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Sleep disorders in patients with acquired brain damage, associated factors and their impact on functionality

Desórdenes de sueño en pacientes con daño cerebral adquirido, factores asociados y su impacto sobre la funcionalidad

Paul Carrillo-Mora,* Beatriz Sidonio-Aguayo,‡ Juan Francisco Márquez-Vázquez,‡ Susana Martín del Campo-Arias,§ Marisela Carrillo-Santos,§ Claudia Hernández-Arenas,§ Nelly Giselle Cervera-Delgadillo,¶ María Alejandra Samudio-Cruz*

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* División de Neurociencias, Instituto Nacional de Rehabilitación «Luis Guillermo Ibarra Ibarra». Mexico City, Mexico.
‡ Centro Estatal de Rehabilitación y Educación Especial de Toluca, Instituto de Salud del Estado de México (ISEM), Mexico.
§ Subdirección de Medicina de Rehabilitación, Instituto Nacional de Rehabilitación «Luis Guillermo Ibarra Ibarra», Mexico City, Mexico.
¶ Facultad de Medicina de la Universidad Nacional Autónoma de México, Mexico City, Mexico.

Abstract

Objective: To identify the prevalence of sleep disorders in chronic post-stroke and post-traumatic brain injury (TBI) patients, to determine the risk factors and their impact on functionality in activities of daily life. **Material and methods:** Cross-sectional clinical study that included adults ≥ 18 years with a history of acquired brain damage (post-stroke or post-TBI). Sociodemographic data and clinical history were obtained, and the following instruments were applied: Pittsburgh Sleep Quality Index, the Epworth Sleepiness Scale, the Berlin Questionnaire© Sleep Apnea, the Barthel's functionality index, the Hamilton Depression Rating Scale and the Beck Anxiety Inventory. **Results:** We included 116 patients, 91 post-stroke (78.4%) and 25 post-TBI (21.6%), mean age was 56.58 years (SD = 19.37). In post-stroke patients, the following risk factors were identified: diabetes (OR = 3.01; 95% CI = 1.13-8.01 for poor sleep quality), multiple comorbidities (OR = 3.78; 95% CI = 1.04-13.67 for quality of sleep), depression (OR = 2.46; 95% CI = 2.46-25.80 for apnea; OR = 7.94; 95% CI = 1.25-10.82 for insomnia) and anxiety (OR = 17.84; 95% CI = 2.28-139.64 for insomnia). In post-TBI patients, the following were identified as risk factors: overweight/obesity (OR = 11.25; 95% CI = 1.64-76.84 for poor sleep quality) and coma (OR = 2.33; 95% CI = 1.42-3.82 for sleepiness). The risk factor for functional loss in post-stroke is apnea (OR = 2.63; 95% CI = 1.05-6.54) and in post-TBI the poor quality of sleep (OR = 8.25; 95% CI = 1.15-50.03). **Conclusion:** Post-stroke and post-TBI patients have a high prevalence of sleep disorders and its comorbidities increase the risk of chronic sleep disorders and functional loss.

Resumen

Objetivo: *Identificar la prevalencia de trastornos en el sueño en pacientes post-stroke y post-TCE, determinar los factores de riesgo y su impacto en la funcionalidad en actividades de la vida diaria.* ***Material y métodos:*** *Estudio clínico transversal que incluyó adultos ≥ de 18 años con antecedente de daño cerebral adquirido (post-stroke o postraumatismo craneoencefálico). Se obtuvieron datos sociodemográficos y antecedentes clínicos, y se aplicaron los siguientes instrumentos: Pittsburgh Sleep Quality Index, the Epworth Sleepiness Scale, the Berlin questionnaire sleep apnea, the Barthel's*

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Correspondence:
María Alejandra Samudio Cruz
E-mail: psic.alejandra.samudio@gmail.com

