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ORIGINAL ARTICLE

# Therapeutic effects of detrusor botulinum toxin A injection on neurogenic detrusor overactivity in patients with different levels of spinal cord injury and types of detrusor sphincter dyssynergia

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**Study design:** The study was designed as a single-arm clinical trial.
**Objectives:** To investigate the therapeutic effects of detrusor botulinum toxin A (BoNT-A) treatment in patients with different levels of spinal cord injury (SCI) and types of detrusor sphincter dyssynergia (DSD).
**Setting:** Patients with chronic suprasacral SCI and neurogenic detrusor overactivity (NDO) were treated with 200-U BoNT-A detrusor injection in a tertiary university hospital in Taiwan.
**Methods:** Outcome assessment of urodynamic parameters and therapeutic satisfaction were performed at 3 and 6 months among patients with different demographics, SCI levels and types of DSD. The treatment outcomes were assessed by changes in Urogenital Distress Inventory 6-item short form (UDI-6), quality of life (QoL) index and detrusor pressure at maximum flow rate (Pdet.Qmax).
**Results:** A total of 38 patients with a mean age of 40.1 ± 12.4 years and median duration of SCI of 10.3 years were enrolled. Satisfactory response was reported in 23 (60%) patients. Significant improvements in the UDI-6 and QoL were reported and significant increases of cystometric bladder capacity and post-void residual were noted in overall patients after treatment. Patients with different clinical demographics and urodynamic parameters had similar treatment outcomes and UDI-6 scores. In 11 patients receiving repeat injections of 300 U of BoNT-A after failure of a previous 200 U injection, the treatment outcomes were not significantly different, except that the PdetQmax at 6 months were higher in the group that received 200 U of BoNT-A.
**Conclusion:** Patients with different SCI levels or DSD types had similar treatment outcomes after detrusor 200 U BoNT-A injections for NDO.
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**Keywords:** spinal cord injury; botulinum toxin; neurogenic bladder; urodynamics

## Introduction

Detrusor overactivity (DO) and detrusor sphincter dyssynergia (DSD) in patients with spinal cord injury (SCI) constantly impair quality of life (QoL), and often pose a threat for the upper urinary tract. The presence of DSD is associated with complete injuries, elevated intravesical pressure and upper urinary tract complications.[1] Patients with DSD usually have urinary incontinence with or without large postvoid residual (PVR) requiring clean intermittent catheterization (CIC) or indwelling Foley catheter. These urological complications usually result in a low QoL and cause more serious complications such as autonomic dysreflexia, urinary tract infection and upper urinary tract deterioration.

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Oral antimuscarinic drugs are the first-line treatment for neurogenic DO (NDO) to reduce urinary incontinence, however, the results are often disappointing because of insufficient dosing.[2] Increasing the dosage can cause troublesome side-effects, such as dry mouth, constipation, dyspepsia, changes in visual accommodation, dizziness and somnolence, which reduce patient compliance. Recently, botulinum toxin A (BoNT-A) emerged as an alternative method for the management of urological complications due to SCI. Detrusor injection of 200–300 U of Botox (Allergan, Irvine, CA, USA) can decrease detrusor contractility, improve bladder compliance and restore urinary continence.[3] However, detrusor underactivity and urinary retention occur in about 70% of patients, requiring periodic CIC, and urinary tract infection could become a *de novo* problem.[4]

Currently, there is no clear consensus about the optimal use of this innovative treatment in clinical practice. Issues of

optimal dose, number and location of injections, type of cystoscope and anesthesia required depending on disease entities, timing of repeat injection and safety profile could have an important impact on therapeutic outcomes and have been discussed extensively.[5] There was no study investigating patient demographic factors and their influence on the therapeutic results after detrusor BoNT-A injection in SCI patients. The purpose of this study was to investigate the therapeutic effects of detrusor BoNT-A treatment in SCI patients with different levels of SCI or types of DSD.

