Causes of Excessive Daytime Sleepiness in Patients with Acute Stroke—A Polysomnographic Study

Katarína Klobučníková, MD, PhD, Pavel Šiarnik, MD, Zuzana Čarnická, MD, Branislav Kollár, MD, PhD, and Peter Turčáni, MD, PhD

Background: Sleep disorders are common in stroke patients. Sleep-disordered breathing (SDB), which is present in up to 72% of stroke patients, is the most frequent cause of excessive daytime sleepiness (EDS) in common population. The aim of this study was to assess the frequency of EDS in stroke patients and to analyze the impact of SDB, stroke severity, and location of stroke on EDS in the acute phase of stroke. *Methods:* We enrolled 102 patients with the clinical diagnosis of acute stroke. Baseline clinical characteristics were recorded on admission. An Epworth sleepiness scale score higher than 9 was considered as EDS. To detect SDB, we performed standard overnight polysomnography within 4 ± 2 days after the stroke onset. *Results:* EDS was present in 21 patients (20.6%). In a population with EDS, we found a significantly higher number of obstructive apneic pauses, central apneic pauses, as well as significantly higher values of respiratory disturbance index (RDI), RDI during nonrapid eye movement sleep, desaturation index, and significant decrease of REM sleep duration. RDI (odds ratio [OR], 1.031; 95% confidence interval [CI], 1.007-1.056; $P = .01$) and duration of REM sleep (OR, .922; 95% CI, .853-.997; $P = .042$) were the only independent variables significantly associated with EDS in a binary multivariate regression model. *Conclusion:* SDB is a common, significant, and treatable cause of EDS in acute stroke patients. We suppose that examination in sleep laboratories is reasonable in all stroke patients with EDS, although the impact of SDB therapy on EDS and overall outcome in acute stroke remains unknown.
Key Words: Excessive daytime sleepiness—acute stroke—polysomnography—sleep-disordered breathing—REM sleep.

Introduction

Excessive daytime sleepiness (EDS) is present in up to 4.8% of the common population and in up to 15.2% of shift workers.[1,2] The most common cause of EDS are sleep disorders including insomnia, sleep-disordered breathing (SDB), restless legs syndrome, periodic limb movement disorder, but also circadian rhythm disorders, improper sleep hygiene, or drug abuse. Structural impairment of the central nervous system can be another cause of EDS.[3] Sleep disorders are common in stroke patients. SDB is present in up to 72% and other sleep disorders (insomnia, hypersomnia, sleep movement disorders, and parasomnias) in 10%-50% of stroke patients. Despite adverse impact on overall outcome, sleep disorders are commonly underdiagnosed in stroke patients.[4,5] The aim of the study was to assess the frequency of EDS in stroke

From the 1st Department of Neurology, Faculty of Medicine, Comenius University, Bratislava, Slovakia.

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Address correspondence to Pavel Šiarnik, MD, 1st Department of Neurology, Faculty of Medicine, Comenius University, Mickiewiczova 13, 81369, Bratislava, Slovakia. E-mail: palo.siarnik@gmail.com.

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patients and to analyze the impact of SDB, stroke severity, and stroke location on EDS in the acute phase of stroke.

