Original Article: Clinical Investigation

Urodynamic efficacy of fesoterodine for the treatment of neurogenic detrusor overactivity and/or low compliance bladder

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Abbreviations & Acronyms
- BVE = bladder voiding efficiency
- CIC = clean intermittent self-catheterization
- DO = detrusor overactivity
- FDV = first desire to void
- FIC = first involuntary contraction
- FVC = frequency volume chart
- ICIQ-SF = International Consultation on Incontinence Questionnaire—short form
- IPSS = International Prostate Symptom Score
- KHO = King’s Health Questionnaire
- LCB = low compliance bladder
- MCC = maximum cystometric capacity
- NDO = neurogenic detrusor overactivity
- OAB = overactive bladder
- OABSS = overactive bladder symptom score
- Pdet = detrusor pressure
- PdetQmax = detrusor pressure at maximum flow rate
- PVR = postvoid residual urine volume
- Qave = average flow rate
- Qmax = maximum flow rate
- SD = standard deviation
- SF-36 = 36-Item Short Form Health Survey
- UDS = urodynamic study
- V-UDS = video urodynamic study

Objective: To examine the urodynamic effects of fesoterodine on neurogenic detrusor overactivity and/or low compliance bladder.

Methods: A total of 77 patients (52 men, 25 women; aged 61.6 ± 20.3 years) were given fesoterodine 4–8 mg/day and prospectively followed for 12 weeks. The primary end-point variable was change in the maximum cystometric capacity on urodynamic study. The secondary end-point was to assess the number of patients whose neurogenic detrusor overactivity disappeared, and the changes in the urodynamic parameters, lower urinary tract symptoms questionnaires and the 3-day frequency volume chart parameters after the treatment.

Results: A total of 13 patients (16.9%) withdrew because of adverse events (dry mouth or blurred vision), and four patients dropped out for unknown reasons. Finally, 60 patients completed the study. Bladder capacity at first desire to void, maximum cystometric capacity and bladder compliance increased by 29.2 mL, 79.9 mL and 22.2 mL/cm H2O, respectively, showed statistical significance (P = 0.026, P < 0.001 and P < 0.001). Neurogenic detrusor overactivity disappeared in 12 of 51 patients (23.5%), and a significant increase was observed in bladder capacity at first involuntary contraction (P < 0.001), and a significant decrease was observed in maximum detrusor contraction (P < 0.001). In patients with low compliance bladder (with detrusor underactivity without neurogenic detrusor overactivity; n = 9), maximum cystometric capacity and bladder compliance increased significantly (P = 0.003 and P = 0.006, respectively). Overactive bladder symptom score, International Consultation on Incontinence Questionnaire–Short Form, most items of King’s Health Questionnaire, and the number of urgency episodes and leaks in a day decreased significantly after treatment.

Conclusions: Fesoterodine seems to be a valid treatment option for neurogenic detrusor overactivity and/or low compliance bladder in neurogenic bladder patients.

Key words: anticholinergic, detrusor overactivity, fesoterodine, low compliance, neurogenic bladder.

Introduction

NDO is recognized when DO is accompanied by a relevant neurological condition. The main concern regarding patients with neurogenic bladder is renal damage attributable to high detrusor storage and/or voiding pressures. These high detrusor pressures can be caused by DO or LCB. NDO might also cause urinary incontinence and deteriorate quality of life. Thus, the main therapeutic goal for NDO and LCB might be achieving a low-pressure reservoir and improvement in the patient’s quality of life.

Anticholinergics alone or in combination with CIC is the mainstay therapy for NDO or LCB, and assessment with UDS is useful both for the diagnosis and treatment evaluation of NDO. There have been some reports on antimuscarinic drugs for the treatment of NDO or LCB, which reported an increase in bladder capacity and reduction in detrusor pressure, and an improvement in urinary incontinence.

Previously, we reported the efficacy and safety of tolerodine for the treatment of NDO and/or LCB. Fesoterodine acts functionally as a prodrug. 5-HMT is the active metabolite of...
both tolterodine and fesoterodine, and this active metabolite has been reported to be responsible for the antimuscarinic activity.\textsuperscript{10} The efficacies of fesoterodine for the treatment of idiopathic OAB have been reported\textsuperscript{11–14} in the treatment with fesoterodine, flexible dosing strategies involving adjustment of doses of 4 and 8 mg were used to optimize the therapeutic balance between efficacy and tolerability.\textsuperscript{11–14}

The aim of the present study was to investigate the effects of fesoterodine on NDO and/or LCB with neurogenic bladder based on V-UDS, FVC and lower urinary tract symptom questionnaires.

