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Early sleep apnea screening on a stroke unit is feasible in patients with acute cerebral ischemia

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Abstract

Early screening for sleep apnea (SA) is rarely considered in patients with acute cerebral ischemia. We aimed to evaluate the feasibility of early SA screening on a stroke unit, its impact on post-discharge SA care and the relation of SA to clinical features. Patients with acute ischemic stroke (AIS) and transient ischemic attack (TIA) prospectively underwent overnight cardiorespiratory polygraphy within 3 ± 2 days of symptom-onset. Feasibility was defined as analyzable polygraphy in 90 % of studied patients. We enrolled 61 patients (84 % AIS, 16 % TIA): mean age 66 ± 8 years, 44 % men, median NIHSS 1 (0–15), median ESS 5 (0–13). Analyzability was given in 56/61 (91.8 %; one-sided 95 % CI, lower-bound 86.0 %) patients indicating excellent feasibility of early SA screening with no significant differences in stroke severity (100 % in TIA, 91 % minor stroke, 83 % major stroke, $p = 0.474$). Ninety-one percent (51/56) had an apnea–hypopnea index ≥5/h (median: 20/h [0–79]); 32 % (18/ 56) mild, 30 % (17/56) moderate, and 29 % (16/56) severe SA. When comparing sleep-related ischemic stroke (SIS) and non-SIS patients, no differences were found regarding the presence (95 vs. 89 %, $p = 0.49$) or severity (e.g., severe SA: 32 vs. 27 %, $p = 0.69$) of SA. After 12 months, 27/38 (71 %) patients given specific recommendations completed in-laboratory sleep work-up and 7/27 (25 %) were prescribed for non-invasive ventilatory correction. In conclusion, early SA screening is feasible in patients with acute cerebral ischemia and may have a positive impact on post-discharge SA care. Given the high frequency and atypical presentation of SA, early screening for SA should be considered in all acute cerebral ischemia patients.

Keywords

Stroke; Sleep apnea; Secondary prevention

Introduction

Sleep apnea (SA) is increasingly recognized by the stroke community as an independent stroke risk factor and serious disorder that contributes to early neurological deterioration, worse outcome and increased mortality in acute ischemic stroke (AIS) patients [1–7].

However, little has been done yet to incorporate facilitation of early evaluation and management of these patients into stroke guidelines [8–10]. Experts advocate that the suspicion of SA should trigger a comprehensive sleep evaluation including sleep-oriented history and objective sleep testing by means of attended, in-laboratory polysomnography (PSG) [11]. The latter approach may be reasonable in the ambulatory setting but in the hospital setting, only a few stroke centers have access to sleep medicine for in-patient populations and even this access may not be continuously available. Furthermore, AIS patients need continuous monitoring as well as constant nursing care and it may be hazardous to move them for in-laboratory sleep testing during the acute phase of stroke.

Identification of stroke etiology and associated vascular risk factors constitutes an integral part of organized inpatient stroke unit care, and its benefit on long-term functional outcome and survival of stroke patients has been clearly demonstrated [12]. Cardiorespiratory polygraphy, a portable SA screening device, reliably allows detection of SA when compared with the gold-standard PSG [13, 14]. Nonetheless, available data on the use of such devices in hospitalized patients, not to mention stroke patients are limited [15]. Thus, it is of particular interest, whether SA monitoring using such devices can be implemented during the early stage of stroke as part of organized stroke unit care.

We aimed to evaluate the feasibility of early bedside SA screening in patients with acute cerebral ischemia on a stroke unit and its impact on post-discharge SA care. As secondary aims we investigated the clinical presentation of SA in this patient population as well as its relation to diurnal variations in stroke occurrence.