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functionality index, the Hamilton Depression Rating Scale and the Beck Anxiety Inventory. ***Resultados:*** *Se incluyeron 116 pacientes, 91 post-stroke (78.4%) y 25 post-TBI (21.6%), media de edad fue 56.58 años (DE = 19.37). En pacientes post-stroke se identificaron como factores de riesgo: diabetes (OR = 3.01; IC 95% = 1.13-8.01 para mala calidad de sueño), múltiples comorbilidades (OR = 3.78; IC 95% = 1.04-13.67 para calidad de sueño), depresión (OR = 2.46; IC 95% = 2.46-25.80 para apnea; OR = 7.94; IC 95% = 1.25-10.82 para insomnio) y ansiedad (OR = 17.84; IC 95% = 2.28-139.64 para insomnio). En el pacientes post-TBI, se identificaron como factores de riesgo: sobrepeso/obesidad (OR = 11.25; IC 95% = 1.64-76.84 para pobre calidad de sueño) y antecedente de coma (OR = 2.33; IC 95% = 1.42-3.82 para somnolencia). El factor de riesgo para pérdida funcional en post-stroke es la apnea (OR = 2.63; IC 95% = 1.05-6.54) y en post-TBI la mala calidad de sueño (OR = 8.25; IC 95% = 1.15-50.03).* ***Conclusión:*** *Los pacientes post-stroke y post-TBI presentan comorbilidades que incrementan el riesgo de presentar trastornos del sueño crónicos y pérdida funcional.*

INTRODUCTION

Sleep disorders are common consequences of acquired brain damage. In patients with a history of cerebrovascular disease (post-stroke), a prevalence of 1.1 to 27% of sleepiness, 50-70% of apnea[1] and 20-56% of insomnia[2] have been identified previously. In patients with a history of traumatic brain injury (post-TBI), sleep disturbances may occur as part of the TBI spectrum or may occur after the injury.[3] The prevalence of sleep disturbances is reported in 30-70%,[3] 41.7% have sleepiness or fatigue (41.7%), 36% apnea, 30% poor quality of sleep and insomnia.[4]

The causes of sleep disorders in patients with acquired brain damage include a complex interrelation between pathophysiological (structural, electrical or neurochemical) processes, variables associated with the injury (severity, site and extent of injury, loss of consciousness, time of evolution), previous sleep habits and alterations, psychological factors (mood changes, neuropsychiatric sequelae), environmental factors (noise, light, invasive treatments, adverse effect of medications), another type of injury sequelae (pain, immobility), social factors (type of care, family role), among others.[2-5]

In addition to altering daytime energy levels, sleep disorders can exacerbate other conditions such as cognitive deficits (predominantly in attention and memory), pain, fatigue, mood disturbances, and contribute to poor rehabilitation achievements, which can have an impact on functional recovery and lead to low quality of life.[2,5,6]

For the diagnosis of sleep disorders, polysomnography (PSG) has been considered the gold standard, providing objective and quantitative information of physiological indicators in a transversal manner, however, its complexity, low availability and high costs make it difficult for it to be widely available in all patients. Therefore, an alternative method to study these disorders is the application of validated scales and questionnaires in which the patient describes different aspects related to the quality and duration of sleep in relation to a longer period of time. These being easily administered and low-cost, the scales are largely used as a resource to improve the initial characterization of patients and identify the parameters to be evaluated in a PSG.[7,8]

Although there are numerous studies that have described the sequelae in sleep after acquired brain damage,[1-4,9] little is known about the chronicity of this type of manifestations and the differential characteristics between post-stroke and post-TBI patients, which may be interesting considering the high prevalence of this type of lesion and its impact on functionality.

The objective of this study was to identify the prevalence of sleep disturbances in post-stroke and post-TBI patients and to determine the risk factors for these disturbances. As a secondary objective, the impact of sleep disorders on functionality in activities of daily life was analyzed.

MATERIAL AND METHODS

Study design and participants. Cross-sectional clinical study conducted in a third-level hospital in Mexico City that included men and women over the age of 18 who met the following inclusion criteria: 1) history of acquired brain damage (first event of ischemic or hemorrhagic stroke or presence of TBI, corroborated by neuroimaging study), 2) absence of neurological, psychiatric or previously diagnosed sleep disturbances (obstructive apnea, narcolepsy, chronic insomnia, hypersomnia, parasomnias), 3) evolution time of at least one month from brain injury, 4) absence of severe cognitive or physical impairment that would prevent the application of the assessment instruments, 5) absence of additional uncontrolled

or untreated medical conditions affecting the sleep, mood or cognition.

