ORIGINAL ARTICLE

The use of oxybutynin in patients treated by means of botulinum neurotoxin A for neurogenic detrusor overactivity: an observational study

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Study design: This is an observational prospective noncontrolled study. We enrolled 105 patients affected by neurogenic detrusor overactivity (NDO) who underwent botulinum neurotoxin A (BONT-A) intradetrusor injection and were followed-up for 270 days.

Objectives: To investigate the use of oxybutynin after BONT-A administration in NDO patients.

Setting: Careggi University Hospital in Florence and Tor Vergata University Hospital in Rome.

Methods: Prospective data from two Italian centers were collected in 1 year. Patients showing limited efficacy but good tolerability and adherence to oxybutynin 5 mg t.i.d. (three times a day), were enrolled in the study. Patients received BONT-A intradetrusor injection (onabotulinumtoxinA, 300 U) with a trigone-sparing technique, and the use of oxybutynin was registered at every visit.

Results: 105 patients were included. At visit 1, only 30 patients (28.6%) used oxybutynin t.i.d., whereas 47 (45.2%) used a lower dosage and 28 (26.7%) stopped the therapy. At visit 2, 77.3% of patients who had reduced oxybutynin intake, maintained the dosage decided at visit 1. At visit 3, 51.9% returned to oxybutynin t.i.d.; 44.8% were on a reduced dosage and only 3 (2.9%) were not taking the drug. At visit 4, only 37.5% of patients were taking less than oxybutynin t.i.d.

Conclusion: This study provides some important insights on the use of oral antimuscarinics in patients treated by means of BONT-A intradetrusor administration for NDO; in particular, it suggests that, after an initial reduction in the use of oxybutynin, patients tend to increase the dosage of this drug during the follow-up after the BONT-A treatment.

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Keywords: neurogenic detrusor overactivity; oxybutynin; antimuscarinics; botulinum toxin-A injection; overactive bladder.

INTRODUCTION

The majority of patients with spinal cord injury (SCI) and multiple sclerosis suffer from lower urinary tract dysfunction.^{1,2} It has been shown that 100% of complete SCI and about 90% of multiple sclerosis patients suffer lower urinary tract symptoms during the course of their lives.^{1,2}

In particular, a suprasacral spinal cord lesion can induce neurogenic detrusor overactivity (NDO), possibly associated with detrusor sphincter dyssynergia: these conditions impair the storage and emptying functions of the urinary bladder, causing high intravesical pressure, reduced bladder capacity, incontinence and potential structural damage of the bladder wall; furthermore, NDO poses a threat to the upper urinary tract.^{2,3}

The first line treatment for NDO is based on the use of antimuscarinic agents with the aim of blocking involuntary detrusor contractions or reducing intravesical pressure, of increasing bladder capacity and of preserving renal function; clean intermittent catheterization is usually associated to antimuscarinics, especially in patients with detrusor sphincter dyssynergia, to allow a complete bladder emptying.^{4,5}

It has been shown that NDO treatment could need higher doses of antimuscarinic drugs than treatment of idiopathic detrusor

overactivity or overactive bladder syndrome; furthermore, a significant number of patients report low efficacy or side effects that limit the possibility to use these drugs.^{5,6}

In 2000, Schurch *et al.*⁷ proposed the use of botulinum toxin type-A injection into the detrusor muscle for treatment of NDO. This therapy represents a minimally invasive alternative treatment option, especially when oral anticholinergic treatment is ineffective or not tolerated by patients.

Botulinum neurotoxin A (BONT-A) is highly recommended for the treatment of NDO, especially in SCI patients.⁸ Its safety and efficacy have been recently confirmed in randomized placebo-controlled studies, allowing the indication of BONT-A for NDO.^{9–10}

However, in the majority of the published studies, the use of antimuscarinics (or drugs with mixed action as oxybutynin) after BONT-A administration was allowed, provided that no dosage escalation was permitted. Only in a minority of reports, the percentage of patients who discontinued the treatment with antimuscarinics was reported, varying from 28 to 58%, with many other patients who reduced the dose.¹¹ Nevertheless, no study specifically investigating how many patients, in a clinical (non-investigational) setting, discontinue the antimuscarinics or reduce their dosage after BONT-A administration, has been published. This data could be of

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interest for economic evaluation of BONT-A treatment; furthermore, assessing the prevalence of antimuscarinic drugs use could address more research aimed to investigate possible interactions between BONT-A and these agents.

The aim of this observational study was to investigate the use of oxybutynin after BONT-A administration in NDO patients.