**Methods**

This study was a single-arm prospective study to evaluate the effects of fesoterodine (4–8 mg/day) on NDO and LCB for 12 weeks. This study was carried out in accordance with the Declaration of Helsinki, registered (UMIN000038269) and was approved by the institutional review board of Dokkyo Medical University, Mibu, Tochigi, Japan (C-272-2). All patients signed informed consent before the treatment. Patients with NDO or LCB in a stable condition for >6 months were included (Table 1). Exclusion criteria were patients having an indwelling catheter, acute urinary tract infection and a history of bladder augmentation. Patients stopped taking medications that might influence voiding function (antimuscarinic drugs, antihistamines, \(\alpha\)- and \(\beta\)-adrenoceptor agonists and antagonists) for >2 weeks. After the washout period, the patients received fesoterodine (4 mg/day). The patient-reported level of satisfaction was evaluated as very satisfied, somewhat satisfied, neither satisfied nor satisfied, or dissatisfied, and was assessed at 4 and 12 weeks of treatment.\textsuperscript{15} If the response was favorable, the doses of fesoterodine (4 mg/day) remained the same, and if the patients were not satisfied, the dose of fesoterodine was increased to 8 mg/day after 4 weeks of treatment.\textsuperscript{14}

Patients recorded a 3-day FVC, and underwent a V-UDS at baseline and at month 3. Statistical significance of changes in parameters between baseline and after the therapy was assessed using the Wilcoxon matched pairs signed-rank test. The level of \(P < 0.05\) was considered to show statistical significance. In the previous study with tolterodine, the sample size was set as 45 patients, based on the ability to detect the deference of 65 mL of MCC at week 12.\textsuperscript{9} In the results of the study, MCC increased by >50 mL in 49% of patients. We considered that 60 patients would yield 80% power to detect such a difference, assuming a SD of 125 mL and \(\alpha\)-error of 0.05. Assuming that approximately 20% of the patients would drop out, 77 patients were considered to be required.

**Results**

Data of 77 patients (52 men, 25 women; aged 61.6 ± 20.3 years) were analyzed. The primary end-point was change from baseline to the end of treatment in the MCC in V-UDS. The secondary end-points were the number of patients whose NDO disappeared, and changes in the following parameters from baseline to the post-treatment: bladder capacity at FDV, bladder capacity at FIC, maximum detrusor pressure and bladder compliance. If patients could void, Qmax, maximum detrusor pressure and PdetQmax were evaluated. Changes in OABSS, IPSS, ICIQ-SF and KHQ,\textsuperscript{18–20} and the changes in the number and the amount of voids, and the number of daily incontinence episodes in a 3-day FVC were evaluated from baseline to 4 and 12 weeks of treatment. In the SF-36, domains of physical functioning, physical role, body pain, general health, vitality, social functioning, role emotional and mental health were evaluated, with a minimum score of 0 (worst health), and the maximum score of 100 (best health).

Adverse events were monitored throughout the study. Changes in PVR and BVE% from baseline to week 12 were calculated.
calculated FIC as equal to MCC to evaluate changes in these patients.

The OABSS, ICIQ-SF, most items of the KHQ, and the number of urgency episodes and leaks in a day decreased significantly after treatment. Therefore, fesoterodine seemed to be effective for the treatment of NDO and/or LCB in increasing bladder capacity and bladder compliance. These results seemed to be pronounced in patients with NDO.

In patients with LCB without detrusor contraction, bladder compliance was not normalized after fesoterodine treatment, although it increased with statistical significance. However, MCC increased from the baseline (188.7 mL) to the post-treatment value (300.3 mL). Therefore, we suppose that the treatment might be meaningful. For the treatment of NDO or LCB without detrusor contraction, bladder compliance should be normalized after surgery.
LCB, double dosages of antimuscarinic drugs have been reported to be necessary compared with those for patients with idiopathic OAB, which in turn might lead to more severe adverse events and, consequently, to termination of treatment. The dose of fesoterodine can be increased to tolerate adverse events and, consequently, to termination of treatment. In patients with idiopathic OAB, which in turn might lead to more severe adverse events and, consequently, to termination of treatment.

