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Nocturia, Sleep and Daytime Function in Stable Heart Failure

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Abstract

Background—To evaluate nocturia severity and nocturia-related differences in sleep, daytime symptoms and functional performance among patients with stable heart failure (HF).

Method & Results—In this cross-sectional observational study we recruited 173 patients [M age = 60.3 ±16.8 years; n = 60 (35%) female; left ventricular ejection fraction M = 32 ±14.6] with stable chronic HF from HF disease management programs in the Northeastern United States. Participants reported nocturia and completed a Six Minute Walk test (6 MWT), one night of ambulatory polysomnography (PSG), and the Medical Outcomes Study SF 36, Epworth Sleepiness, Pittsburgh Sleep Quality Index, Multi-Dimensional Assessment of Fatigue, and the Centers for the Epidemiological Studies of Depression scales. Participants reported no (n = 30/17.3%), one or more (n = 87/50.2%), and three or more (n = 56/32.4%) nightly episodes of nocturia. There were decreases in sleep duration and efficiency, stages REM and 3–4 sleep,

Disclosures
There is nothing to disclose.

physical function, and 6 MWT distance; and increases in the percent wake after sleep onset, insomnia symptoms, fatigue and sleepiness across levels of nocturia severity.

Conclusions—Nocturia is common, severe, and closely associated with decrements in sleep and functional performance and increases in fatigue and sleepiness in patients with stable HF.

Keywords

heart failure; insomnia; nocturia; sleep; fatigue; physical function; quality of life

Introduction

Nocturia is common among patients with heart failure (HF) and is often a reported cause of poor sleep.[1–3] In the general population, nocturia was closely associated with poor sleep quality,[4–7] a 75% increased risk of insomnia, and a 71% increased risk of poor sleep in men and women between the ages of 55 and 84.[8]

Although it is often presumed that nocturia leads to disturbed sleep, it is also plausible that awakening results in the perception of the need to void.[8] Awakenings may result from a host of environmental, social, psychological, and health-related reasons, including sleep disordered breathing (SDB), that are closely associated with poor sleep architecture and decrements in sleep continuity.[9] Resulting decreases in slow wave sleep may lead to decreased secretion of renin and aldosterone,[10] and decreased REM sleep may lead to increased urine flow and decreased osmolality.[11] These factors may lead to nocturia.

Sleep disordered breathing (SDB), including Cheyne Stokes breathing/central sleep apnea and obstructive sleep apnea, are common in patients with HF.[12] Obstructive sleep apnea is associated with elevated intrathoracic pressure which leads to increased atrial natruretic peptide, while central sleep apnea is usually associated with decompensated HF[13] - both of which may lead to nocturia. Although studies have shown that obstructive sleep apnea is associated with nocturia in the general population,[13–15] a recent study demonstrated that nocturia and SDB were not related in HF patients.[16]

Numerous studies have demonstrated that nocturia is associated with daytime symptoms (fatigue, depression, sleepiness) and poor daytime function and quality of life in middle-aged and older adults.[4,5,7,17,18] Although these daytime problems, as well as nocturia and poor self-reported sleep and insomnia are common in patients with HF, there have been few attempts to quantify nocturia severity or the differences in sleep, daytime symptoms (e.g., depression, fatigue, sleepiness) or functional performance across levels of nocturia severity in these patients. Given the high burden of symptoms and poor functional performance among HF patients, it is important to understand and address possible contributing factors, such as nocturia. Therefore, the purposes of this study were to evaluate: 1) the extent to which HF patients reported nocturia and nocturia severity; 2) clinical and demographic differences across levels of nocturia severity; 3) differences in self-reported and objective characteristics of sleep across levels of nocturia severity; and 4) differences in daytime symptoms (depression, fatigue, sleepiness), and self-reported and objective functional performance across levels of nocturia severity in community-residing patients with stable HF.

Methods

Design

We conducted a cross-sectional study to evaluate the study aims described above. This report was part of a larger study that had the overall purpose of evaluating the extent to which sleep and sleep disordered breathing explained daytime symptoms and functional performance. Full details of the study design and methods have previously been published,[9,19] but are summarized here as relevant to the current report.