Methods

Study population

We prospectively enrolled consecutive patients with AIS and transient ischemic attack (TIA) age 18–75 years who were admitted to our stroke unit at the Dresden University Stroke Center from November 2009 to May 2011 (convenience sampling). Exclusion criteria were an unfavorable premorbid functional condition defined by a modified Rankin Scale (mRS) score >3 points, pre-existing SA and severe comorbidity (i.e., congestive heart failure, chronic lung disorder, any actual malignant disease, dementia) that would compromise therapeutic consequences. Patients who were not able to give written consent (e.g., severe aphasia) were excluded from this study. Our study was approved by the local Institutional Review Board and written informed consent was obtained from each patient.

Clinical evaluation at baseline

Demographic and anthropometric (weight, height, body mass index [BMI]) data, baseline National Institutes of Health Stroke Scale (NIHSS) scores (categorized as minor <6 and major stroke ≥6 points), vascular risk factors, stroke etiology (classified by Trial of Org 10172 in Acute Stroke Treatment [TOAST] criteria) [16] and brain imaging findings were assessed prospectively. Presumptive onset of stroke symptoms was obtained from each patient and/or their relatives. Sleep-related ischemic stroke (SIS) was defined as the presence

of stroke symptoms on awakening (wake-up stroke, WUS) or symptom-onset within 1 h of awakening from sleep. All other patients were considered non-SIS.

Evaluation for sleep apnea

We interviewed patients for the presence of excessive daytime sleepiness using the Epworth Sleepiness Scale (ESS). The ESS was considered abnormal when the score was ≥10 points [17].

All patients underwent overnight SA screening within 3 ± 2 days from symptom-onset while admitted to the stroke unit using a 6-channel portable cardiorespiratory polygraphy device (SOMNOcheck effort®, Weinmann Medical Technology, Hamburg, Germany). With this cardiorespiratory polygraphy device, nasal airflow was measured by using a thermistor, body position and respiratory movements by a piezoelectric sensor located in chest and abdomen fixing straps, and capillary oxygen saturation and heart rate were measured by a pulse oximeter finger clip. The patients were monitored from 11 pm to 7 am without interfering with conventional stroke care. Stroke fellows were instructed to attach the device properly by 10:30 pm and the device was detached by a nurse the next morning.

Polygraphy data were transmitted to a PC and SOMNOcheck analysis software (Weinmann Medical Technology, Hamburg, Germany) automatically generated the sleep polygram. The sleep polygrams were analyzed manually by a sleep neurologist (JK, WS) according to the American Academy of Sleep (AASM) guidelines (AASM Manual for Scoring of Sleep and Associated Events) [18]. An apnea during sleep was defined as cessation of airflow (>90 % fall in the amplitude of airflow signal compared to the baseline airflow) lasting at least 10 s. Apnea was further classified as obstructive with continued respiratory effort, as central with absent respiratory effort and as mixed with absent respiratory effort followed by resumption of respiratory effort during apnea. Hypopnea was defined as a 50 % or greater fall in airflow lasting ten or more seconds associated with a 3 % or greater fall in oxygen saturation from baseline. The apnea–hypopnea index (AHI) was calculated using the total number of respiratory events (apneas and hypopneas) per hour sleep, and categorized in any (AHI ≥ 5/h), mild (AHI 5–14/h), moderate (AHI 15–29/h) and severe (AHI ≥ 30/h) SA as previously reported [11].

Feasibility of overnight cardiorespiratory polygraphy was defined as an analyzable polygraphy in 90 % of studied patients. Analyzability was mainly based on completeness of polygraphy (i.e., all parameters were captured for at least 6 h allowing proper calculation of the AHI), but eventually at the discretion of the sleep neurologist (particularly for borderline findings). Reasons for non-analyzable polygraphies were recorded.

Post-discharge observation

According to polygraphy findings, each patient with moderate and severe SA was given a specific recommendation by a sleep neurologist (JK, WS) in order to undergo further in-laboratory sleep work-up (i.e., nocturnal PSG). In patients with mild SA, recommendations were given only when concurrent sleep-related symptoms were present as suggested by the AASM guidelines [11]. Further diagnostic and therapeutic sleep studies were performed outside this study. A follow-up at 6 and 12 months was performed by telephone interview

including assessment of the mRS score and recurrent cardio- or cerebrovascular events. Results of further sleep laboratory work-up and corresponding SA therapy (if prescribed) were retrieved from sleep laboratory reports.