Previously, we studied the effects of 4 mg/day tolterodine ER in 46 patients with NDO or LCB. Bladder volume at FDV and MCC increased by 36.8 and 82.3 mL, both showing statistical significance (P < 0.0001). NDO disappeared in three of 32 patients with NDO; bladder volume and maximum detrusor pressure showed a significant increase (P < 0.0009), and maximum detrusor pressure showed a significant decrease (P = 0.0025). However, bladder compliance did not increase significantly.9 We could not directly compare these results between fesoterodine and tolterodine, but the improvements in urodynamic parameters might be comparable in the two treatment groups. However, the number of patients whose NDO disappeared was higher after the fesoterodine treatment (23.5%) than the tolerodine treatment (9%).

### Table 3 Urodynamic parameters at baseline and at week 12

|                         | Before | After | P-value |
|-------------------------|--------|-------|---------|
| **All patients (n = 60)** |        |       |         |
| Bladder capacity at FDV (mL) | 157.8 ± 87.9 | 187.0 ± 109.3 | 0.026 |
| MCC (mL)                 | 246.1 ± 123.46 | 326.0 ± 127.6 | <0.001 |
| Bladder compliance (mL/cm H2O) | 28.0 ± 31.8 | 50.2 ± 127.6 | <0.001 |
| **Patients with NDO (n = 51)** |        |       |         |
| Bladder capacity at FDV (mL) | 161.6 ± 73.0 | 187.6 ± 111.3 | 0.038 |
| MCC (mL)                 | 256.2 ± 116.0 | 330.5 ± 123.6 | <0.001 |
| Bladder compliance (mL/cm H2O) | 32.3 ± 32.7 | 57.8 ± 92.5 | 0.016 |
| Bladder capacity at FIC (mL) | 203.9 ± 126.3 | 289.4 ± 131.8 | <0.001 |
| Amplitude of NDO (cm H2O) | 48.1 ± 22.2 | 33.0 ± 26.6 | <0.001 |
| **Free uroflowmetry (n = 28)** |        |       |         |
| Voided volume (mL)       | 149.2 ± 137.3 | 145.9 ± 141.7 | 0.942 |
| Qave (mL/s)              | 9.3 ± 5.9 | 8.8 ± 8.3 | 0.303 |
| Qmax (mL/s)              | 15.4 ± 10.8 | 14.7 ± 14.6 | 0.974 |
| PVR (mL)                 | 14.5 ± 19.2 | 35.5 ± 38.9 | 0.008 |
| BVE (%)                  | 85.5 ± 26.8 | 72.7 ± 29.6 | 0.190 |
| **Pressure/Flow study (n = 32)** |        |       |         |
| Qmax (mL/s)              | 11.5 ± 8.4 | 13.8 ± 9.7 | 0.033 |
| Pdet at Qmax (cm H2O)    | 38.7 ± 21.0 | 36.6 ± 20.7 | 0.299 |
| Watt factor at Qmax      | 8.3 ± 3.7 | 10.0 ± 7.7 | 0.387 |
| Bladder outlet obstruction index | 15.7 ± 29.5 | 9.0 ± 31.4 | 0.058 |
| **Male (n = 18)**        |        |       |         |
| Qmax (mL/s)              | 10.4 ± 6.3 | 11.1 ± 7.2 | 0.396 |
| Pdet at Qmax (cm H2O)    | 48.4 ± 19.4 | 47.0 ± 25.9 | 0.713 |
| Watt factor at Qmax      | 9.0 ± 3.5 | 11.4 ± 9.2 | 0.545 |
| Bladder outlet obstruction index | 27.5 ± 21.8 | 24.8 ± 29.4 | 0.296 |
| **Female (n = 14)**      |        |       |         |
| Qmax (mL/s)              | 13.4 ± 10.3 | 16.9 ± 11.9 | 0.034 |
| Pdet at Qmax (cm H2O)    | 25.9 ± 18.1 | 23.9 ± 12.0 | 0.680 |
| Watt factor at Qmax      | 7.4 ± 3.9 | 7.6 ± 4.1 | 0.652 |
| **Patients with LCB without DO (n = 9)** |        |       |         |
| Bladder capacity at FDV (mL) | 136.4 ± 151.9 | 183.6 ± 103.0 | 0.385 |
| MCC (mL)                 | 188.7 ± 155.6 | 300.3 ± 154.0 | 0.003 |
| Bladder compliance (mL/cm H2O) | 4.0 ± 2.5 | 7.0 ± 3.8 | 0.006 |