MATERIALS AND METHODS

This is an observational prospective noncontrolled study. The use of a control group was considered unethical or not feasible for this study. The STROBE guidelines were used to ensure the reporting of this study.¹²

All patients who received intradetrusor injections of BONT-A for NDO in two Italian centers (Tor Vergata University Hospital, Rome and Careggi University Hospital, Florence) in 1 year (between January and December 2009) were considered eligible for this study.

Inclusion criteria were:

- Patients satisfied, needing no further treatment; subjective satisfaction was investigated by the following simple question: 'Are you completely satisfied of the improvement of your lower urinary tract symptoms after the BONT-A administration?' Patients responding 'yes' were considered satisfied; the need for further treatment was assessed by the physician on the basis of clinical and urodynamic data.
- Use of onabotulinumtoxin A (Botox, Allergan Inc., Irvine, CA, USA) at the dosage of 300 U.
- Previous and ongoing unsuccessful treatment with high dosage oxybutynin (mixed action drug with prevalent antimuscarinic action).

This drug was chosen because it is the only reimbursable (thus, the most commonly used) drug for neurogenic patients in Italy.

Exclusion criteria were:

- Intolerance or reduced compliance to antimuscarinic drugs.
- Use of a different antimuscarinic drug.

Patients received BONT-A by means of rigid cystoscopy and the use of an endoscopic 24 G needle in 20–30 sites of the bladder wall, with a trigone-sparing technique.

Patients were followed-up at 30 days (visit 1), by means of bladder diaries and (in some cases) urodynamic examination. After this visit, patients were suggested to continue, reduce or interrupt the oral treatment (oxybutynin) on the basis of the physician's opinion (based only on clinical/urodynamic findings, without a pre-established strategy). Visit 2, 3 and 4 were performed 120, 180 and 270 days, respectively, after BONT-A administration. The use of oxybutynin was registered during every visit. To avoid any influence on patients/physicians behavior, no specific questions on the reasons for drug intake or discontinuation were posed and the physician was instructed to feel completely free in his clinical practice.

Statistical analysis

For each visit frequencies of categorical variables were calculated. Changes of doses between two consecutive visits were calculated. χ^2 test was used to evaluate relationship between the dose at first visit with the dose at 180 days. Data are presented as mean $\pm$ s.d. A *P*-value <0.05 was considered statistically significant. Stata 12.0 (StataCorp LP, College Station, TX, USA) was used for all analysis.

RESULTS

On 174 patients treated in both centers, during the study period, 105 were included in this observational study.

Sixty-nine patients were not included because they were:

- Treated with a different BONT-A or with a different dosage of onabotulinumtoxin A ($n = 24$ patients).
- Treated with an antimuscarinic drug other than oxybutynin ($n = 18$ patients).
- Not tolerating antimuscarinic drugs ($n = 15$ patients).
- Not responding to BONT-A ($n = 12$ patients).

Ninety-five were suprasacral SCI patients (76 patients were evaluated in Florence and 19 patients in Rome), whereas the remaining 10 (7 patients were evaluated in Florence and 3 patients in Rome) were multiple sclerosis patients. All patients had been taking oxybutynin tablets (5 mg) 1 t.i.d. All patients had been treated with oxybutynin for at least 3 months, with a majority of them (64%) who had undergone this therapy for more than 4 months. Ninety-five percent of patients reported to have used the antimuscarinic therapy on a regular basis, without discontinuing it for more than 3 days. All patients were on clean intermittent catheterization regimen. Sixty-six out of the 105 patients (62.9%) were male and their mean age was 42.3 ± 12.5 years.

At visit 1, only 30 patients (28.6%) remained on oxybutynin t.i.d., whereas 47 (44.7%) reduced the dosage and 28 (26.7%) stopped the oral therapy (Table 1). Globally, a reduction of 158 tablets per day (around 1.5 tablets per day per patient, 50% less than the initial dose) was registered.

Urodynamic and clinical findings were considerably improved after BONT-A administration. In particular, maximum cystometric capacity was increased from a mean of 226.04 ml (range 150–298 ml) to a mean of 372.42 ml (range 270–500 ml, $P = 0.0001$). A significant improvement in incontinence episodes per week was also noticed: 0.72 (range 0–3) at visit 1 from 30.3 (range 42–15) at baseline ($P = 0.0001$).

We divided patients in four groups, after visit 1, according to the dosage of oxybutynin used.