## Methods

A total of 38 patients with chronic suprasacral SCI were enrolled for detrusor BoNT-A injections to treat urinary incontinence. All patients underwent video urodynamic studies before enrollment, and all patients were proven to have urodynamic DO with or without DSD. DSD was diagnosed as the presence of increased urethral sphincter activity at uninhibited detrusor contractions or during the voiding phase.[6] Detrusor BoNT-A injection was recommended mainly to increase the bladder capacity, decrease detrusor pressure and improve urinary incontinence in these patients. The institutional review board and ethics committee of the hospital approved this study. All patients provided written informed consent before electing BoNT-A treatment.

Patients with urinary tract infection were appropriately treated before they were scheduled for BoNT-A injection. All patients underwent BoNT-A injection in the operating room with/without general anesthesia or spinal anesthesia to eliminate the occurrence of autonomic dysreflexia or hyperreflexia of extremities during detrusor injections. Botox (200 U) was dissolved in 20 ml of normal saline and was injected into 40 detrusor sites, sparing the trigone. After the BoNT-A injections, the patients were monitored in the outpatient department at monthly intervals, thereafter, and video urodynamic studies were performed at 3 and 6 months after treatment. Patients who had a PVR of greater than 150 ml were advised to perform CIC, in addition to voiding by abdominal tapping or the Credé or Valsalva maneuver. Outcome assessment of urinary storage and voiding conditions and satisfactory response to treatment were performed at 3 and 6 months after BoNT-A treatment.

Patient perceptions of lower urinary tract dysfunction were assessed using the validated Urogenital Distress Inventory 6-item short form (UDI-6) questionnaire[7] and QoL index adapted from the International Prostate Symptom Score.[8] The UDI-6 was scored at baseline, 3 and 6 months after administration of BoNT-A and the changes from baseline were assessed. The urodynamic parameters at baseline and 6 months after treatment were also compared to demonstrate the urodynamic effects of BoNT-A. In addition to UDI-6, the treatment outcomes were also assessed according to changes of the detrusor pressure at maximum ow rate (Pdet.Qmax). Patients who had a reduction of QoL index of two or more points were considered responders, otherwise, the treatment was considered to have failed.

The pretreatment clinical data collected from the medical records included age, gender, level of SCI, completeness of injury, DSD types, bladder compliance, Pdet.Qmax and PVR. Completeness of SCI was classified according to the American Spinal Injury Association impairment scale.[9] The level 'A' represented complete SCI, with total motor and sensory loss below the injury level, whereas levels 'B'–'E' indicated incomplete injuries, with preservation of perianal sensation ('B') or residual motor ('C', 'D' and 'E') function, with 'D' and 'E' representing useful motor function with ambulatory capability.

DSD was classified into three types according to the Blaivas classification.[6] Type 1 DSD was characterized by a crescendo increase in sphincter activity that reached its maximum at the peak of detrusor contraction. As the detrusor pressure began to decline, sudden complete external sphincter relaxation occurred. Voiding occurred only during the down slope of the detrusor pressure as the sphincter relaxed. Type 2 DSD was characterized by clonic contractions of the external urethral sphincter, interspersed throughout the detrusor contraction. These patients usually voided with an interrupted spurting stream. In type 3 DSD, the external urethral sphincter contraction persisted throughout the entire detrusor contraction. These patients voided with an obstructed stream or could not void at all.

When patients felt the therapeutic effect of BoNT-A had gone after the first 200 U injection, a repeat BoNT-A injection of 300 U was given. The time gap between the first 200 U injection and repeat injection of 300 U was more than 6 months. This time gap may minimize the cumulative Botox effect of the first dose. The treatment outcomes were also compared between these two treatment doses in 11 patients.

Paired Student's *t*-tests and analysis of variance were used for statistical analyses of the urodynamic and clinical outcome parameters. Multiple measurement analysis was used to compare the changes of urodynamic parameters and UDI-6 after BoNT-A injections between subgroups. $P<0.05$ was considered statistically significant.

