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INFLUENCE OF NOCTURNAL POLYURIA TO NOCTURIA IN PARKINSON'S DISEASE

Hypothesis / aims of study

Parkinson's disease is a progressive neurodegenerative disease, mainly characterized by the loss of dopamine neurons in the substantia nigra pars compacta, culminating in motor symptoms.

In addition to motor symptoms, there are a variety of non-motor symptoms associated with Parkinson's disease (PD).

Lower urinary tract dysfunction such as daytime/nocturnal frequency, urgency and urge incontinence is a common non-motor dysfunction of PD.

In LUTD, nocturia (nocturnal frequency) impairs quality of life, disturbs sleep, and may result in falling, fracture, depression and cognitive impairment.

Detrusor overactivity and decrease in bladder volume occur both in untreated and treated PD and are considered to be one of the causes of nocturia.

However, recent report show that increased nocturnal polyuria has a greater role in the pathogenesis of nocturia in older patients, and little is known about the possible contribution of nocturnal polyuria to nocturia in PD.

Thus, we evaluate the association between nocturnal frequency and nocturnal polyuria, and the pathophysiology of nocturnal polyuria in PD.

Study design, materials and methods

Forty-six consecutive PD patients (mean age, 69) and forty-two healthy older volunteers (mean age, 75) were recruited; those with other conditions that might have influenced urinary function were excluded. All studied subjects were evaluated using a urinary questionnaire and bladder diary, 30 patients with PD were also evaluated using urodynamic studies, and serial plasma and urine samples were collected over 24 hours to evaluate the circadian levels of melatonin, arginine vasopressin (AVP), urine specific gravity and so on in 20 patients with PD.

Results

Twenty-one patients with PD (45.7%) and 10 healthy subjects (23.8%) complained of nocturia, and patients with PD were larger than healthy subjects significantly ($P < 0.05$). Twenty-seven patients with PD (58.7%) and 14 healthy subjects (33.3%) had nocturnal polyuria, and patients with PD were larger than healthy subjects significantly ($P < 0.05$). Ten patients with PD had nocturnal polyuria not but nocturia. Average daytime and nocturnal voided volume in patients with PD were smaller than those in healthy subjects significantly (152.0ml vs 202.6ml, $P < 0.001$; 203.4 vs 278.6ml, $P < 0.001$, respectively). Average daytime and nocturnal voided volume in patients with PD were smaller than those in healthy subjects significantly (152.0ml vs 202.6ml, $P < 0.001$; 203.4 vs 278.6ml, $P < 0.001$, respectively). Average proportion of urine excretion at night (nocturnal voided volume /total 24hr voided volume x 100%) in patients with PD was larger than that in healthy subjects significantly (35.3% vs 28.8%, $P < 0.01$). Urodynamic studies showed abnormal findings in the storage phase in all studied patients, with detrusor overactivity (DO) and increased bladder sensation without DO in 90.0% and 10.0% of patients, respectively. All studied patients had abnormal circadian rhythm of melatonin secretion, AVP secretion and urine specific gravity.

Interpretation of results

Patients with PD had nocturia, which might be due to not only decreased in bladder volume by detrusor overactivity or increased bladder sensation but also nocturnal polyuria with abnormal circadian rhythm of melatonin and AVP.

Concluding message

This is the first reports to evaluate the association between nocturnal frequency and nocturnal polyuria, and the pathophysiology of nocturnal polyuria in PD. In patients with PD, not only decreased nocturnal bladder capacity but also nocturnal polyuria may have a greater role in the causes of nocturia symptoms independently. And the pathophysiology of nocturnal polyuria in PD may be related with abnormal circadian rhythm of melatonin and AVP.

Disclosures

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