Table 1 Dosage of oxybutynin for each visit

Oxybutynin dosage	Baseline		30 days (visit 1)		120 days (visit 2)		180 days (visit 3)		270 days (visit 4)	
	Number of patients	%	Number of patients	%	Number of patients	%	Number of patients	%	Number of patients	%
0	105		105		105		104		72	
1 $\times$ 1			28	26.7	25	23.8	3	2.9		
1 $\times$ 2			27	25.7	21	20.0	14	13.5	9	12.5
1 $\times$ 3			20	19.0	28	26.7	33	31.7	18	25.0
	105	100	30	28.6	31	29.5	54	51.9	45	62.5

Abbreviation: b.i.d., twice a day.

0, no oxybutynin tablet; 1 $\times$ 1, one oxybutynin tablet 5 mg per day; 1 $\times$ 2, oxybutynin tablet 5 mg b.i.d.; 1 $\times$ 3, oxybutynin tablet 5 mg t.i.d.

Table 2 Urodynamic parameters and weekly episodes of UUI at visit 1

	Group 1	Group 2	Group 3	Group 4
	Patients who discontinued oxybutynin	Patients on oxybutynin 1 × 1	Patients on oxybutynin b.i.d.	Patients on oxybutynin t.i.d.
<i>MCC (50 patients)</i>				
Mean	425.63*	401.67**	367	330.88
Median	412.5	405	370	350
s.d.	33.96	45.09	46.91	57.53
Range	400–500	320–500	270–450	200–400
<i>IE per week (105 patients)</i>				
Mean	0.18*	0.40**	0.55***	1.3
Median	0	0	0.5	1
s.d.	0.39	0.57	0.60	0.60
Range	0–1	0–2	0–2	0–2

Abbreviations: b.i.d., twice a day; IE, incontinence episodes; MCC, maximum cystometric capacity; UUI, urgency urinary incontinence.

* $P < 0.001$ vs group 3 and 4.

** $P < 0.01$ vs group 4.

*** $P < 0.001$ vs group 4.

Maximum cystometric capacity and incontinence episodes in the four groups are reported in Table 2. It is worthy to note that the group of patients assuming no oxybutynin or only one tablet showed better clinical and urodynamic data. In particular, maximum cystometric capacity was significantly higher and weekly incontinence episode number significantly lower for groups 1 and 2 in comparison with group 4 (for group 1 in comparison with group 3 as well). The urodynamic evaluation was performed in 50 out of 105 patients.

At visit 2 (120 days), 58/75 (77.3%) of patients who had reduced oxybutynin intake, maintained the dosage decided at visit 1; 16 patients increased the dosage of one tablet per day. On the other hand, three patients further reduced the dosage suggested at visit 1.

At visit 3 (180 days), one patient was lost at follow-up. Fifty-four patients of the remaining 104 patients (51.9%) returned to oxybutynin t.i.d.; 47 patients (45.2%) were on a reduced dosage of one (14, 13.5%) or two (33, 31.7%) tablets per day and only three (2.9%) were not taking the drug.

At visit 4 (270 days), data were available only for 72/105 patients (68.6%). Eighteen patients (17.1%) were lost at follow-up, whereas the remaining 15 (14.3%) had been retreated with BONT-A before visit 4. Only 27 patients (37.5%) were taking less than oxybutynin t.i.d.

The higher overall increase in oxybutynin tablets use was observed at visit 3, where globally, patients were using 75 tablets per day more than that at visit 2 (Table 3).

Finally, data show that there is a significant relationship ($P = 0.001$) between the dosage of oxybutynin used at visit 1 and the dosage used at visit 3 (Figure 1). In particular, only 15% of patients who discontinued the oxybutynin treatment at visit 1 were taking oxybutynin t.i.d. at visit 3, in comparison with 30% and 65% of patients using 1 and 2 tablets, respectively, at visit 1. On the other hand, no patient who was on oxybutynin t.i.d. at visit 1 showed a dosage reduction at the following visits.