## Results

A total of 38 patients including 21 women and 17 men were enrolled from February 2007 to March 2009. The mean age was 40.1 ± 12.4 years (range, 20–72). The median duration of SCI was 10.3 years (range, 1–35). All patients had urodynamically proven DO and different types of DSD. Urinary incontinence was noted in all patients, with difficult urination in 4 patients and without difficult urination in 34 patients. Autonomic dysreflexia was also noted in eight patients. During detrusor Botox injection, 30 patients received general anesthesia, 6 received no anesthesia and 2 received spinal anesthesia.

A satisfactory response was reported by 23 (60.5%) patients. Significant improvements in the UDI-6 scores and QoL index were reported at 3 months and at 6 months in overall patients after detrusor BoNT-A injection (Table 1). The urodynamic parameters at baseline, 3 and 6 months are listed in Table 1. Cystometric bladder capacity and PVR

**Table 1** Changes in urodynamic parameters and quality of life indexes in patients with detrusor BoNT-A injection

| | *Qmax (ml s$^{-1}$)* | *PVR (ml)* | *CBC (ml)* | *Pdet.Qmax (cm $H_2O$)* | *UDI-6* | *QoL-I* |
|---|---|---|---|---|---|---|
| Baseline | 5.2 ± 5.7 | 138 ± 102 | 214 ± 115 | 40.7 ± 21.3 | 11.3 ± 3.65 | 4.47 ± 1.37 |
| 3 months | 3.3 ± 4.7 | 293 ± 203 | 375 ± 177 | 26.1 ± 17.2 | 7.72 ± 4.01 | 2.31 ± 1.23 |
| 6 months | 2.0 ± 2.9 | 263 ± 183 | 306 ± 186 | 29.4 ± 25.5 | 8.46 ± 4.29 | 2.89 ± 1.49 |
| *P-value* | | | | | | |
| BL vs 3 months | 0.049 | <0.001 | <0.001 | 0.001 | <0.001 | <0.001 |
| BL vs 6 months | 0.004 | <0.001 | 0.006 | 0.064 | 0.001 | <0.001 |

Abbreviations: BL, baseline; BoNT-A, Botulinum toxin A; CBC, cystometric bladder capacity; Pdet.Qmax, detrusor pressure at maximum ow rate; PVR, post-void residual; UDI-6, urogenital distress inventory 6-item short form; Qmax, maximum flow rate; QoL-I, quality of life index.

**Table 2** Multivariate analysis of factors and UDI-6 at different time points in SCI patients with detrusor BoNT-A injection

