ANTIMUSCARINIC THERAPY INEFFECTIVE IN HIGH PRESSURE NON-NEUROGENIC DETRUSOR OVERACTIVITY WITH INCONTINENCE: SHOULD BOTULINUM TOXIN A BE USED PRIMARILY?

Hypothesis / aims of study
Urinary incontinence due to detrusor overactivity can be difficult to manage, and is not always responsive to first and second line therapies. The purpose of this study is to report observations on the use of onabotulinum toxin A in a subset of patients with high pressure overactivity of the bladder.

Study design, materials and methods
This is a retrospective case series of 11 patients treated with onabotulinum toxin A from Jan 2016 to March 2017. The inclusion criteria were patients without spinal cord injury or disease, who had failed antimuscarinic therapy, who had urodynamics, and subsequently treated with onabotulinum toxin A. The criterion for high pressure overactivity of the bladder (pDet) was a spontaneous contraction(s) during filling cystometry of greater than 40cm water. The criteria for antimuscarinic therapy failure was failure to suppress urinary incontinence and relieve urgency with at least 3 months of high does therapy, or inability to tolerate at least two antimuscarinic agents. The patients case history and treatments were reviewed. Results are presented as mean ± Standard deviation.

Results
10 patients, 9 male, and 2 female with treated with onabotulinum toxin A using 100 to 200 units by intravesical injection. The mean age was 66 ± 15 years. All the patients had failed high dose antimuscarinic therapy (e.g. oxybutynin 30mg sustained release daily, or solifenicen 10mg Daily), or could not tolerate the side effects. All had several incontinence episodes daily despite therapy. No patient had urinary tract infections. All had a urodynamic study which showed high pressure spontaneous contractions 103.8 ± 38.8 cm water at 175 ± 75.1 ml volume. Two patients had temporary urinary retention responsive to clean intermittent catheterization for 2-3 weeks. No UTIs were found post procedurally. All reported cessation of urinary incontinence at 1 month follow up and this was persistent for 8.4±3.3 months.

Interpretation of results
All these patients spent many months undergoing trials of antimuscarinic therapy with various agents and doses. All ultimately failed antimuscarinic therapy, and thus had a urodynamic study. In all these cases, high pressure overactivity was found to be the cause of incontinence. All responded, with resolution of incontinence, to onabotulinum toxin A therapy.

Concluding message
This small case series would suggest that patients found to have high pressure overactivity may be better managed with early use of intravesical onabotulinum toxin A. Failure of antimuscarinic therapy due to high pressure overactive bladder and a potential solution has not been reported before.

Disclosures
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