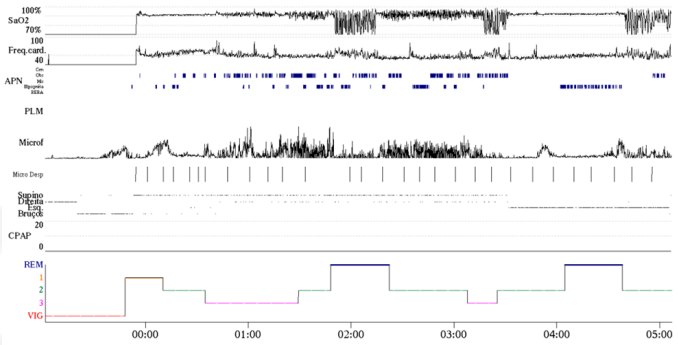

Figure 2. Type 1 Polysomnography

DISCUSSION

The case presents a patient with classic risk factors for ischemic stroke. However, the presence of a chronic sleep disorder is an aggravating factor, as it is associated with hemodynamic and metabolic alterations that predispose to cerebrovascular events. Intermittent hypoxia and sympathetic hyperactivity promote blood pressure fluctuations, systemic inflammation, and hypercoagulability, favoring the development of WUS[3,4]. Additionally, sleep fragmentation and autonomic oscillation during the sleep-wake cycle can increase the risk of arrhythmias, heightening the likelihood of thromboembolic events[5].

Obstructive sleep apnea (OSA), frequently underdiagnosed, is an independent risk factor for ischemic stroke and WUS[4,6]. Intermittent hypoxia in OSA disrupts cerebral autoregulation, increasing the risk of thrombosis and endothelial dysfunction[5]. Patients with severe OSA

exhibit a higher frequency of WUS due to a combination of pathophysiological factors and increased predisposition to cardioembolic events during sleep, when symptoms often go unnoticed[7]. Heart failure, closely linked to OSA, is also a risk factor. The "fluid-shift" mechanism – the nocturnal redistribution of fluids from the lower extremities to the cervical region – worsens upper airway obstruction, exacerbating apnea and cardiovascular overload[8].

The American Heart Association (AHA) guidelines recommend screening and treatment of OSA in patients with a history of stroke or transient ischemic attack due to the strong correlation between OSA and stroke risk[9,10]. Nevertheless, evidence is still lacking to justify widespread early intervention. Respiratory disorder assessment questionnaires are useful tools in primary care and outpatient settings for OSA screening. The STOP-BANG questionnaire (an acronym for Snoring, Tiredness, Observed apnea, high blood Pressure, Body mass index, Age, Neck circumference, and Gender)[11,12] and the Berlin Questionnaire[13] are validated instruments that practically and objectively assess predisposition to OSA.

Definitive diagnosis of OSA is made through polysomnography, which evaluates disorder severity based on the apnea-hypopnea index, oxygen desaturation, and apnea episode distribution across sleep phases. Indices ≥ 5 events/hour indicate OSA, while values above 30/hour characterize severe OSA[14]. In the present case, the examination indicated severe apnea, reinforcing the need for CPAP titration. These findings highlight the importance of objective sleep assessment in treating and preventing cerebrovascular complications associated with untreated OSA.

CONCLUSION

This case highlights the clinical relevance of understanding the causal relationship between OSA and stroke in the context of WUS. Given the global impact of cerebrovascular disorders and the role of OSA as a risk factor before and after stroke, efforts should focus on:¹ defining screening criteria;² determining which post-stroke patients should receive CPAP as secondary prevention;³ further exploring the relationship between OSA, stroke, and WUS.

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