ORI G I N A L A R T I C L E

Relationship between nocturnal polyuria and non-dipping blood pressure in male patients with lower urinary tract symptoms

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Objective: The aim of the present study was to examine factors of nocturnal polyuria and blood pressure variability in male patients with lower urinary tract symptoms (LUTS).
Methods: Two hundred and forty-two male patients with LUTS who were treated recorded frequency volume charts. We investigated their urinary condition and characteristics, medical history, and medications were investigated. Thirty-four of these patients underwent ambulatory blood pressure monitoring (ABPM) for 24 hours to evaluate blood pressure variability.
Results: In the present study, 194 patients (80.2%) had nocturia and 136 (56.2%) had nocturnal polyuria (NP). Among patients with nocturia (≥2 voids/night), 130 (67.0%) had nocturnal polyuria, and 26 of those with nocturia (13.4%) had reduced functional bladder capacity. The use of 2 or more antihypertensive medications was significantly higher in the NP than non-NP group (22.8% vs. 12.3%; P = .035). Significantly more patients in the NP group had non-dipping blood pressure (P = .037). Non-dipping blood pressure was considered a potential factor for NP.
Conclusion: We suggest that treatment of non-dipping blood pressure may improve NP.

KEYWORDS
ambulatory blood pressure monitoring, lower urinary tract symptoms, nocturia, polyuria

INTRODUCTION

Lower urinary tract symptoms (LUTS) are storage, voiding, and postmicturition symptoms affecting the lower urinary tract. The prevalence of LUTS increases with age, leading to various problems in patients' daily lives. LUTS also place a heavy burden on the medical economy. Japanese epidemiological surveys have identified nocturia as one of the most prevalent symptoms of LUTS.1 Nocturia is defined as the need to wake up 1 or more times at night to void.[2] It was reported that nocturia is associated with quality of life,[3] the risk of falls, fractures and mortality4 Therefore, nocturia is an important symptom for clinicians.

The causes of nocturia include bladder storage disorders, sleep disorders, and nocturnal polyuria (NP). Among these factors, NP is considered the largest contributor to nocturia.1,5–7 Treatment for nocturia has often targeted storage disorders. Therefore, nocturia that persists even after initiating treatment for voiding disorders should be further investigated for internal causes, such as NP. There are a few evidence-based epidemiological studies on NP.[1,4]

A relationship between nocturia and non-dipping blood pressure has been already indicated.8 It was also reported that acute sleep deprivation induces higher nocturnal blood pressure, natriuresis, and osmotic dieresis.[9] However, no study has investigated the relationship between NP and blood pressure variability. We hypothesized that NP and blood pressure variability are related. In the present study we investigated the factors contributing to NP, and the relationship between NP and blood pressure variability in male patients with LUTS who were treated.

METHODS

This study was conducted from April 2015 to July 2017, and was approved by the Institutional Ethics Committee of Iwate Medical University.

Among 256 male patients with LUTS who were treated,patients with polyuria (daily urine volume ≥40 mL/kg), a history ofprostate surgery within the 6 months before study entry, or those who were self-catheterized were excluded. This left 242 patients for analysis. Patient characteristics, including age, body mass index (BMI), medical history, and medications, were determined from medical charts. Patients recorded a frequency volume chart for 3 days at home, and the mean for those 3 days was calculated and assessed.

The NP index (NPI) was calculated as the volume of nocturnal urine/24-hour voided volume, where volume of nocturnal urine is defined as the volume voided between bedtime and the hour of rising plus the first morning void. Functional bladder capacity (FBC) is the maximum voided volume. Reduced FBC was defined as the maximum voided volume <4 mL/kg × body weight.10 NP was defined as NPI >0.33.[3]

Informed consent for ambulatory blood pressure monitoring (ABPM) was obtained from 34 male patients of the patients included in the present study to investigate the relationship between NP and

blood pressure variability. These 34 patients were monitored every 30 minutes for 24-hours using portable automatic blood pressure monitors (TM-2433; A&D, Tokyo, Japan).

Data were assessed according to the guidelines for the clinical use of 24-hour ABPM (Japanese Circulation Society [JCS] 2010).[11] "Extreme dipper", "dipper", and "non-dipper blood" pressures were defined as declines in nocturnal systolic blood pressure (SBP) of ≥20%, 10%–19%, and 0%–9%, respectively. "Riser" blood pressure was defined as increases in nocturnal SBP. Patients classified as extreme dippers and dippers were grouped as "dipping", whereas non-dippers and risers were grouped as "non-dipping".