DISCUSSION

To our knowledge, this is the first study aimed to evaluate the use of antimuscarinics or drugs with mixed action in a population of

Table 3 Change of the number of tablets used in comparison with the previous visit

	30 days (visit 1)		120 days (visit 2)		180 days (visit 3)		270 days (visit 4)	
	Number of patients	%	Number of patients	%	Number of patients	%	Number of patients	%
Table changes								
N	105		105		104		72	
3					3	2.9		
2					16	15.4		
1			16	15.2	34	32.7	31	43.1
0	30	28.6	86	81.9	51	49.0	41	56.9
–1	20	19.0	3	2.9				
–2	27	25.7						
–3	28	26.7

Figure 1 Visit 3 (180 days) oxybutynin dosage in relation to the dosage used at visit 1 (30 days). 0, no oxybutynin tablet; 1 × 1, one oxybutynin tablet 5 mg per day; 1 × 2, oxybutynin tablet 5 mg b.i.d.; 1 × 3, oxybutynin tablet 5 mg t.i.d. b.i.d. = twice a day.

patients treated with detrusor injections of BONT-A for NDO. Data coming from studies on efficacy and safety of BONT-A show that a percentage comprised between 28 and 58% of patients discontinued oral medications.^{11–15} Unfortunately, these data represent only observations coming from patients included in clinical trials; furthermore, a huge variability of findings among studies is present; thus, up to now, it is not possible to draw conclusions about the real behavior of patients treated with BONT-A, in the common clinical practice, with regard to antimuscarinic agents use.

Our study could help and give some interesting insights at this point, in patients treated by means of 300 U onabotulinumtoxin A detrusor injections, previously under oxybutynin t.i.d. with unsatisfactory clinical results.

According to our findings, it is possible to observe that around 27% of patients discontinued the oral drug treatment after BONT-A administration. Further, 44.7% of patients reduced the dose of one (19%) or two (25.7%) tablets per day, producing a global reduction of 50% of the initial dose.

At visit 2, performed 120 days after BONT-A administration, 77.3% of patients who had reduced oxybutynin intake, maintained

the dosage established at visit 1. It is interesting to note that the remaining 22.7% of patients increased the dosage established at visit 1 of only one tablet per day, and that few patients (three in our series) decide to further reduce the dosage of one tablet per day. All patients who remained under oxybutynin t.i.d. at visit 1 were still on the same dosage of the drug. These data show a substantial stability of the dosage decided after BONT-A administration at 120 day follow-up.

At 180 day follow-up, the situation appeared to be significantly changed: only 3% of patients did not use oral agents, whereas 51% increased the dosage or restarted the antimuscarinic therapy. Globally, between visit 2 and 3, an increase of 75 tablets per day was observed; this represents the higher dose escalation observed during our study (about plus 50% in comparison with the previous visit).

At 270 day follow-up (with 17% of patients globally lost to follow-up), 14% of patients had already been retreated by BONT-A intradetrusor injections. The majority of the remaining patients (62.5%) were under the pretreatment regimen of oxybutynin (t.i.d.). No patient was avoiding the use of the oral agent.

Finally, there was a clear correlation between the dosage of oxybutynin established at visit 1 and the amount of drug used at the following visits: in particular, only 15% of patients, who had discontinued the oral treatment, was using oxybutynin t.i.d. at visit 3 (180 days); on the other hand, all patients using oxybutynin t.i.d. at visit 1 were under the same dosage at visit 3.

This study presents several limitations. We believe that the presence of a control group could have increased the strength of the evidence obtained. Unfortunately, the design of the study did not allow to consider a control group: we deemed unethical to leave untreated for several months NDO patients not responding to antimuscarinic drugs. Moreover, a control group with patients responding to oral therapy could produce biases in the interpretation of the results. Nevertheless, all of our patients showed a very good adherence to the oral treatment before BONT-A administration, with a 95% of them who reported to have used the antimuscarinic therapy continuously. Thus, the discontinuation rate after BONT-A administration seems to be presumably due to an improvement of the bladder condition.

Several patients have been excluded by the study because of the established inclusions criteria: those criteria were posed for ethical and study feasibility reasons or to avoid confounding factors that could have influenced the final results. Nevertheless, these criteria could have selected a specific population, and data coming from this study could not be generalized to the whole SCI/multiple sclerosis patients' population treated with BONT-A detrusor injections. Furthermore, data were obtained in two Italian centers and could not be generalized to different Italian or international centers. In particular, immediate-release oxybutynin is the drug most commonly used in Italian patients, and was thus chosen as the oral agent used for this study: in different countries, different agents could be used, producing different results. Finally, the reasons for antimuscarinic drugs use or discontinuation were not investigated.

According to our data, it seems that a better clinical and urodynamic picture could have suggested to the physician and/or to the patient to discontinue or reduce significantly the oxybutynin regimen; in fact, in our study, patients who reduced more significantly the oral agent use, showed a significantly higher increase of maximum bladder capacity and a significantly lower number of incontinence episodes per week. Nevertheless, this observational study was intentionally designed not to investigate these aspects to avoid any conditioning to patients and physicians behavior. Thus, the reasons for oxybutynin discontinuation were not reported and a definitive conclusion is not provided.