Instruments. After having signed the informed consent, a semi-structured interview was conducted in which information about sociodemographic variables and clinical history was obtained. For the evaluation of sleep disorders, functional and mental state the following instruments were applied: Pittsburgh Sleep Quality Index (PSQI ≥ 6), the Epworth Sleepiness Scale (ESS ≥ 10), the Berlin questionnaire sleep apnea (BQ ≥ 3), the Barthel's functionality index (≥ 80), the Hamilton Depression Rating Scale (HDRS ≥ 8) and the Beck Anxiety Inventory (BAI, 1 mild anxiety). Evaluations were conducted by qualified personnel in individual cubicles with appropriate conditions.

Statistical methods. Descriptive statistics were used to analyze the characteristics of the sample. The Shapiro-Wilk test was used to determine the normal distribution of variables. Student t and Mann-Whitney U tests were used to compare means between groups and χ^2 to compare proportions. Multiple linear regressions were performed to identify variables associated with the presence of sleep disturbances and variables associated with functional loss and odds ratio (OR) were determined with a 95% confidence interval (CI). All analyses were performed with the SPSS 21 statistical program. To establish significant differences and correlations, a value of $p \leq 0.05$ was considered.

RESULTS

The sample consisted of 116 patients, 91 post-stroke (78.4%) and 25 post-TBI (21.6%), the mean age was 56.58 years (SD = 19.37), with the post-TBI group being younger in a statistically significant way (37.64, SD = 19.80 with respect to post-stroke: 61.79, SD = 15.74, p 0.001), 56.8% of the total sample were men, 50.5% of the post-stroke group and 80% of the post-TBI group. In both groups there was predominance of right manual laterality (96.7% in post-stroke and 100% in post-TBI). The average evolution time was 17.9 months, the post-TBI group had a longer evolution time (27.75, SD = 39.27) than the post-VC group (15.08, SD = 18.49), however, this was not statistically significant. In post-stroke patients, the most prevalent type of lesion was ischemic (64.8% with respect to 34.1% hemorrhagic), predominantly left (50.8%). In post-TBI patients, 73.9% had a right hemisphere lesion, 84% had a history of coma, and 60% required surgical treatment *(Table 1)*.

Regarding the clinical variables, the post-stroke group presented a greater number of comorbidities (3.31 with respect to 2.20, p = 0.005), a higher prevalence of hypertension (65.9% with respect to 4%, p = 0.001) and diabetes (34.1% with respect to 8%, p = 0.011) with respect to the post-stroke, while the post-TBI group had a higher prevalence of alcoholism compared to the post-stroke group (56% compared to 15.4%, p = 0.001) *(Table 1)*.

Table 1 shows the clinical scales used to assess sleep disorders (quality of sleep, sleepiness and apnea), mood (depression and anxiety), as well as functionality in both groups of patients. Only statistically significant differences were identified between groups in the symptoms of apnea, with the post-stroke group having the highest score (3.21, SD = 1.61 with respect to 2.12, SD = 1.39 in post-TBI, p = 0.002).

Prevalence of sleep disturbances in post-stroke and post-TBI patients. The post-stroke group had a higher prevalence of poor sleep quality (61.3% with respect to post-TBI: 52%), risk of apnea (63.4% with respect to post-TBI: 44%) and insomnia (26.9% with respect to post-TBI: 12%). The post-TBI group had a higher prevalence of sleepiness (52% with respect to post-stroke: 36.6%). None of the differences were statistically significant *(Table 1 and Figure 1)*.

Risk factors for sleep disturbances in post-stroke and post-TBI patients. In post-stroke patients, the presence of diabetes (OR = 3.01; 95% CI = 1.13-8.01) and multiple comorbidities (OR = 3.78; 95% CI = 1.04-13.67) were identified as risk factors for poor sleep. For sleepiness, only the presence of diabetes was identified as a risk factor (OR = 3.98; 95% CI = 1.59-9.91). For apnea, the presence of diabetes (OR = 4.57; 95% CI = 1.55-13.45) and depression (OR = 7.97; 95% CI = 2.46-25.80) were observed as risk factors. Depression and anxiety were the risk factors identified for insomnia (OR = 3.68; 95% CI = 1.25-10.82 and OR = 17.84; 95% CI = 2.28-139.64, respectively).