Sample

The sample included patients with stable HF recruited from five specialized HF disease management programs in the Northeastern United States. Participants had stable New York Heart Association Class II – IV HF. Stability was defined as the absence of hospital admissions, emergency department visits, or titration of vasoactive medications within a month before the sleep evaluation. We excluded patients who had cognitive impairment, end stage renal failure, neurological or musculoskeletal conditions affecting mobility of the non-dominant arm (due to the use of wrist actigraphy), or previously identified SDB.

Procedures

Human subject's approval was obtained, and all patients provided written informed consent. Participants were recruited during routine visits to the HF programs. Medical records were reviewed, and participants completed the Six Minute Walk Test (6 MWT) in the clinic setting. Participants underwent a one night unattended ambulatory polysomnographic (PSG) study at home and completed sleep/symptom diaries and a packet of questionnaires to evaluate self-reported habitual sleep characteristics; nocturnal symptoms (nocturia, pain, and dyspnea); daytime symptoms (sleepiness, depression, fatigue); and self-reported functional performance. Each participant was paid $50 at the conclusion of data collection.

Variables and Measures

Nocturia was elicited in 2 ways in order to capture its frequency at a time proximal to the PSG and its "habitual" (over the past month) nature: 1) Participants were asked to record the number of times that they awoke each night to void in a daily sleep/symptom diary. We used the data from the night of the PSG as the proximal measure of nocturia severity and classified it as follows: 0 voids/night (Group I); 1–2 voids/night (Group II); 3 or more voids (Group III). 2) Habitual nocturia was elicited through the Sleep Habits Questionnaire[20] and defined as a response of "often or almost always" to the item eliciting the frequency of awakening at night to use the bathroom over the past month.

Sleep and Sleep Disorders

We used self-report (questionnaires), and physiological (polysomnography – PSG) measures of sleep in order to obtain information on patient perceptions, as well as its objective characteristics.

Polysomnography—We recorded unattended nocturnal polysomnography (PSG) for one night in participants' homes with the Safiro (Compumedics, Inc.) sleep recorder. We obtained electroencephalograms (C3/A2 and C4/A1), electro-oculograms, and bipolar submental electromyograms, respiratory effort, nasal flow via a pressure transducer, oxygen saturation, electrocardiogram, body position, and leg movements.

PSG studies were downloaded into a personal computer. They were scored manually on a high resolution monitor using 30- second epochs and standardized scoring methods. [21] Sleep duration, wake time, sleep latency (time from lights out until the first epoch of stage 1 sleep), the percentage of wake time after sleep onset and sleep stages were calculated. Sleep stages were calculated in minutes and expressed as the percentage of the total sleep period.

Self-Reported Sleep Characteristics—The Pittsburgh Sleep Quality Index (PSQI), a widely-used and reliable and valid measure of sleep quality[22] was used to obtain participants' perception of habitual sleep duration and latency (time to lights out until the beginning of sleep). Sleep efficiency was calculated as: [sleep duration/time in bed] X 100.

Insomnia Symptoms were evaluated with questions from the Sleep Habits Questionnaire (SHQ).[20] The presence of insomnia symptoms was determined by a response of "often" or "almost always" to one or more insomnia symptoms (difficulty initiating sleep, difficulty maintaining sleep, and awakening too early in the morning).[20] Coefficient alpha was 0.83 as calculated from the current data.[19]

Daytime Symptoms—We evaluated sleepiness, fatigue, and depressive symptoms – common and disabling daytime symptoms of HF and sleep disorders. The Epworth Sleepiness Scale (ESS), a reliable and valid measure of sleepiness occurring in everyday life[23–25] was used to measure sleepiness. Coefficient alpha was 0.77 on data obtained in this study.[9] A score of > 10 is indicative of clinically significant excessive daytime sleepiness.

The global fatigue score of the Multi-Dimensional Assessment of Fatigue Scale (MAF)[26,27] was used to measure fatigue. Reliability was documented in HF patients.[9]

The Center for Epidemiological Studies Depression Scale (CESD)[28,29] was used to measure depressive symptoms. It is reliable, valid, sensitive, and specific in a variety of populations.[30 31] The total scale score and the dichotomized scale score (CESD ≥ 16), indicating likelihood of clinically relevant depression, were used.

Functional Performance—Functional Performance is the "day to day corporeal activities people do in the normal course of their lives to meet basic needs, fulfill usual roles, and maintain health and wellbeing."[32 p. 198] The Six Minute walk test (6MWT) and the Medical Outcomes Study SF-36v2 Physical function subscale were used to evaluate objective and subjective attributes of functional performance, respectively.