Statistical analysis

We assumed an analyzable polygraphy in 90 % of the study population. With a one-sided confidence interval (CI) of 95 % and a lower confidence limit of 82.5 %, a minimum of 44 analyzable patients would be needed to proof feasibility of polygraphy in our study population. Categorical variables were assessed using Chi-square tests or Fisher's exact test while continuous variables were assessed using Student's *t* test and Wilcoxon rank sum test, where appropriate. Spearman's correlation was used to assess correlations between the AHI and clinical variables of interest. Logistic regression was used to determine if AHI was a significant independent predictor of SIS or WUS. Since the latter was an exploratory analysis, no adjustments for multiple comparisons were made. Adjusted models were not used when the crude model was not significant. Sensitivity with corresponding 95 % CI's of cardiorespiratory polygraphy for detection of SA was calculated after computation of true positive and false negative values according to follow-up PSG results. Missing data for mRS were imputated using the last-observation-carried-forward method. A p value of <0.05 was considered to be statistically significant.

Results

Of approximately 800 patients with acute cerebral ischemia admitted to our tertiary stroke center during the study period, we enrolled a convenience sample of 61 patients: mean age was 65.6 ± 7.5 years, 44 % were men, median baseline NIHSS score was 1 (range 0–15) point, median ESS was 5 (0–13) points. Fifty-one out of 61 (84 %) patients had an AIS, 10/61 (16 %) had a TIA. Baseline characteristics and clinical data of the study population are presented in Table 1 .

Cardiorespiratory polygraphy was performed 2.1 ± 1.1 days after stroke onset. Analyzability was given in 56/61 (91.8 %; one-sided 95 % CI, lower-bound 86.0 %) patients indicating excellent feasibility of overnight SA monitoring in patients with acute cerebral ischemia. There were no significant differences in terms of analyzability according to stroke severity (100 % in TIA, 91 % minor stroke, 83 % major stroke, $p = 0.474$). Cardiorespiratory polygraphy was not analyzable due to missing data for nasal airflow in 1/61 (1.6 %) patient, for oxygen saturation in 2/61 (3.3 %), and due to patients' adherence in 2/61 (3.3 %) patients.

Median AHI was 20/h (0–79/h). Fifty-one out of 56 (91 %, 95 % CI, 80.7–96.1 %) analyzable patients had an AHI ≥ 5/h: 32 % (18/56) mild, 30 % (17/56) moderate and 29 % (16/56) severe SA. The predominant type of SA was obstructive (44/51, 86 %), whereas only 2/51 (4 %) patients had central and 5/51 (10 %) mixed SA. There was no significant difference in the prevalence of SA in TIAs, minor strokes, and major strokes (100, 88, and 100 %, respectively). According to stroke etiology (as indicated by TOAST classification), the prevalence of SA seemed higher in patients with large artery atherosclerosis, cardioembolism and small artery occlusion, as compared to those with undetermined cause

(LAA 94 % [32/34] vs. CE 100 % [10/ 10] vs. SAO 100 % [7/7] vs. UND 40 % [2/5]; $p < 0.001$, respectively). In addition, the magnitude of AHI differed significantly across stroke etiologies with undetermined cause the lowest and small vessel disease as well as cardioembolism the highest (mean AHI: 6.2/h vs. 31.4/h vs. 33.4/h; $p = 0.039$). In terms of clinical presentation of SA, no significant correlation was found between excessive daytime sleepiness (ESS) and AHI ≥ 5/h ($r = 0.123$, $p = 0.366$). The moderate strength positive correlation seen between BMI and AHI ≥ 5/h ($r = 0.250$, $p = 0.063$) appeared slightly stronger in the AIS subgroup ($r = 0.276$, $p = 0.063$).