Methods

From the population of patients hospitalized in the stroke unit of the 1st Department of Neurology, Comenius University, Bratislava, we have prospectively enrolled 102 patients with a clinical diagnosis of acute stroke. The stroke workup included neuroimaging (computed tomography or magnetic resonance imaging of the brain). Baseline clinical characteristics were recorded on admission. Severity of stroke was assessed according to The National Institutes of Health Stroke Scale (NIHSS). Presence of obesity was assessed according to body mass index and presence of EDS according to Epworth sleepiness scale (ESS).[6] The sleep study was performed within 4 ± 2 days after the stroke onset. We performed standard overnight polysomnography in sleep laboratory settings using Alice 5 device (Philips Respironics, Best, The Netherlands). Total sleep time, proportion of particular sleep stages (N1, N2, N3, REM in %), sleep efficiency, arousal index, number of apneic pauses, respiratory disturbance index (RDI), and desaturation index were recorded. Sleep parameters and respiratory events were scored according to standardized criteria. Apnea was defined as the cessation or the reduction of airflow of 90% or more for more than 10 seconds, hypopnea as a reduction in airflow of 50% or more for 10 seconds with oxygen desaturation of more than 3%. RDI was defined as the total number of apneas, hypopneas, and respiratory effort-related arousals per hour of sleep. Respiratory effort-related arousals were estimated by flattening of the inspiratory airflow profile associated with an arousal, when airflow changes did not meet apnea or hypopnea criteria.[1] The study was approved by the institutional ethics committee and all patients signed the informed consent. The statistical analyses were performed using SPSS version 18 (SPSS Inc., Chicago, IL). Categorical variables were expressed as numbers and proportions (%), and continuous variables as means ± standard deviation. Chi-squared test, Student *t*-test, and Mann–Whitney test were used for group comparison. Binary logistic regression analysis was used to identify factors that contributed to the EDS. *P* values less than .05 were considered statistically significant.

Results

Our study population consisted of 58 males and 44 females with a mean age of 68 ± 12 years. Brain infarction was present in 93 patients, transient ischemic attack in 4 patients, and intracerebral hemorrhage in 5 patients. Supratentorial strokes were present in 85 patients and infratentorial strokes in 17 patients. EDS (ESS score > 9) was present in 21 patients (20.6%). The characteristics of populations with and without EDS are included in Table 1.

In both groups, we found no significant difference in age and body mass index. The patients with EDS had nonsignificantly higher NIHSS scores (5.83 ± 1.52 versus 3.91 ± .34) and a nonsignificantly higher proportion of infratentorial strokes (23.8% versus 14.8%). We found no significant difference in total sleep time; duration of sleep stages N1, N2, and N3; sleep efficiency; and arousal index. In the population with EDS, we found a significant decrease in REM sleep duration.

In the population with EDS, we found a significantly higher number of obstructive apneic pauses, central apneic pauses, as well as significantly higher values of RDI, RDI during nonrapid eye movement, and desaturation index. RDI (odds ratio, 1.031; 95% confidence interval, 1.007-1.056; *P* = .01) and duration of REM sleep (odds ratio, .922; 95% confidence interval, .853-.997; *P* = .042) were the only independent variables significantly associated with EDS in a binary multivariate regression model (see Figs 1, 2).

Discussion

In our study, EDS was present in 20.6% of patients with acute stroke. RDI and decrease of REM sleep duration were the only independent variables significantly associated with EDS in the binary multivariate regression model. We found no significant impact of stroke site and severity on EDS in acute phase of stroke.

We emphasize high frequency of EDS in acute stroke because such patients are known to have worse clinical outcome.[7] The frequency of EDS in our population with acute stroke is higher than in the common population, where it is present in up to 4.8%.[8] SDB is a known cause of EDS and it is also a risk factor for the development of vascular disease including stroke.[9,10] SDB can be present in up to 72% of stroke patients.[5,11] High frequency of sleep apnea syndrome was present also in our population (77.8% in the population without EDS and 76.2% in the population with EDS). SDB in the population with EDS was significantly more severe (RDI: 32.56 ± 5.18 versus 20.37 ± 2.16). SDB is a cause of changes in sleep architecture as well as a cause of REM sleep disruption.[12] The recuperative effect of REM sleep was described in previous studies.[13] The significant decrease in REM sleep duration in the population of patients with EDS in our study is consistent with these findings.

Our study has several limitations. Although we have found a significant effect of SDB severity and a significant effect of REM sleep duration on EDS, we have not studied the possible impact of sleep movement disorders and depression, which can also lead to increase of daytime sleepiness.[14,15] Another limitation of the study is the absence of premorbid sleep complaint scoring, which could elucidate the possible impact of premorbid SDB symptoms on EDS. Although we found nonsignificantly higher NIHSS scores in patients with EDS, the role of stroke site and size in neuroimaging findings was not