In the post-TBI group, overweight or obesity were identified as risk factors for poor sleep quality (OR = 11.25; 95% CI = 1.64-76.84), while the history of coma was identified as a risk factor for sleepiness (OR = 2.33; 95% CI = 1.42-3.82) *(Table 2)*.

Effect of sleep disorders on functionality. In the post-stroke group, apnea was the only sleep disturbance that represents a risk factor to generate

Table 1: General characteristics of the sample.

Variable	Total	Post-stroke	Post-TBI	p
Sociodemographic variables				
n	116 (100)	91 (78.4)	25 (21.6)	
Age	56.58 (19.37)	61.79 (15.74)	37.64 (19.8)	0.001*
Sex (men)	65 (56.8)	46 (50.5)	20 (80)	0.008*
Laterality	73 (97.3)	59 (96.7)	25 (100)	0.492
Variables associated with the lesion				
Evolution time	17.87 (24.94)	15.08 (18.49)	27.75 (39.27)	0.142
Stroke type				
Ischemic	—	57 (64.8)	—	—
Hemorrhagic	—	30 (34.1)	—	—
Injured hemisphere (right)	50 (50)	33 (42.9)	17 (73.9)	0.009*
History of coma	21 (84)	—	21 (84)	—
Surgical treatment	—	—	12 (60)	—
Clinical variables				
Total comorbidities	3.03 (1.75)	3.31 (1.73)	2.20 (1.47)	0.005*
Stroke preious	9 (11.8)	9 (12.5)	0 (0)	0.102
Hypertension	61 (52.6)	60 (65.9)	1 (4)	0.001*
Diabetes	33 (28.4)	31 (34.1)	2 (8)	0.011*
Dyslipidemia	30 (25.9)	25 (27.5)	5 (20)	0.450
Depression	19 (16.4)	18 (19.8)	1 (4)	0.059
Heart disease	15 (12.9)	13 (14.3)	2 (8)	0.407
BMI	24.89 (3.92)	24.77 (3.85)	25.35 (4.19)	0.505
Alcoholism	28 (24.1)	14 (15.4)	14 (56)	0.001
Smoking	30 (25.9)	23 (25.3)	7 (28)	0.783
Clinical scales				
PSQI	7.88 (4.91)	8.18 (5.16)	7.08 (3.91)	0.361
ESS	8.40 (6.21)	8.21 (6.45)	8.6 (5.58)	0.618
BQ	2.98 (1.61)	3.21 (1.61)	2.12 (1.39)	0.002*
Hamilton	8.69 (7.86)	8.92 (8.04)	8 (7.48)	0.721
Beck	4.80 (5.59)	5.06 (5.90)	3.92 (4.51)	0.448
Barthel	57.80 (38.68)	59.41 (39.20)	51.82 (36.84)	0.329
Prevalence of sleep				
Por quality of sleep	70 (59.3)	57 (61.3)	13 (52)	0.401
Dayitme sleepiness	47 (39.8)	34 (36.6)	13 (52)	0.161
Apnea risk	70 (59.3)	59 (63.4)	11 (44)	0.079
Insomnia	28 (23.70)	25 (26.9)	3 (12)	0.120

TBI = traumatic brain injury; BMI = Body mass index; PSQI = Pittsburgh Sleep Quality Index; ESS = Epworth Sleepiness Scale; BQ = Berlin questionnaire sleep apnea. * Statistically significant

functional dependence (OR= 2.63; 95% CI = 1.05-6.54), however, risk factors are also identified as age over 60 years (OR = 3.75; 95% CI = 1.50-9.36) and the presence of depression (OR = 3.60; 95% CI = 1.07-12.09).

In the post-TBI group, the only risk factor for functional dependence was poor sleep quality (OR = 8.25; 95% CI = 1.15-59.03) *(Table 3)*.