At discharge, recommendations for further in-laboratory sleep work-up were given to 38/51 (75 %) patients (Fig. 1). Mean elapsed time from recommendation to sleep laboratory work-up was 136 ± 62 days. After 6 months, 23 of 38 (61 %) patients completed in-laboratory PSG, of which 2/23 (9 %) started treatment with non-invasive ventilatory correction. One out of 51 (2 %) patients expired due to a recurrent ischemic stroke. After 12 months, 27/38 (71 %) patients completed in-laboratory PSG. Of these patients, 19/27 (70 %) had moderate-to-severe SA (i.e., clinically relevant) and 7/27 (25 %) were prescribed non-invasive ventilatory correction. The remaining 12/27 (75 %) patients refused non-invasive ventilatory correction or favored conservative treatment (i.e., sleep postural changes, weight loss, oral appliances). As compared with the follow-up complete PSG, cardiorespiratory polygraphy yielded sensitivity of 60 % (95 % CI 31.3–83.2 %) for detection of moderate and 77.8 % (95 % CI 45.3–93.7 %) for severe SA; however, in combined moderate-to-severe SA, sensitivity of early polygraphy reached 94.7 % (95 % CI 75.4–99.1 %). Two out of 51 (4 %) patients had a recurrent ischemic stroke, one of which expired. These two patients were diagnosed with severe SA, but noninvasive ventilatory therapy was not started yet. No cardiovascular events were reported during the post-discharge observation period. Functional outcome is presented in Table 1.

Diurnal variations of stroke

Sleep-related ischemic stroke was found in 19/56 (34 %) patients including 14/56 (25 %) patients with WUS. Fewer men were among SIS patients (26 vs. 57 %, $p = 0.03$) and SIS patients had a higher prevalence of atrial fibrillation (32 vs. 5 %, $p = 0.008$). There were no statistically significant differences in the remainder of baseline characteristics (Table 2). When comparing SIS and non-SIS patients, no differences were found regarding the presence (95 vs. 89 %, $p = 0.49$) or severity (e.g., severe SA: 32 vs. 27 %, $p = 0.69$) of SA. Similarly, there were no differences in the presence (100 vs. 88 %, $p = 0.17$) or severity (e.g., severe SA: 32 vs. 29 %, $p = 0.66$) of SA when comparing WUS and non-WUS patients. The AHI did not prove to be a significant independent predictor of SIS ($p = 0.45$) or WUS ($p = 0.56$).

Discussion

Our study showed that early sleep apnea screening is feasible in the acute phase of stroke when a portable cardio-respiratory polygraphy device is used. High frequency of SA in our study population with no significant relation to clinical features highlights the importance of

SA screening in all acute stroke patients. This implies a first step toward its routine implementation in organized stroke unit care.

Untreated SA of variable degree constitutes an independent risk factor for recurrent ischemic stroke [19–21]. As part of secondary prevention in stroke survivors with SA, initiation of non-invasive ventilatory correction reduces the risk of recurrent ischemic stroke and mortality [21–23]. Moreover, continuous positive airway pressure improves short-term functional outcome in stroke survivors with SA undergoing rehabilitation [24]. Apart from that, fewer publications showed feasibility and safety of early non-invasive ventilatory correction in the acute phase of stroke addressing the important association of SA and early neurological deterioration [10, 25–27]. However, timely detection of vascular risk factors remains a crucial key issue when goals for secondary prevention of stroke are defined [12]. Although awareness of SA and its sequelae in stroke survivors have increased among stroke-neurologists [28], post-stroke care commonly lacks accessible resources compromising proper screening and treatment of SA in these patients [29, 30]. A recently conducted survey revealed that ischemic stroke survivors perceived their risk of SA lower than it was and only a few of those who were at high risk of SA underwent corresponding screening or treatment after being discharged from the hospital [31]. Organized stroke unit care, which has a positive impact on outcome and recurrent stroke risk, may overcome this shortcoming [12], and so it is potentially worth devoting further in-hospital resources.