| *Variable* | *Cases* | *UDI-6* | | | P-*value*[a] |
|---|---|---|---|---|---|
| | | *Baseline* | *3 months* | *6 months* | |
| *Level of injury* | | | | | |
| C-spine | 13 | 10.5 ± 3.5 | 6.5 ± 4.0 | 5.4 ± 2.3 | 0.995 (3 months vs BL) |
| T-spine | 19 | 11.6 ± 3.6 | 8.7 ± 4.0 | 10.2 ± 4.4 | 0.22 (6 months vs BL) |
| L-spine | 6 | 12.5 ± 4.1 | 7.2 ± 4.0 | 9 ± 4.2 | |
| *Baseline Pdet* | | | | | |
| Pdet ⩽ 40 | 17 | 10.5 ± 2.7 | 6.4 ± 4.4 | 8.7 ± 4.9 | 0.86 (3 months vs BL) |
| Pdet > 40 | 21 | 12.1 ± 4.1 | 8.7 ± 3.5 | 8.3 ± 3.8 | 0.82 (6 months vs BL) |
| *Baseline DSD type* | | | | | |
| Type 1 | 14 | 11.6 ± 3.3 | 7.4 ± 3.3 | 8.8 ± 4.5 | 0.22 (3 months vs BL) |
| Type 2 | 14 | 9.9 ± 2.7 | 7.9 ± 4.5 | 8.1 ± 4.9 | 0.22 (6 months vs BL) |
| Type 3 | 10 | 11.3 ± 4.6 | 8.4 ± 4.7 | 8.6 ± 4.2 | |
| *Baseline PVR* | | | | | |
| ⩽100 | 20 | 11.5 ± 3.6 | 8.1 ± 4.1 | 8.1 ± 4.6 | 0.89 (3 months vs BL) |
| >100 | 18 | 11.2 ± 3.6 | 7.3 ± 4.0 | 8.9 ± 4.0 | 0.53 (6 months vs BL) |
| *Gender* | | | | | |
| Male | 17 | 10.5 ± 3.6 | 8.2 ± 4.3 | 9.5 ± 4.4 | 0.53 (3 months vs BL) |
| Female | 21 | 12.1 ± 3.5 | 7.4 ± 4.2 | 8.9 ± 4.5 | 0.602 (6 months vs BL) |
| *Compliance* | | | | | |
| ⩽30 | 27 | 12.1 ± 3.1 | 8.2 ± 4.3 | 9.5 ± 4.4 | 0.77 (3 months vs BL) |
| >30 | 11 | 9.6 ± 4.3 | 6.6 ± 3.3 | 6 ± 2.9 | 0.76 (6 months vs BL) |
| *ASIA scale* | | | | | |
| Non-A | 12 | 12.4 ± 3.6 | 7.8 ± 3.9 | 10.3 ± 3.7 | 0.19 (3 months vs BL) |
| A | 26 | 10.9 ± 3.6 | 7.7 ± 4.2 | 7.7 ± 4.3 | 0.48 (6 months vs BL) |
| *Dosage* | | | | | |
| 200 U | 11 | 13.1 ± 3.9 | 9.6 ± 3.8 | 10.6 ± 4.3 | 0.6 (3 months vs BL) |
| 300 U | 11 | 10.6 ± 4.4 | 7.5 ± 3.4 | 9.2 ± 3.9 | 0.359 (6 months vs BL) |

Abbreviations: ASIA, American Spinal Injury Association; BL, baseline; BoNT-A, botulinum toxin A; DSD, detrusor sphincter dyssynergia; Pdet.Qmax, detrusor pressure at maximum ow rate; PVR, post-void residual; UDI-6, urogenital distress inventory 6-item short form.
[a]*P*-value using paired Student's *t*-tests for dichotomous variables and analysis of variance for more than two variables to compare changes of UDI-6 among patients with different variables.

increased significantly at 3 and 6 months after treatment. Pdet.Qmax significantly decreased at 3 months, but was elevated at 6 months after treatment. Qmax was significantly decreased after treatment, and urodynamic DO disappeared in 10 patients (26%) at 3 months after treatment.

Comparing different baseline demographics and clinical outcome measures, no pretreatment demographic was significantly associated with changes in UDI-6 parameters (Table 2). Patients with different SCI levels or DSD types had similar therapeutic outcomes and changes of Pdet.Qmax. However, a higher baseline Pdet.Qmax (⩾40 cm $H_2O$) had a significantly greater reduction in detrusor pressure than patients with a lower baseline Pdet.Qmax (< 40 cm $H_2O$) at 3 and 6 months (−28.3 ± 22.4 vs 2.3 ± 18.1, $P<0.001$ and −24.8 ± 21.3 vs 5.3 ± 16.3 cm $H_2O$, $P=0.007$), respectively (Table 3).

Among the 11 patients who received an initial 200 U and another 300 U of Botox treatment, the treatment outcomes did not differ significantly, except that the Pdet.Qmax at 6 months was higher in the group that received 200 U of Botox.