DISCUSSION

Acquired brain damage is a major cause of disability that generates several sequelae, with sleep disorders being one of the most frequent. Given the effect of sleep disorders in other conditions that can affect the patient's recovery, it is necessary to detect, characterize and consider this type of

alterations in the management of patients with acquired brain damage.

The present study found a high prevalence of sleep disturbances, 61.3% of post-stroke patients and 52% of post-TBI patients reported poor sleep quality. In post-stroke patients, there was a high prevalence of risk of apnea (63.4%) and insomnia (26.9%). These results agree with what was described by various authors,[1,2,9,10] who have described 50-70% prevalence of apnea and 20-56% insomnia, however, the prevalence of daytime sleepiness in the present study at 17.9 months (36.6%) was slightly higher than that reported by Hermann and Bassetti[1], who reported 27% to 21 months; and does not agree with what was identified by Winward et al.[10] who described a significant reduction in sleepiness six months after injury in patients with minor stroke. The increased severity of the patients included in this study is likely to explain the permanence of daytime sleepiness several months after injury.

Risk factors for sleep disturbances in post-stroke patients. According to our results, the presence of diabetes mellitus and multiple comorbidities have an impact on the quality of sleep of post-stroke patients. It has been described that the quality of sleep may be affected by the medical conditions of patients, so the presence of various comorbidities, including diabetes, can exacerbate poor sleep quality. Specifically, diabetes has been associated with numerous sleep disorders, the most prevalent being apnea, hypersomnia and insomnia. This has been explained by both the effects of diabetes on the central control of breathing that can trigger episodes of apnea and by glycemic imbalances during the day that can exacerbate hypersomnia that can generate effects in night sleep generating insomnia.[11]

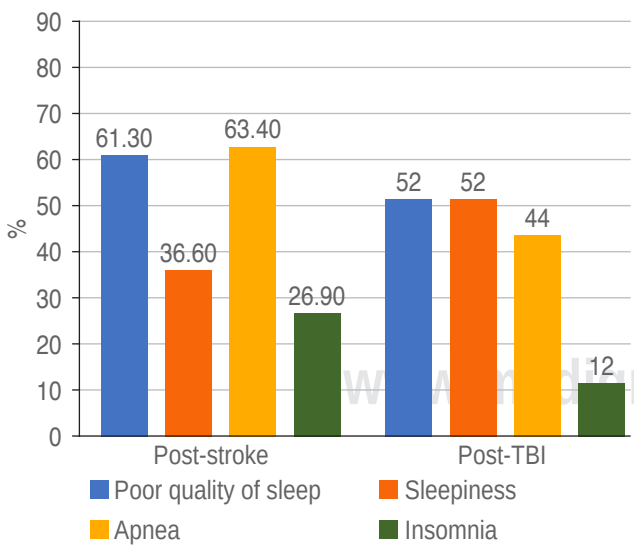

Figure 1: Prevalence of sleep disturbances between post-stroke and post-TBI patients.

Table 2: Risk factors of sleep disturbances in post-stroke and post-TBI patients.

Variables	OR (95% CI)
Post-stroke	
Sleep quality	
Diabetes	3.01 (1.13-8.01)
More than one comorbidity	3.78 (1.04-13.67)
Sleepiness	
Diabetes	3.98 (1.59-9.91)
Apnea	
Diabetes	4.57 (1.55-13.45)
Depression (self report)	2.87 (1.19-6.89)
Depression (Hamilton scale)	7.97 (2.46-25.80)
Insomnia	
Depression	3.68 (1.25-10.82)
Depression (Hamilton scale)	7.97 (2.46-25.80)
Anxiety (Beck scale)	17.84 (2.28-139.64)
Post-TBI	
Sleep quality	
Overweight/obesity	11.25 (1.64-76.84)
Sleepiness	
Coma	2.33 (1.42-3.82)

OR = odds ratio; CI = confidence interval; TBI = traumatic brain injury.