The 6MWT[33] is a reliable and valid objective measure of the distance walked under controlled conditions.[33–35] A distance of < 1000 feet predicted mortality.[36] We reported 6 MWT as both a continuous and dichotomous (< 100 or ≥ 1000 feet) variable.

The Medical Outcomes Study SF 36 v2 Physical Function Subscale [37,38] was used to elicit self-reported physical function (PF). The SF36 has well-documented reliability and validity in healthy and chronically ill populations.[39–44] The Charlson comorbidity index was used as an indicator of comorbidity.[45]

Data Analysis

Data obtained from the questionnaires, diaries and PSG data were double-entered into a database and analyzed with SPSS v. 18. Data were cleaned and evaluated for missing values and corrected for skewness. We computed descriptive statistics (means, standard deviations, frequencies, and percentages) to describe the primary study variables. Analysis of variance was used to compare the nocturia severity groups on the continuous sleep, symptom, and functional performance variables, and chi square was used to evaluate the associations

between nocturia severity categories and the categorical clinical, demographic, sleep, symptom, and functional performance variables. Analysis of covariance was used to evaluate the extent to which the effects of nocturia severity on the dependent continuous variables were independent of the effects of covariates, including clinical and demographic characteristics and percentage of time awake at night. Logistic regression analysis was used to evaluate the odds ratios for the associations between nocturia severity and insomnia, 6MWT and excessive daytime sleepiness dichotomized at levels that had clinical meaning, while controlling for the effects of clinical and demographic covariates and the percentage of time awake at night.

Results

Sample characteristics

The sample consisted of 173 patients who had stable HF (LVEF $\underline{M}$ = 32.6 + 15.2; Age $\underline{M}$ = 60. 35 ± 16.07 years; N = 60/35% women). The sample included 110 (63.6%) White, 50 (29%) African American, 7 (4%) Asian, and 10 (6%) Latino participants. The majority of participants used diuretics, including loop (n = 139/80.3%), thiazides (n = 29/16.7%) and potassium-sparing (n = 50/28.9%) drugs. Detailed information on the clinical comorbidity and additional medications used by the participants was previously reported.[9,19]

Habitual nocturia and nocturia severity

Habitual nocturia was reported by 109 (63.0%) participants. Participants reported voiding from 0 to 8 times per night ($\underline{M}$ = 1.87 ± 1.45). We classified nocturia severity as follows: 0 voids/night (Group I: N = 30/17.3%); 1–2 voids/night (Group II: N = 87/50.2%); 3 or more voids (Group III: N = 56/32.4%). (See table 1).

Clinical and demographic characteristics and nocturia

Habitual nocturia was not associated with age, gender, race, comorbidity, left ventricular ejection fraction (LVEF), NY Heart Association Classification, or use of diuretics. There was a significant overall difference between nocturia severity groups on age ($p < .05$), but there were no other clinical or demographic differences between these groups. (See table 1). There were no differences in nocturia severity based on diuretic use, frequency of dosing, or types of diuretics (data not shown).

Nocturia severity and sleep characteristics

There were statistically significant overall differences between nocturia severity groups on self-reported sleep duration ($p < .05$) and sleep efficiency ($p < .01$), but no difference in sleep latency. Participants with 3 or more episodes of nocturia (group III) had an hour less self reported sleep than those in group I. (See table 1).

There was also a significant difference in the proportion of patients reporting insomnia symptoms across levels of nocturia severity in the bivariate analyses. (See table 1). Logistic regression analysis was used to calculate the odds ratios of the effects of nocturia severity on insomnia, while controlling for covariates (age, gender, and comorbidity). Patients in Group III had a nearly 7-fold increase in the odds of reporting insomnia symptoms, but participants in Group II had no increased odds of having insomnia, compared with those who had no nocturia (Group I). (See table 2).

There were statistically significant differences across levels of nocturia severity in PSG-measured sleep duration, wake time, percent wake after sleep onset, and the percentages of stage 3–4 and REM sleep, but not sleep latency. Nocturnal wake time measured with PSG

was almost two hours in group III, approximately twice the duration of that of participants in Group I.