Among the patients with satisfactory responses, 23 had reductions of QoL index by two or more points. No significant differences were found between the subgroups with QoL reductions of two or more points and those with fewer than two points (Table 4).

## Discussion

Among our SCI patients, 60% had satisfactory responses to detrusor BoNT-A injections for lower urinary tract dysfunction.

**Table 3** Multivariate analysis of factors and Pdet.Qmax at different time points in SCI patients with detrusor BoNT-A injection

| *Variable* | *Cases* | *Pdet.Qmax (cm $H_2O$)* | | | P-*value* |
|---|---|---|---|---|---|
| | | *Baseline* | *3 months* | *6 months* | |
| *Level of injury* | | | | | |
| C-spine | 13 | 37 ± 21.2 | 19.4 ± 14.8 | 12.2 ± 11.3 | 0.874 (3 months vs BL) |
| T-spine | 19 | 43.2 ± 21.9 | 29.7 ± 18.4 | 35 ± 26.6 | 0.44 (6 months vs BL) |
| L-spine | 6 | 41.7 ± 22.4 | 29.2 ± 16.6 | 38.2 ± 28.7 | |
| *Baseline Pdet* | | | | | |
| Pdet ⩽ 40 | 17 | 21.4 ± 12.5 | 23.7 ± 14.3 | 26.7 ± 26.2 | 0.00 (3 months vs BL) |
| Pdet > 40 | 21 | 56.4 ± 11.7 | 28.1 ± 19.4 | 31.6 ± 25.5 | 0.007 (6 months vs BL) |
| *Baseline DSD type* | | | | | |
| Type 1 | 14 | 36.4 ± 17.1 | 30.1 ± 11.5 | 34.9 ± 29.3 | 0.26 (3 months vs BL) |
| Type 2 | 14 | 38.8 ± 17.1 | 23.1 ± 19.2 | 31.3 ± 25.3 | 0.24 (6 months vs BL) |
| Type 3 | 10 | 49.6 ± 21.9 | 27.9 ± 22.2 | 19.9 ± 20.4 | |
| *Baseline PVR* | | | | | |
| ⩽100 | 20 | 40 ± 16.1 | 26.3 ± 14.4 | 31.7 ± 24.6 | 0.83 (3 months vs BL) |
| >100 | 18 | 41.6 ± 26.4 | 25.9 ± 20.3 | 26.5 ± 27.2 | 0.61 (6 months vs BL) |
| *Gender* | | | | | |
| Male | 17 | 35.4 ± 21.4 | 20.7 ± 15.0 | 23.9 ± 20.8 | 0.99 (3 months vs BL) |
| Female | 21 | 45.1 ± 20.7 | 30.5 ± 18.0 | 33.9 ± 28.7 | 0.807 (6 months vs BL) |
| *Compliance* | | | | | |
| ⩽30 | 27 | 42.1 ± 22.0 | 30.2 ± 18.1 | 31.2 ± 27.4 | 0.32 (3 months vs BL) |
| >30 | 11 | 37.4 ± 20.2 | 16.2 ± 9.7 | 24.4 ± 20.1 | 0.39 (6 months vs BL) |
| *ASIA scale* | | | | | |
| Non-A | 12 | 43.3 ± 24.2 | 25.2 ± 16.5 | 31 ± 23.0 | 0.58 (3 months vs BL) |
| A | 26 | 39.6 ± 20.3 | 26.5 ± 17.9 | 28.7 ± 27.0 | 0.62 (6 months vs BL) |
| *Dosage* | | | | | |
| 200 U | 11 | 43.9 ± 22.1 | 26.1 ± 17.2 | 48.3 ± 12.6 | 0.07 (3 months vs BL) |
| 300 U | 11 | 47.6 ± 13 | 24.4 ± 19 | 25.9 ± 18.9 | 0.026 (6 months vs BL) |

Abbreviations: ASIA, American Spinal Injury Association; BL, baseline; BoNT-A, botulinum toxin A; DSD, detrusor sphincter dyssynergia; Pdet.Qmax, detrusor pressure at maximum ow rate; PVR, post-void residual; UDI-6, urogenital distress inventory 6-item short form.[2] *P*-value using paired Student's *t*-tests for dichotomous variables and analysis of variance for more than two variables to compare changes of Pdet.Qmax among patients with different variables.