To the best of our knowledge, fewer studies assessed feasibility of early SA screening applying a portable easy-to-operate device in the acute phase of stroke as most studies utilized complete nocturnal PSG for SA testing in stroke patients and were primarily conducted to pursue epidemiological rather than feasibility objectives [3, 32, 33]. Furthermore, in previous studies, diagnostic testing for SA was mostly performed outside the acute phase and rather in a general neurology or rehabilitation ward [34, 35]. Furthermore, even though PSG is considered the gold-standard for diagnosis of SA [11], it requires considerable experience and constant access to sleep medicine that only a few stroke centers provide. Also, in the acute phase of stroke this approach may interfere with basic stroke care.

Aside from an association between the prevalence and severity of SA and presumed stroke etiology in our patient population (which should be interpreted with caution due to small absolute numbers in certain TOAST subtypes), we did not find further links and, therefore, no certain subgroup of stroke patients that preferably should be screened for SA. This might be due to the overall high frequency (91 %) of SA in our study population with no relevant differences according to stroke severity. Although some debate exists in this matter, our findings are in line with most previous studies [36, 37]. As demonstrated by Chan and colleagues [34] the majority of stroke patients with SA does not present with typical SA-related clinical features such as obesity and excessive daytime sleepiness. In our population, excessive daytime sleepiness did not prove to be associated with SA severity confirming the aforesaid results, whereas BMI was linked to severity of SA though. Beyond that, there is an ongoing controversy whether sleep-related stroke onset is associated with SA [33, 34, 38]. However, we did not find any relevant relation between SA and diurnal variations of stroke onset, not even when solely focusing on wake-up strokes as opposed to recent findings by

Hsieh et al. [39]. Nonetheless, the question arises whether clinical features are necessary to select patients for SA screening, or selection criteria can be omitted and all stroke patients should be screened as recently suggested [37]. The overall excellent feasibility of SA screening in patients with TIA and AIS observed in our study aids this approach.

Within one year of discharge from our hospital, nearly three out of four patients with findings suggestive of treatable SA underwent comprehensive in-laboratory sleep work-up and therewith followed our recommendations. This is in contrast to a recent survey study in ischemic stroke survivors which showed that only 26 % of respondents were asked at least one SA symptom screening question by a healthcare provider after being discharged from the hospital [31]. Moreover, <20 % of those who responded to the survey underwent any SA screening. As with other modifiable stroke risk factors, SA requires timely detection, and early implementation of SA screening in acute stroke patients may have a positive impact on post-stroke care. However, elapsed time between initial SA screening and in-laboratory work-up appears rather long (on average 5 months). On the one hand, this was partly intended by our sleep neurologists who gave specific recommendations to stroke survivors as SA may slightly improve during the post-stroke phase [37]. On the other hand, stroke survivors' motivation for further SA work-up was rather driven by these recommendations than self-determination as SA-related symptoms were widely not present in these patients (e.g., most of the patients did not complain of excessive daytime sleepiness as assessed by the ESS). This might also explain that only a minority of patients who were found to have treatable SA eventually received non-invasive ventilatory correction, which was mostly due to patients' refusal of treatment. This emphasizes the need for constant medical attendance and continuing education provided to stroke survivors after discharge [40].

Our study has some limitations: First of all, our study population may reflect a sampling bias since very few had major strokes (median NIHSS was 1 point) and severe comorbidities were largely not present. In these patients early SA screening may be less feasible due to agitation and non-compliance. Thus, our feasibility rate might be overestimated and not entirely generalizable. Although we enrolled patients consecutively, we did not apply a screening log and therefore cannot provide the exact number of patients who did not fulfill inclusion and exclusion criteria. Secondly, we used a conservative definition for SA (which does not necessarily require treatment) and therefore might have overestimated the frequency of SA in our patient population. However, we strictly complied with the AASM guidelines as opposed to a less conservative cut-off frequently used in other trials [41]. Also, certain subanalyses may have been affected by the overall high frequency of SA in our study population. Lastly, we were not able to evaluate sleep stages during SA monitoring since the polygraphy device does not allow electroencephalography recording. However, cardiorespiratory polygraphy has been shown to reliably detect SA when compared with the gold-standard PSG [13].