Nocturia severity, daytime symptoms, and functional performance

Mean levels of fatigue and sleepiness were higher and 6 MWT distance was shorter across levels of nocturia severity, but there were no overall differences in depression or self-reported physical function. (See table 1). There was also an overall increase in the proportion of patients who had a 6 MWT distance less than 1000 feet and those who reported excessive daytime sleepiness (Epworth Score > 10) across levels of nocturia severity, with 51% of the sample in Group III walking less than 1000 feet.

To evaluate the differences in fatigue, depression, sleepiness, physical function and 6 MWT distance across levels of nocturia severity, independent of the effects of age, gender, comorbidity, and the percentage of time awake after sleep onset, we conducted separate analyses of covariance with nocturia severity as the independent variable and these clinical and demographic variables and percent wake after sleep onset as covariates. There were statistically significant nocturia severity - related differences in sleepiness (p. = .009), fatigue (p = .001), self-reported physical function (p = .040), and 6 MWT (p = .047), but not depression (p = .740).

We used logistic regression to evaluate the odds ratios of the associations between nocturia severity and the dichotomized dependent variables (Epworth scale > 10), and 6 MWT distance less than 1000 feet. (See table 2). As in the ANCOVA described above, we included age, gender, comorbidity and percent wake after sleep onset as covariates. Nocturia severity (Group II) was associated with more than a four-fold increase in the odds of excessive daytime sleepiness, while the most severe nocturia (Group III) was associated with almost an 8-fold increase in the odds of excessive daytime sleepiness, compared with those with no nocturia (Group I). Patients with the highest nocturia severity (group III) had more than a five-fold increase in the odds of walking less than 1000 feet on the 6 MWT, compared with those who had no nocturia. (See table 2).

Discussion

Nocturia is common and often severe in patients with stable HF. A full third of patients awakened three or more times/night to void. The prevalence of nocturia concurrent with the PSG recording is consistent with the report of another study of HF patients,[1] but the rate of habitual nocturia is lower, and there are no comparative data on the nocturia severity as indicated by voiding frequency (such as measured in this study) in patients with HF. Prevalence rates are higher than rates reported in older adults with mixed cardiovascular disorders (55%)[46] and a population-based sample of older adults.[8] The reasons for the discrepancies between the reports of habitual nocturia and nocturia severity within our study and compared to other studies are not known, but may result from differences in measurement methods, as well as patient recall. The lower rate of habitual nocturia compared to the previously reported rate[1] may also be related to the younger age of the participants in this study, compared with other samples recruited from the population of elderly patients with HF.

We quantified large nocturia severity-related differences in sleep duration and time awake during the night by both self-report and polysomnographic measures, extending earlier studies that evaluated only self-reports.[1,2] Although there is some debate about normative values for sleep duration, typical adults obtain approximately 7 hours of sleep per night. Based on this norm, the patients with nocturia demonstrated deprivation in the overall quantity of sleep, as indicated by sleep duration, as well as deprivation of specific sleep

stages [REM and slow wave (Stages 3–4) sleep] in excess of the levels found in those without nocturia. The high proportion of patients with insomnia at all levels of nocturia severity exceeds the 10% prevalence in the U.S. population,[47] but the percentage (71.4%) is particularly striking in those with the highest nocturia severity. These findings underscore the important intersection of nocturia severity and sleep among HF patients.

Although causality cannot be determined in this cross-sectional study, decreases in slow wave sleep may lead to decreased secretion of renin and aldosterone,[10] and decreased REM sleep may lead to increased urine flow and decreased osmolality[11] – both of which may lead to nocturia. On the other hand, frequent nocturia and difficulty returning to sleep after awakening to void may also lead to sleep deprivation and further limit REM and stage 3–4 sleep. Further study is needed to evaluate the temporal relationships among these phenomena.

The importance of nocturia to HF patients is underscored by its robust independent associations with fatigue, sleepiness, and self-reported and objective measures of functional performance. As few as one to two episodes of nocturia increased the odds of excessive daytime sleepiness, while three or more episodes of nocturia increased the odds of both excessive daytime sleepiness and decrements in six minute walk test distance. In contrast with past studies that found associations between nocturia and poor mental health,[4,8] we found no differences across levels of nocturia severity in depression.

