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VIDEO-URODYNAMIC PREDICTIVE FACTORS OF SUCCESSFUL ONABOTULINUMTOXINA URETHRAL SPHINCTER INJECTION FOR NEUROGENIC OR NON-NEUROGENIC DETRUSOR UNDERACTIVITY

Hypothesis / aims of study

Although onabotulinumtoxinA urethral sphincter injection seems effective in treating voiding dysfunction due to detrusor underactivity (DU), not all patients have successful treatment results. Therefore, this study analyzed the treatment outcomes and identify videourodynamic predictive factors for successful outcome in patients with neurogenic and non-neurogenic DU.

Study design, materials and methods

A total of 60 patients including 27 with non-neurogenic and 33 with neurogenic DU were treated with injections of total 100U of onabotulinumtoxinA into the urethral sphincter. Treatment outcomes were assessed 1 month after treatment using the Global Response Assessment. The treatment outcome was analyzed by the baseline video-urodynamic characteristics..

Results

Overall, good outcomes were reported in 36 (60%) patients of DU. The treatment outcome was significantly better in patients with non-neurogenic DU than neurogenic DU (74.1% VS 48.5%, p=0.039)(Table 1). However, a good treatment outcome was not related to age, gender, or any videourodynamic variables except for the condition of bladder neck during voiding (the rate of good outcome, open 94.3% vs tight 12.0%, p<0.0001). In the patients who had good treatment outcome after onabotulinumtoxinA treatment, the IPSS, Qmax, voided volume and PVR all improved in neurogenic or non-neurogenic DU (Table 2). However, the changes of measured parameters from baseline to post-treatment between groups showed no significant difference. A total of 12 patients (20%) reported de novo urinary incontinence after urethral onabotulinumtoxinA injection, including 4 developed stress urinary incontinence and 8 had exacerbated urgency urinary incontinence.

Interpretation of results

Voiding is a complex interaction of central and peripheral neural control, detrusor contraction, bladder neck relaxation, external sphincter relaxation, and adequate pelvic floor relaxation. Either neurogenic or non-neurogenic voiding dysfunction develops through one or more defects of the micturition mechanisms, including neuropathy, true detrusor failure, a tight bladder neck or a non-relaxing urethral sphincter. In neurogenic DU patients, inadequate abdominal straining voiding, more reduced bladder sensation, and more complex voiding dysfunction might contribute to the poor treatment outcome after onabotulinumtoxinA urethral sphincter injection.

DU patients can spontaneous void or by abdominal straining to overcome the reduced urethral resistance and achieve efficient voiding after urethral sphincter onabotulinumtoxinA injection. Bladder neck narrowing or tightness indicates the more complex underlying voiding dysfunction and the higher resistance of bladder outlet. And these patients should be treated with surgery of transurethral incision of bladder neck first followed by onabotulinumtoxinA urethral sphincter injection. Therefore, pre-operative bladder neck opening status could help urologist not only in the selection of treatment options but also to predict the surgical outcome of onabotulinumtoxinA urethral sphincter injection

Concluding message

OnabotulinumtoxinA urethral sphincter injection is effective in 60% of patients with voiding dysfunction due to neurogenic or non-neurogenic DU. Careful videourodynamic interpretation of bladder neck opening enables urologists to select appropriate candidates for onabotulinumtoxinA treatment.

Table 1. Treatment outcome according to patients' characteristics at baseline

	Good outcome (n= 36)	Poor outcome (n= 24)	Univariate P value	Multivariate P value
Age	63.7 ± 15.6	63.1 ± 15.5	0.887	
Sex (M/F)	11/25	6/18	0.434	
Non-neurogenic	20 (74.1%)	7 (25.9%)	0.039	
Neurogenic	16 (48.5%)	17 (51.5%)		
BN open	33 (94.3%)	2 (5.7%)	<0.0001	<0.001
tight	3 (12.0%)	22 (88.0%)		
FSF (ml)	173.3 ± 89.5	211.1 ± 87.6	0.111	
CBC (ml)	379.6 ± 130.8	408.1 ± 138.2	0.423	
Pdet (cmH2O)	7.06 ± 8.33	4.08 ± 5.69	0.133	
Pabd (cmH2O)	53.5 ± 40.2	59.0 ± 39.3	0.604	
Qmax (ml/s)	4.61 ± 5.03	3.88 ± 3.52	0.536	
PVR (ml)	265.9 ± 157.4	312.3 ± 165.5	0.278	

BN: bladder neck, DU: detrusor underactivity, FSF: first sensation of filling, CBC: cystometric bladder capacity, Pdet: detrusor pressure, Pabd: abdominal pressure, Qmax: maximum flow rate, PVR: post-void residual

Table 2. The changes of lower urinary tract symptoms and uroflowmetry parameters in detrusor underactivity patients with good treatment outcomes after urethral sphincter onabotulinumtoxinA injection

		N	Baseline	Post-treatment	P value
IPSS	Non-neurogenic DU	20	23.2 ± 3.83	15.1 ± 5.03 *	0.844
	Neurogenic DU	16	22.0 ± 3.97	14.2 ± 4.37 *	
Qmax(ml/s)	Non-neurogenic DU	20	5.15 ± 5.57	12.7 ± 7.12 *	0.946
	Neurogenic DU	16	4.06 ± 4.07	11.8 ± 6.36 *	
Volume(ml)	Non-neurogenic DU	20	89.9 ± 102.5	217.4 ± 118.9*	0.408
	Neurogenic DU	16	105.8 ± 138.1	188.6 ± 116.5*	
PVR(ml)	Non-neurogenic DU	20	254.5 ± 151.7	97.3 ± 116.2*	0.993
	Neurogenic DU	16	281.3 ± 147.4	124.4 ± 84.3*	
Duration(M)	Non-neurogenic DU	20		7.37 ± 3.69	0.788
	Neurogenic DU	16		7.69 ± 3.18	

Table 3. The adverse events after urethral sphincter onabotulinumtoxinA injection for patients with detrusor underactivity

	Idiopathic DU (n= 27)	Neurogenic DU (n=33)	P value
Urinary tract infection	7 (25.9%)	5 (15.2%)	
Stress urinary incontinence	2 (7.4%)	2 (6.1%)	
Urgency urinary incontinence	4 (14.8%)	4 (12.1%)	
Acute urinary retention	1 (3.7%)	0	
None	13 (48.1%)	22 (66.7%)	0.545

DU, detrusor underactivity

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