In conclusion, early SA screening is feasible in patients with acute cerebral ischemia and may have a positive impact on post-discharge SA care. Given the high frequency and atypical presentation of SA in this patient population, early screening for SA should be considered in all acute cerebral ischemia patients.

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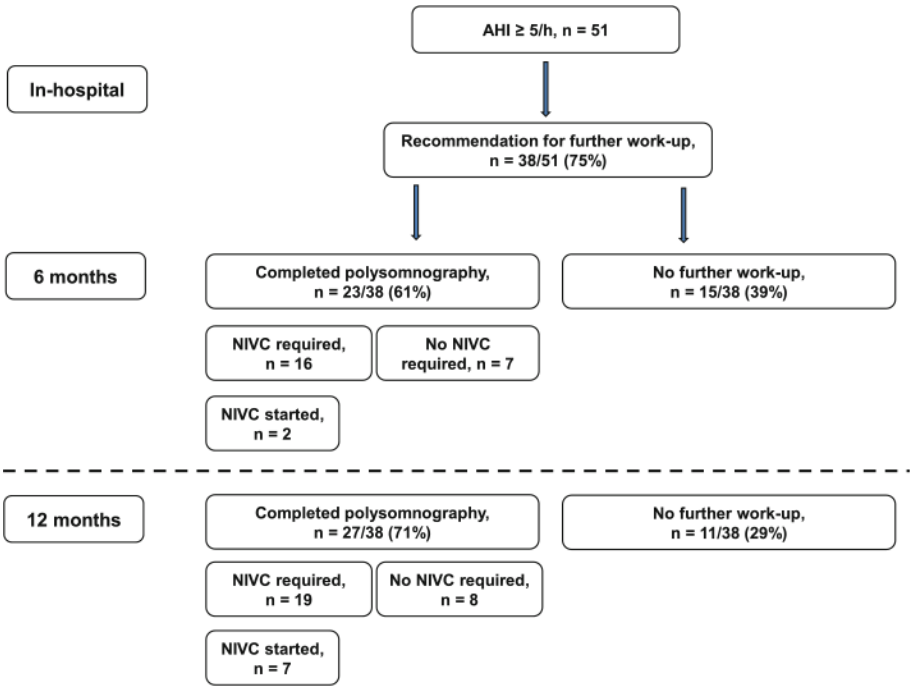

Fig. 1.

Post-discharge flow chart. *AHI* apnea–hypopnea index, *NIVC* non-invasive ventilatory correction