**Table 4** Baseline parameters in responders and non-responders

| | *Responders QoL reduction ⩾2 (n = 23)* | *Non-responders QoL reduction <2 (n = 15)* | P-*value* |
|---|---|---|---|
| Gender (M/F) | 11/12 | 10/5 | 0.254 |
| SCI level (C/T/L) | 9/10/4 | 4/11/0 | 0.106 |
| DSD type (1/2/3) | 9/10/4 | 5/4/6 | 0.341 |
| Pdet.Qmax (cm $H_2O$) | 40 ± 20.3 | 41.8 ± 23.52 | 0.808 |
| PVR (ml) | 119.13 ± 102.2 | 166.47 ± 96.9 | 0.163 |
| Compliance | 25.87 ± 25.3 | 28.38 ± 26.6 | 0.771 |

Abbreviations: DSD, detrusor sphincter dyssynergia; M/F, male to female; Pdet.Qmax, detrusor pressure at maximum ow rate; PVR, post-void residual; SCI, spinal cord injury; QoL, quality of life.

The urodynamic parameters improved significantly and UDI-6 scores also improved up to 6 months after detrusor BoNT-A injection. There was no significant difference in the treatment outcome among patients of different SCI levels or DSD types. The only factor that predicted a greater reduction of detrusor pressure after BoNT-A injection was a higher baseline Pdet.Qmax.

Most of the previous open-label studies confirmed the positive impact of BoNT-A on the decrease of Pdet.Qmax in NDO.[4,10–12] The mean percentage reduction in Pdet.Qmax from baseline was approximately from 40 to 60%, and in most of these studies, the mean Pdet.Qmax was reduced to less than 40 cm $H_2O$ after BoNT-A treatment, which is generally considered as the desired Pdet.Qmax for upper urinary tract protection.[13] We found a positive impact of BoNT-A on Pdet.Qamx reduction in this study (40.7 ± 21.3 vs 26.1 ± 17.2 cm $H_2O$, $P = 0.01$). Furthermore, if we compared the therapeutic effects of subgroups between baseline Pdet.Qmax <40 and ⩾40 cm $H_2O$, the patients with Pdet.Qmax ⩾40 cm $H_2O$ had a significantly greater reduction of voiding pressure than patients with Pdet.Qmax <40 cm $H_2O$ at 3 and 6 months after BoNT-A treatment. A baseline higher Pdet.Qmax could be a predictive factor affecting the therapeutic results of BoNT-A injection in SCI patients.

BoNT-A inhibited acetylcholine release in the cholinergic nerve terminals.[3] Recent studies showed that BoNT-A also affected the afferent pathways; henceforth, an alternative hypothesis of a dual mechanism of action was proposed for treating overactive bladders (OAB).[14] The bladder-afferent neuron receptors implicated include vanilloid, purinergic (P2X) and neurokinin receptors. The neurotransmitters functioning at these receptors, such as adenosine triphosphate, substance P, neurokinin A, nitric oxide, nerve growth factor and calcitonin gene-related peptide are believed to have important roles in modulating the sensory afferent nerves in the human detrusor, especially in diseased bladder states such as DO.[15] We found that patients with baseline lower Pdet.Qmax had almost no change in the reduction of Pdet.Qmax after BoNT-A treatment, but their perception of satisfaction, as assessed by UDI-6, was similar to the subgroups with higher Pdet.Qmax. The dual mechanism of action might explain why BoNT-A could exert an inhibitory effect on the motor innervation by reducing detrusor

pressure in patients with high baseline Pdet.Qmax, although acting on afferent pathways in patients with lower baseline Pdet.Qmax.