Various studies have shown that patients with diabetes have worse quality of night sleep, so they have excessive daytime sleepiness. Some of the causes that explain sleep disorders in patients with diabetes are the presence of pain due to the presence of peripheral neuropathy, or nocturia generated by poor glycemic control. In addition, a specific abnormal breathing pattern has been identified in patients with diabetes, which is still confusing.[12-14]

In our study, the presence of diabetes was associated with the presence of apnea. Several studies have described that the presence of diabetes affects central respiratory control and that this can promote the presence of apnea, being observed in some studies

prevalence of 27% in diabetes compared to 15.6% in non-diabetic, and in others, presence of apnea in more than half of patients with type 2 diabetes. It should be emphasized that these variables are associated regardless of age and degree of obesity.[11]

Regarding the relationship between depression and sleep apnea, several studies have shown the interrelation between these variables, so that the presence of sleep disturbances such as apnea have repercussions on mental health and, on the other hand, the presence of mood disturbances can contribute to the exacerbation of sleep disturbances. Given the lack of clarity of the pathophysiological mechanisms that explain this relationship, it has been proposed that both sleep disorders and mental health disorders could be a consequence of the same neurobiological process.[14]

There is a great deal of evidence to support the interrelationship between mood disturbances (such as anxiety and depression) and their relationship to sleep. Some studies have shown that genetic factors related to the etiology of insomnia overlap with those related to depression and anxiety, however, there are various biological mechanisms, psychosocial and environmental factors involved in the presence of these symptoms.[15]

Risk factors for sleep disturbances in post-TBI patients. Obesity and overweight have been considered one of the factors of greater risk to present alterations in sleep through various pathogenetic mechanisms, However, it has also been described those subjects with sleep disturbances are also more likely to be overweight and obese.[16] Some studies have identified that a mechanism that links obesity to sleep disorders is the quality of the diet, because of nutrients acting on inflammation or hormonal responses involved in the mechanisms of hungersatiety, energy metabolism and circadian rhythm.[17] On the other hand, it has been described that in obesity, the cephalic displacement of the diaphragm by abdominal fat affects lung volumes, producing a restrictive pattern characterized by the reduction of functional residual capacity and expiratory reserve volume that affects the quality and quantity of sleep.[18]

Although there is no direct link between post-TBI coma and daytime sleepiness, it has been reported that in general almost half of people with a history of severe TBI have a pathological level of sleepiness with latency times of less than 10 minutes. The association between history of coma and the presence of sleepiness is likely to be associated with direct injury of histaminergic tuberomammillary alert neurons, which are reduced by about 40% after severe traumatic brain injury.[19-21]

Table 3: Risk factors of functional dependency in post-stroke and post-TBI.

Variables	OR (95% CI)
Post-stroke	
Age over 60 years	3.75 (1.50-9.36)
Depression	3.60 (1.07-12.09)
Apnea	2.63 (1.05-6.54)
Post-TBI	
Poor quality of sleep	8.25 (1.15-59.03)

OR = odds ratio; CI = confidence interval; TBI = traumatic brain injury.

Effect of sleep disorders on functionality. In this study, it was identified that in post-stroke patients, the age over 60 years, depressive symptoms and apnea, are risk factors to present a lower functionality, while the poor quality of sleep affects the functional capacity in post-TBI patients. These findings correspond to what has been described in various studies, in which it has been considered that sleep disorders impact the rehabilitation capacity of patients with acquired brain injury and functional recovery.[22]

Limitations of the study. It is recognized that the evaluation of sleep disorders was carried out clinically only with specific and standardized scales for these purposes, it was not possible to corroborate the findings with other techniques such as polysomnography. On the other hand, given that the study is cross-sectional, it is not possible to determine whether the variables identified as risk factors occurred prior to sleep disorders or whether it is an interrelation between variables. It should be noted that the sample of patients with TBI is very small and not balanced with those with stroke, so comparisons between these groups would not be completely valid.

Ethical considerations: This study was carried out in accordance with the Declaration of Helsinki (1964) and with the current national guidelines for human research. The research was evaluated and approved by the research and ethics committees of the National Institute of Rehabilitation LGII of Mexico City.

All participants signed and received a copy of the informed consent in which they voluntarily accept-ed their collaboration in the study.

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