The significance of differences between the NP and non-NP(NNP) groups was analyzed using the Chi-squared test and Wilcoxon signed-rank test. All statistical analyses were performed using JMP11 statistical software (SAS Institute Japan, Tokyo, Japan), with $P < .05$ considered significant.

RESULTS

Of the 242 male patients with LUTS who underwent treatment, 194 patients (80.2%) had nocturia (nocturnal frequency ≥2). Of the patients with nocturia, 26 (13.4%) had reduced FBC and 130 (67.0%) had NP (Figure 1). The rate of NP increased as nocturia was more frequent (Figure 2).

The patient characteristics and frequency volume charts for the NP and NNP groups are given in Table 1. The mean age of patients tended to be higher in the NP than NNP group, but the difference was not significant. No significant differences were found in LUTS diseases and complications between the 2 groups. There were significantly more patients using 2 or more antihypertensive medications (including combination drugs) in the NP than NNP group (22.8% vs. 12.3%; $P = .035$). There were no significant differences in FBC and urine volume per day between the 2 groups.

The characteristics of the patients who underwent ABPM are given in Table 2. Waking and sleeping SBP in the NP and NNP groups are shown in Figure 3. There was no significant difference of the decrease in mean SBP in the NP and NNP groups (4.2% vs 10.2%; $P = .35$).

Blood pressure variability in both groups is given in Table 3. There were significantly more patients with non-dipping blood pressure in the NP than NNP group ($P = .037$, Chi-squared test).

DISCUSSION

Sometimes patients continue to have complaints of nocturia despite being treated for LUTS. In the present study, 67.0% of patients with nocturia had NP, as reported previously in epidemiological studies.12,13 In addition, the prevalence of patients with reduced FBC was low (13.4%) in the present study. This indicates that bladder storage disorders were adequately treated in patients with LUTS. We suggest that an approach for NP may be more appropriate for the treatment for nocturia. NP is attributed to hypertension,14–20 the accumulation of water in the body during the day, overhydration, diabetes mellitus,

congestive heart failure, anomalous peripheral venous drainage, excessive salt intake, hypoalbuminemia21 and sleep apnea syndrome(SAS).[22]

In the present study, there were significantly more patients who used 2 or more antihypertensive medications (including combination drugs) in the NP than NNP group (P = .035). It was considered that NP was related to resistant hypertension. According to an international database,[23] SBP decreases approximately 13% at night. In the Ohasama study, among 1542 general residents (mean age 61.0 years), 27% of residents were non-dippers and 7% were risers.[14]

There is no report regarding the relationship between NP and blood pressure variability. In the present study, the frequency of patients with non-dipping blood pressure was higher than in the general population. Furthermore, there were significant more patients with non-dipping blood pressure in the NP than NNP group.

There are numerous factors and mechanisms for non-dipping blood pressure, for example sodium retention and compensatory nocturnal natriuresis,[15] congestive heart failure, chronic renal failure, and so on. Several studies have investigated the overactive sympathetic nervous system at night caused non-dipping blood pressure.[24–26] Non-dipping blood pressure is also related to medications. For example, the effects of antihypertensive drugs may not continue throughout the night. Calcium antagonists, particularly amlodipine, increase nocturnal urine volume by selectively dilating renal afferent arterioles. Olmesartan normalizes non-dipping blood pressure.[27] It was reported that taking an α1 receptor blocker at night significantly decreased nocturnal blood pressure in non-dippers and risers.[28,29] Therefore, selection of antihypertensive medications is considered very important for patients with NP. The results of the present study suggest a relationship between non-dipping blood pressure and NP. However, we cannot confirm whether non-dipping blood pressure is a cause or result of NP, because there are many causes of and mechanisms underlying non-dipping blood pressure. The present study has several limitations. First, because frequency volume charts were recorded by the patients themselves, we cannot exclude the possibility of measurement errors. The second limitaton of the study is the reproducibility of ABPM. For example, 1 study found that approximately 70% of patients reported the same blood pressure patterns over 2 days.[30] Blood pressure may rise by light stimulation when patients go to toilet during the night or when sleep is disturbed due to the cuff pressure. Third, we may not have counted the patients with SAS correctly, because only those patients with a diagnosis of SAS were counted: we did not include patients with undiagnosed SAS. Polysomnography is necessary to diagnose SAS. Finally, blood pressure variability is affected by medication compliance and the time when patients take antihypertensive medications (e.g. in the morning or at night). We consider that further investigation in a larger sample size is needed.

CONCLUSION

Non-dipping blood pressure was considered to be a potential factor for NP. We suggest that treatment for non-dipping blood pressure may improve NP.

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Conflict of interest

The authors declare no conflicts of interest.

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