Previous reports demonstrated that clinically relevant increases in cystometric bladder capacity and decreases in detrusor pressure can be achieved with either lower mean baseline bladder compliance (6.5 ml per cm of $H_2O$)[16] or high values, as reported by Schurch *et al*.[3] (32.6 ml per cm of $H_2O$) and Reitz *et al*.[4] (32 ml per cm of $H_2O$).We also found no significant difference in therapeutic effects, either in UDI-6 or reduction of Pdet.Qmax, between patients with higher and lower baseline bladder compliances. The improvement in patients with low compliance, even with a long duration of illness, suggests that detrusor fibrosis is not solely responsible for severely decreased bladder compliance.

DSD type correlates well with neurological deficit.[17] Patients with complete lesions usually had either type 2 or type 3 DSD compared with patients, with incomplete sensory and motor deficits, who had type 1 DSD. Additionally, DSD tended to worsen over time. Weld *et al*.[1] found that the proportion of patients with type 1 DSD decreased with time and the proportion of patients with type 2 DSD increased with time. It is rational to hypothesize that in patients with more severe lesions such as type 3 DSD or complete (American Spinal Injury Association-A), the therapeutic effect of BoNT-A treatment would be less efficacious. However, we found that the type of DSD or completeness of the lesion was not associated with the therapeutic results of BoNT-A treatment, suggesting that the severity of lesions in patients with SCI cannot predict success of the treatment after BoNT-A injection.

In treating patients with urinary incontinence, due to NDO, a dose of 300 U of Botox is commonly used for detrusor injection, with excellent success.[1,3] However, a dose of 200 U of Botox was also reported to give satisfactory results for most patients with SCI and DSD.[3,18] Although a higher dose of Botox provided a longer lasting decrease of Pdet.Qmax in this study, the patients treated with an initial 200 U dose and a repeated 300 U dose had similar therapeutic UDI-6 and QoL results, suggesting a higher dose of Botox might not achieve a better treatment result in real-life practice. The therapeutic effect of BoNT-A on NDO might not solely depend on paralysis of the detrusor. It seems that the dosage of 200 or 300 U for treatment of NDO was all optional for detrusor BoNT-A injection to achieve satisfactory results.

This study revealed that SCI patients with different SCI level or types of DSD had similar therapeutic results. The age, gender and American Spinal Injury Association subtype did not affect the BoNT-A treatment outcome. Because the aim of treatment was to decrease the degree of incontinence, patients usually accepted the increase in PVR and the need of CIC. Treatment of voiding dysfunction in patients with spinal cord lesions and DSD is a challenge for physicians. About 95% of patients with suprasacral SCI or disease have DO, with or without DSD.[1] Their hand dexterity, abdominal muscle power, bladder sensation and the degree of urethral sphincter dyssynergia may affect voiding efficiency and lower urinary tract dysfunction.[1] Currently, most SCI patients with DSD are treated with detrusor BoNT-A injections followed by CIC to restore urinary continence and achieve better QoL.[19–21] Although patients treated with 200 U of Botox might still have mild urinary incontinence, some patients preferred retaining spontaneous voiding and were satisfied with this treatment outcome.[18] It is important to realize the desire and willingness of patients with SCI to deal with low urinary tract dysfunction. The limitation of this study is the small patient number in each subgroup.

*Conclusion*

A total of 60% of patients with SCI and DSD had satisfactory responses to detrusor BoNT-A injection. Patients with different SCI levels or DSD types had similar treatment outcomes after detrusor 200 U BoNT-A injections for NDO. A higher baseline Pdet.Qamx predicted a greater reduction of detrusor pressure after BoNT-A injection, but did not affect the improvement of UDI-6.

## Conflict of interest

The authors declare no conflict of interest.

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