Although numerous population-based studies have found associations between nocturia and quality of life,[4,5,18] differences between the methods used in these studies and the current project preclude direct comparison of the results. It is possible that our findings are not unique to HF but reflect relationships found in the general population. However, the high burden of daytime symptoms and functional performance among HF patients suggests that any additional factors that increase this burden, such as nocturia, are meaningful in this vulnerable group of patients and should be a focus of assessment and treatment.

Although there were differences in sleep duration, sleep efficiency and percentage wake after sleep onset based on nocturia severity, decrements in sleep did not explain the differences in fatigue, sleepiness or functional performance found across levels of nocturia severity. Although one explanation might be that nocturia severity and the symptom and functional decrements are a consequence of underlying HF-related pathophysiology, the absence of differences in LVEF, comorbidity, or New York Heart Association classification across levels of nocturia severity do not support this explanation. Further study is needed to evaluate the reasons for the high symptom and functional burden associated with nocturia severity

Strengths of this study were inclusion of a clinically stable, demographically heterogeneous group of HF patients and the use ambulatory polysomnographic sleep measurement that enabled quantification of sleep characteristics, including sleep stages, in participants' home environments. While self-report does not always accurately reflect objective findings due to the limitation of patient recall, patient perceptions are important to symptom interpretation and clinical assessment, and therefore provide an important perspective. The overall shorter sleep duration elicited with self-report compared to PSG may reflect to some extent the intrusive effects of the sleep recording.

Although comorbidity was not associated with nocturia severity, it is possible that chronic conditions that were not included in the Charlson Comorbidity index (e.g., prostatic hypertrophy) and developmental changes (e.g., menopause) may contribute to nocturia and poor sleep. Although the use of specific diuretics or the frequency of dosing was not

associated with nocturia, we did not have complete information on dosage or precise timing --- both of which may contribute to nocturia.

Future longitudinal experimental studies are needed to explain the causal directions and mechanisms underlying the findings of this study. Use of voiding diaries, assessment of fluid intake, and detailed information on medication usage is likely to be useful in understanding the nature of nocturia and related differences in sleep, fatigue, sleepiness, depression and daytime function.

It is possible that behavioral or pharmacological interventions that improve sleep may also improve nocturia. Manipulation of diuretic dosage and timing to reduce nocturia severity may improve sleep, as well as fluid overload among HF patients, but the effects of this treatment on nocturia and sleep have been under-studied. From a clinical perspective, the findings of this study suggest the importance of eliciting nocturia severity among HF patients, as it is readily elicited and may be a cue to the presence of poor sleep, fatigue, sleepiness and poor functional performance.

Future research is needed to better understand the likely complex and multivariate factors that contribute to nocturia severity in HF patients, the direction of the relationship between nocturia severity, sleep, daytime functional performance, and daytime symptoms, including fatigue and sleepiness, and the effects of treatment.

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Table 1

Comparison of clinical, demographic, sleep, and symptom and functional performance variables on nocturia severity

N = 173	Group I (No Nocturia) (n = 30/17.3%)	Group II (Nocturia 1–2/night) (n = 87/50.2%)	Group III (Nocturia 3 or more/night) (n = 56/32.4%)
	M (SD)/N (%)	M (SD)/N (%)	M (SD)/N (%)
Demographic Variables			
Age (Years) *	53.70 (16.04)	63.38 (14.89)	59.20 (16.88)
Gender			
Female	13 (43.3)	31 (35.6)	16 (28.6)
Male	17 (56.7)	56 (64.4)	40 (71.4)
Race			
White	21 (70.0)	59 (67.8)	30 (53.6)
Minority	9 (30.0)	28 (32.2)	26 (46.4)
Clinical Variables			
LVEF < 45%	26 (86.7)	64 (73.6)	41 (75.9)
New York Heart Functional Class	2.37 (0.72)	2.51 (0.63)	2.45 (0.71)
II	21(70.0)	47 (54.1)	32 (57.6)
III	6 (20.0)	35 (40.2)	20 (35.7)
IV	3 (10.0)	5 (5.7)	4 (7.1)
Body Mass Index	29.68 (6.45)	30.90 (8.32)	31.01 (8.42)
Comorbidity	2.27 (1.79)	2.54 (1.61)	2.39 (1.20)
Use diuretics	22 (81.5)	75 (86.2)	48 (85.7)
Self-Reported Habitual Sleep Characteristics			
Sleep latency (min.)	21.70 (15.14)	27.80 (28.08)	35.51 (38.48)
Sleep duration (min.) *	421.20 (80.4)	393.60 (99.6)	358.80 (106.20)
Sleep efficiency (%) **	87.72 (14.05)	81.38 (15.93)	73.76 (20.22)
Insomnia symptoms **	10 (33.3)	37 (43.0)	40 (72.2)
Polysomnographic Characteristics			
Sleep Duration (min.) *	335.72 (99.04)	336.12 (84.98)	297.83 (108.34)
Sleep Latency (min.)	36.57 (53.89)	26.45 (29.00)	32.50 (31.58)
Wake time (min.) **	59.5 (45.08)	105.59 (69.4)	117.56 (78.39)
% Wake after sleep onset **	15.11 (10.99)	13.99 (5.92)	28.65 (17.99)
% Stage 1	19.23 (7.21)	21.09 (8.89)	19.28 (7.69)
% Stage 2	43.38 (11.81)	39.34 (11.24)	37.83 (12.92)
% Stage 3–4 **	8.83 (7.62)	4.97 (5.78)	4.15 (5.20)
% Stage REM *	13.40 (6.46)	11.05 (5.18)	10.08 (6.75)
Daytime Symptoms and Functional Performance			