Table 1
Baseline characteristics and clinical data of the study population

	Total (*n* = 61)	Analyzable (*n* = 56)	Non-analyzable (*n* = 5)	*p* value
Demographics				
Mean age ± SD, years	64.6 ± 7.5	64.3 ± 7.7	67.6 ± 3.5	0.359
Gender, male, *n* (%)	27 (44)	26 (46)	1 (20)	0.254
Risk factors				
Hypertension, *n* (%)	54 (89)	49 (88)	5 (100)	0.401
Diabetes, *n* (%)	21 (34)	18 (32)	3 (60)	0.209
Dyslipidemia, *n* (%)	35 (57)	33 (59)	2 (40)	0.412
Smoking (past 5 years), *n* (%)	13 (21)	12 (21)	1 (20)	0.940
Coronary artery disease, *n* (%)	4 (7)	4 (7)	0	0.536
Atrial fibrillation, *n* (%)	10 (16)	8 (14)	2 (40)	0.137
Previous ischemic stroke/TIA, *n* (%)	22 (36)	20 (36)	2 (40)	0.426
Alcohol daily, *n* (%)	8 (13)	7 (12.5)	1 (20)	0.634
Clinical data				
Mean BMI ± SD	27.2 ± 3.8	27.1 ± 3.9	27.9 ± 1.6	0.708
Median ESS, range	5 (0–13)	5 (0–13)	5 (2–7)	0.559
Median NIHSS, range	1 (0–15)	1 (0–15)	0 (0–7)	0.191
Stroke severity				0.544 *
TIA, *n* (%)	10 (16)	10 (18)	0	
Minor stroke, *n* (%)	45 (74)	41 (73)	4 (80)	
Major stroke, *n* (%)	6 (10)	5 (9)	1 (20)	
TOAST	0.529 *			
Large artery, *n* (%)	37 (61)	34 (61)	3 (60)	
Cardioembolism, *n* (%)	12 (20)	10 (18)	2 (40)	
Small vessel, *n* (%)	7 (11)	7 (12.5)	0	
Other, *n* (%)	0	0	0	
Undetermined, *n* (%)	5 (8)	5 (9)	0	
Median mRS 6 months, range	1 (0–6)	1 (0–6)	1 (0–4)	0.794
Median mRS 12 months, range	1 (0–6)	1 (0–6)	1 (1–2)	0.469

TIA transient ischemic attack, *BMI* body mass index, *ESS* Epworth Sleepiness Scale, *NIHSS* National Institutes of Health Stroke Scale, *TOAST* Trial of Org 10172 in Acute Stroke Treatment, *mRS* modified Rankin scale

* Overall *p* value for all categories

Table 2

Baseline characteristics and clinical data of the study population (separated by sleep-related- and non-sleep-related stroke onset)

	SIS (*n* = 19)	Non-SIS (*n* = 37)	*p* value
Demographics			
Mean age ± SD, years	65.8 ± 7.0	63.6 ± 8.1	0.304
Gender, male, *n* (%)	5 (26)	21 (57)	0.031
Risk factors			
Hypertension, *n* (%)	17 (89)	32 (88)	0.749
Diabetes, *n* (%)	6 (32)	12 (32)	0.948
Dyslipidemia, *n* (%)	9 (47)	24 (41)	0.208
Smoking (past 5 years), *n* (%)	2 (11)	10 (65)	0.154
Coronary artery disease, *n* (%)	1 (5)	3 (8)	0.696
Atrial fibrillation, *n* (%)	6 (32)	2 (5)	0.008
Previous ischemic stroke/TIA, *n* (%)	4 (21)	16 (43)	0.183
Alcohol daily, *n* (%)	2 (11)	5 (13.5)	0.749
Clinical Data			
Mean BMI ± SD	27.5 ± 2.9	27.0 ± 4.4	0.706
Median ESS, range	5 (0–13)	5 (0–11)	0.701
Median NIHSS, range	1 (0–15)	1 (0–7)	0.177
Stroke Severity			0.078*
TIA, *n* (%)	1 (5)	9 (24)	
Minor stroke, *n* (%)	16 (84)	25 (68)	
Major stroke, *n* (%)	2 (11)	3 (8)	
TOAST			0.051*
Large artery, *n* (%)	10 (53)	24 (65)	
Cardioembolism, *n* (%)	7 (37)	3 (8)	
Small vessel, *n* (%)	1 (5)	6 (16)	
Other, *n* (%)	0	0	
Undetermined, *n* (%)	1 (5)	4 (11)	
Sleep apnea, *n* (%)	18 (95)	33 (89)	0.801*
Mild	5 (26)	13 (35)	
Moderate	7 (37)	10 (27)	
Severe	6 (32)	10 (27)	

SIS sleep-related ischemic stroke, *TIA* transient ischemic attack, *BMI* body mass index, *ESS* Epworth Sleepiness Scale, *NIHSS* National Institutes of Health Stroke Scale, *TOAST* Trial of Org 10172 in Acute Stroke Treatment

*Overall *p* value for all categories