

TRIGONAL INJECTION OF BOTULINUM TOXIN TYPE A DOES NOT CAUSE VESICoureTERAL REFLUX IN PATIENTS WITH REFRACTORY NEUROGENIC DETRUSOR OVERACTIVITY

Hypothesis / aims of study

The injection of botulinum toxin type A (BTX-A) into the bladder has recently emerged as an attractive alternative for the treatment of refractory neurogenic detrusor overactivity, but controversy exists regarding the dose of BTX-A to be injected as well as number and location of the injections. Most investigators have spared the trigone when injecting BTX-A because of the potential of inducing vesicoureteral reflux (VUR). However, BTX-A injections into the trigone might improve the effect on bladder afferent pathways which could be beneficial for patients with conditions such as idiopathic OAB and interstitial cystitis. We evaluated the effect of BTX-A injections in the trigone on the antireflux mechanism and analyzed its short-term efficacy.

Study design, materials and methods

Between April and December 2006, 21 patients (10 men and 11 women) with a mean age of 35.4 ± 15.9 years were prospectively evaluated. All patients complained of urinary incontinence which was caused by detrusor overactivity as demonstrated by urodynamics. The condition resulted from spinal cord injury in 12 (57%) patients, viral myelitis in 8 (38%) and multiple sclerosis in one (5%) patient. Preoperative evaluation included urine culture, urinary tract ultrasound to evaluate upper urinary tract dilation and a cystogram to evaluate VUR. A standard urodynamic study was performed in each patient. The evaluated parameters were maximum cystometric capacity, reflex detrusor volume, maximum detrusor pressure during bladder contraction and compliance. All patients underwent detrusor injection of 300 units of BTX-A, including 50 units into the trigone. Postoperative evaluation after 8 weeks of the injection included a clinical assessment of continence, urodynamics, urinary tract ultrasound and cystogram. The primary outcome was the presence or absence of VUR at cystogram study before and 8 weeks after BTX-A injection. The secondary outcomes were the continence status and urodynamic parameters.

Results

At baseline, 20 patients had no vesicoureteral (VUR) and one had grade II unilateral VUR. Postoperative evaluation revealed no cases of de novo VUR and the patient with preinjection VUR had complete resolution of the reflux. Urinary tract ultrasound showed 5 (23.8%) patients with hydronephrosis before BTX-A injection and only one (4.8%) at the followup evaluation ($p=0.125$). After treatment, 9 (42.8%) patients became dry, 11 (52.4%) were improved and one (4.8%) had no improvement. Improved patients received antimuscarinic treatment and 8 (38.1%) became dry, with a final total continence rate of 80.1%. Cystometric capacity increased from 271 ± 92 to 390 ± 189 ml ($p=0.002$), reflex volume varied from 241 ± 96 to 323 ± 201 ml ($p=0.020$) and maximum detrusor pressure reduced from 66 ± 39 to 38 ± 37 cmH₂O ($p<0.001$).

Interpretation of results

In this series, BTX-A injection into the trigone was safe in terms of the possibility of causing vesicoureteral reflux. It did not induce de novo or worsen preexisting VUR in any patient. Likewise, upper urinary tract was not adversely affected by the trigone injections of BTX-A. On the contrary, a tendency to improvement of the upper tracts was observed in terms of hydronephrosis. Both findings are probably explained by the decreased bladder pressures that are observed after treatment. To our knowledge, only one previous study evaluated the effect of BTX-A injection in the trigone in terms of the antireflux mechanism. (1) In the present study we evaluated a different and larger patient population, with refractory detrusor overactivity secondary to overt neurological diseases. Moreover, we used a different injection protocol and also evaluated the upper urinary tracts.

Concluding message

Our results confirm the safety of trigone injections of BTX-A in terms of development of VUR and upper urinary tract damage. Whether trigone injections are beneficial for patients with neurogenic detrusor overactivity or other causes of voiding dysfunction will need further studies.

References

(1) J Urol (2007) 177; 1011-1014.

FUNDING: None

CLINICAL TRIAL REGISTRATION: This clinical trial has not yet been registered in a public clinical trials registry.

HUMAN SUBJECTS: This study was approved by the Institutional Review Board and Ethics Committee of the Sarah Kubitschek Hospital, Salvador, BA, Brazil and followed the Declaration of Helsinki Informed consent was obtained from the patients.