Fesoterodine is not approved for neurogenic bladder. Some patients without urgency or bladder sensation cannot be precisely diagnosed as OAB. However, many kinds of anticholinergic drugs and even β3-agonists have been prescribed to patients with neurogenic bladder without urgency, or even patients with only urinary frequency or nocturia without urgency. The Japanese OAB guideline categorized OAB as neurogenic OAB and non-neurogenic OAB, and the former included spinal cord injury. Therefore, anticholinergics, including fesoterodine, can be prescribed for neurogenic bladder patients with NDO. We also explained to the institutional review board of our institution and obtained approval for use in these patients.
Table 4 OABSS, ICIQ-SF, and IPSS at baseline and at weeks 4 and 12

|                  | Before | At 4 weeks | P-value | At 12 weeks | P-value | 4 mg at 12 weeks | 8 mg at 12 weeks |
|------------------|--------|------------|---------|-------------|---------|-----------------|-----------------|
| OABSS (n = 55)   |        |            |         |             |         |                 |                 |
| Total score      | 6.7 ± 4.1 | 5.6 ± 4.0 | 0.050  | 5.3 ± 4.0   | 0.006  | 4.9 ± 3.8       | 6.2 ± 4.2       |
| Frequency score  | 0.7 ± 0.6 | 0.6 ± 0.6 | 0.109  | 0.6 ± 0.6   | 0.135  | 0.6 ± 0.5       | 0.7 ± 0.7       |
| Nocturia score   | 1.4 ± 1.1 | 1.3 ± 1.1 | 0.626  | 1.2 ± 1.1   | 0.017  | 1.2 ± 1.1       | 1.2 ± 1.2       |
| Urgency score    | 2.5 ± 1.7 | 2.1 ± 1.7 | 0.089  | 1.9 ± 1.7   | 0.011  | 1.8 ± 1.6       | 2.2 ± 1.7       |
| Urgency incontinence score | 2.1 ± 1.8 | 1.6 ± 1.8 | 0.144  | 1.6 ± 1.8   | 0.066  | 1.3 ± 1.6       | 2.1 ± 1.9       |

| ICIQ-SF (n = 55) |         |            |         |             |         |                 |                 |
| Total score      | 9.1 ± 5.4 | 7.6 ± 5.8 | 0.055  | 6.9 ± 5.5   | <0.001 | 6.0 ± 5.0       | 8.9 ± 5.6       |
| Frequency of leaks score | 2.5 ± 1.6 | 1.9 ± 1.7 | 0.003  | 1.9 ± 1.7   | <0.001 | 1.7 ± 1.6       | 2.5 ± 1.6       |
| Amount of leaks score | 2.8 ± 1.7 | 2.3 ± 1.7 | 0.102  | 1.9 ± 1.5   | <0.001 | 1.7 ± 1.4       | 2.3 ± 1.5       |
| Quality of life score | 3.8 ± 3.0 | 3.5 ± 3.2 | 0.690  | 3.1 ± 3.0   | 0.033  | 2.8 ± 2.7       | 4.2 ± 3.1       |

| IPSS (n = 54)    |         |            |         |             |         |                 |                 |
| Total score      | 10.0 ± 8.6 | 8.2 ± 7.9 | 0.133  | 8.5 ± 8.2   | 0.110  | 5.8 ± 7.0       | 12.1 ± 8.6      |
| Storage subscore | 5.8 ± 4.3 | 4.7 ± 3.9 | 0.141  | 4.5 ± 3.5   | 0.068  | 3.7 ± 2.9       | 5.7 ± 3.9       |
| Voiding subscore | 4.0 ± 4.6 | 3.3 ± 4.2 | 0.083  | 3.4 ± 4.4   | 0.266  | 2.3 ± 3.9       | 5.1 ± 4.6       |