N = 173	Group I (No Nocturia) (n = 30/17.3%)	Group II (Nocturia 1–2/night) (n = 87/50.2%)	Group III (Nocturia 3 or more/night) (n = 56/32.4%)
	M (SD)/N (%)	M (SD)/N (%)	M (SD)/N (%)
SF 36 Physical Function Subcale	23.33 (2.39)	22.73 (2.13)	22.29 (2.07)
Six Minute walk (feet) *	1162.46 (476.70)	985.13 (435.16)	905.03 (411.50)
Walk less than 1000 feet *	7 (23.3)	35 (40.2)	29 (51.8)
Depressive Symptoms	17.57 (10.60)	16.22 (11.12)	17.84 (11.17)
Depressed (CESD > 16	14 (46.2)	38 (43.2)	27 (48.2)
Global Fatigue **	28.51 (14.89)	26.83 (13.98)	35.08 (14.27)
Daytime Sleepiness *	6.37 (2.89)	8.32 (3.94)	9.32 (5.20)
Sleepy (ESS > 10) *	3 (12.5)	21 (26.2)	19 (40.4)

* p < .05;

** p< .01;

*** p < .001,based on analysis of variance (continuous variables) or chi-square (categorical variables)

Group I = No nocturia; Group II = One or two voids per night; Group III = Three or more voids/night.
LVEF= left ventricular ejection fraction
CESD = Centers for the Epidemiological Studies of Depression Scale

Table 2

Odds ratios for the associations between nocturia severity and insomnia symptoms, excessive daytime sleepiness (Epworth Score > 10) and Six Minute Walk distance less than 1000 feet with 95% CI (N = 173)

	OR	CI	P-value
Insomnia Symptoms [#]			
Group II (1–2 voids/ night)	1.76	0.70–4.42	.230
Group III (3 or more voids/night)	6.52	2.35–18.09	<001
Age	0.99	0.97–1.01	.404
Gender	2.15	1.06–4.33	.033
Comorbidity	1.19	0.95–1.49	.125
Excessive Daytime Sleepiness [@] **(Epworth scale > 10)** **Nocturia severity**			
Group II (1–2 voids/night)	4.63	1.14–16.37	.031
Group III (3 or more voids/night)	8.61	1.99–30.09	.003
Age	0.99	0.96–1.01	.352
Gender	1.17	0.55–2.49	.681
Comorbidity	1.33	1.04–1.70	.023
WASO%	0.99	0.96–1.10	.296
Six Minute Walk < 1000 feet [@] **Nocturia severity**			
Group II (1–2 voids/night)	2.22	.70–6.99	.175
Group III (3 or more voids/night)	5.99	1.69–21.54	.006
Age	1.04	1.02–1.07	.001
Gender	7.10	3.09–16.32	.000
Comorbidity	1.38	1.09–1.76	.009
WASO%	1.01	0.98–1.03	.679

Reference group: No nocturia (group I)

[#]Covariates: age, gender, comorbidity (Charlson Comorbidity Index)

[@]Covariates: age, gender, comorbidity (Charlson Comorbidity Index, WASO%)

WASO% = percent wake after sleep onset