| FVC (n = 47)     |         |            |         |             |         |                 |                 |
| No. voids/day time | 7.5 ± 2.8 | 6.6 ± 2.6 | 0.043  | 6.7 ± 2.2   | 0.011  | 6.5 ± 1.9       | 7.0 ± 2.5       |
| No. voids/night  | 1.3 ± 1.4 | 1.1 ± 1.2 | 0.322  | 1.1 ± 1.1   | 0.412  | 1.1 ± 1.2       | 1.0 ± 1.0       |
| No. urgency episodes/24 h | 2.6 ± 5.3 | 1.4 ± 2.8 | 0.061  | 1.3 ± 2.7   | 0.032  | 1.0 ± 1.8       | 1.8 ± 3.5       |
| No. leaks/24 h   | 1.5 ± 1.6 | 1.1 ± 2.1 | 0.011  | 1.0 ± 1.9   | 0.006  | 0.6 ± 1.4       | 1.4 ± 2.4       |
| Amount of leaks/24 h (mL) | 124.7 ± 191.4 | 97.3 ± 238.6 | 0.311 | 92.0 ± 198.1 | 0.425 | 53.0 ± 161.1 | 143.3 ± 223.7 |
| No. pad changes/24 h | 1.4 ± 1.6 | 1.1 ± 2.1 | 0.007  | 1.0 ± 2.0   | 0.010  | 0.7 ± 1.5       | 1.4 ± 2.4       |
| Mean voided volume (mL) | 166.6 ± 81.5 | 198.7 ± 90.0 | 0.044 | 197.6 ± 98.2 | 0.011 | 208.5 ± 107.8 | 180.8 ± 73.6 |
| Max voided volume (mL) | 286.6 ± 133.5 | 324.2 ± 149.6 | 0.101 | 311.9 ± 132.6 | 0.365 | 320.4 ± 138.5 | 300.0 ± 119.0 |

Fig. 1 Results of KHQ at baseline (straight line), and at weeks 4 (dotted line) and 12 (bold line; n = 49). Numbers on the axis of the radar chart indicate the following domains: 1. General Health Perceptions; 2. Incontinence Impact; 3. Role Limitations; 4. Physical Limitations; 5. Social Limitations; 6. Personal Relationships; 7. Emotion problems; 8. Sleep and Energy; and 9. Severity (Coping) Measures. *P < 0.05 versus baseline (Wilcoxon matched pairs signed-rank test).

OAB symptoms and incontinence also improved after fesoterodine treatment. OABSS, ICIQ-SF and KHQ decreased both at 4 weeks and at 12 weeks after fesoterodine therapy.

SF-36 domains, such as physical functioning, physical role, body pain, general health, vitality, social functioning, role emotional and mental health, did not change significantly at 12 weeks. The possible reasons could be that patients with neurogenic disorders might still be suffering in general health domains, and quality of life might not relate to the improvement in lower urinary tract dysfunction.

Mild adverse events were noted in 51.9%, and 16.9% of patients who dropped out due to adverse events, including...
one patient with urinary retention. These events occurred in 80.0% of the patients after fesoterodine 4 mg/day, and in 20.0% of those after 8 mg. The adverse events were like the reported adverse events in idiopathic OAB patients. A total of 20 patients (33.3%) were “very satisfied” or “satisfied,” and 19 (31.7%) were “somewhat satisfied” after fesoterodine treatment. Consequently, fesoterodine appeared to be effective and tolerable in patients with NDO, and in those with LCB.

A limitation of the present study was that this was a non-controlled study, because it was difficult to recruit enough neurogenic patients to provide controls in this study, and we could not obtain approval from the institutional review board to use a placebo in these patients for ethical reasons. The infusion rate of 50 mL/min seemed a little higher in some patients with NDO or LCB. As we used an infusion rate of 50 mL/min in a routine urodynamic study, we did not change it. However, the condition of urodynamic study before and after the treatment was the same.

In the present study, five patients (8.3%) had no bladder sensation. FDV cannot be evaluated in patients without bladder sensation. However, if we excluded the amount, we could not evaluate differences. Therefore, we temporarily calculated FDV as an equal amount with MCC for patients without bladder sensation.

Another limitation was that the participants had mixed types of neurogenic bladder. It would be better to carry out subanalysis in a group of causative neurogenic disorders. However, the number of these patients was limited, and it was difficult to analyze between the subgroups.

In conclusion, fesoterodine seemed to be effective for the treatment of NDO and/or LCB.

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Conflict of interest

None declared.

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