Despite these limitations, this study, for the first time, could give some important insights.

- Around 72% of patients reduce the oral antimuscarinic dosage after BONT-A administration, but only 27% of them discontinue the treatment. The global reduction in drug dosage is roughly 50%.
- This dosage decrease is maintained at 120 day follow-up in the majority of patients.
- At 180 day follow-up, a significant increase in oral drug dose is noted (roughly 50% increase in comparison with the previous visit), with 51% of patients who increase the dose.
- At 270 day follow-up, with some patients (14.3%) already retreated with BONT-A, the majority of patients is on the pretreatment oxybutynin dosage (t.i.d.).

These findings could help in precisely calculating the economic impact of BONT-A treatment of NDO, taking into account the reduction of the antimuscarinic drug used. Furthermore, it seems that the use of antimuscarinic therapy is increasing during the course of the follow-up, with an important increase of dosage observed at 180 day (6 month) follow-up. As already stated, our study do not provide the reasons for patients' use of the oral agent, but it seems reasonable to suppose that it could be due to a compensation of the progressive loss of efficacy of BONT-A. It is known that the intervals between BONT-A intradetrusor injections vary, in literature, between 6 and 16 months.¹⁶ Thus, using or increasing the dosage of antimuscarinic drugs, patients could try to increase the interval before retreatment with the toxin. These interactions between BONT-A and oral antimuscarinic agents have not been investigated deeply so far, and could be the object of future research.¹⁷ In particular, it could be interesting to evaluate whether the intervals between BONT-A administrations could be increased by the use of antimuscarinic drugs and, eventually, the economic impact of this adjuvant therapy.

In conclusion, these observations suggests that, in an economic evaluation of BONT-A for NDO, the reduction (or interruption) of oral agents should be considered. On the other hand, in a clinical setting, many patients use antimuscarinics or mixed action drugs for NDO after BONT-A detrusor injection, especially at a follow-up ≥ 6 months; thus, in clinical practice, these drugs could probably exert the role of an adjuvant therapy.

DATA ARCHIVING

There were no data to deposit.

CONFLICT OF INTEREST

Dr Finazzi-Agrò and Dr Del Popolo are consultants for Allergan; Dr Finazzi-Agrò is consultant for Pfizer and principal investigator in Astellas trials. Dr Del Popolo is consultant for Ipsen, Hollister, Pfizer. The remaining authors declare no conflict of interest.

- 1 Abrams P, Cardozo L, Fall M, Griffiths D, Rosier P, Ulmsten U *et al*. The standardisation of terminology in lower urinary tract function: report from the Standardisation Sub-committee of the International Continence Society. *Urology* 2003; **61**: 37–49.
- 2 Hicken BL, Putzke JD, Richards JS. Bladder management and quality of life after spinal cord injury. *Am J PhysMed Rehabil* 2001; **80**: 916–922.
- 3 Perkash I. Long-term urologic management of the patient with spinal cord injury. *Urol Clin North Am* 1993; **20**: 423–434.
- 4 MacDiarmid SA. How to choose the initial drug treatment for overactive bladder. *Curr Urol Rep* 2007; **8**: 364–369.
- 5 Kennelly MJ, Devoe WB. Overactive bladder: pharmacologic treatments in the neurogenic population. *Rev Urol* 2008; **10**: 182–191.

- 6 Finney SM, Andersson KE, Gillespie JI, Stewart LH. Antimuscarinic drugs in detrusor overactivity and the overactive bladder syndrome: motor or sensory actions? *BJU Int* 2006; **98**: 503–507.
- 7 Schurch B, Schmid DM, Stohrer M. Treatment of neurogenic incontinence with botulinum toxin A. *N Engl J Med* 2000; **342**: 665.
- 8 Abrams P, Cardozo L, Khoury S and Wein A eds. *Incontinence*, edPlymouth, UKHealth Publication Ltd, 2008.
- 9 Cruz F, Herschorn S, Aliotta P, Brin M, Thompson C, Lam W *et al*. Efficacy and safety of onabotulinumtoxinA in patients with urinary incontinence due to neurogenic detrusor overactivity: a randomised, double-blind, placebo-controlled trial. *Eur Urol* 2011; **60**: 742–750.
- 10 Ginsberg D, Gousse A, Keppenne V, Sievert KD, Thompson C, Lam W *et al*. Phase 3 efficacy and tolerability study of onabotulinumtoxin A for urinary incontinence from neurogenic detrusor overactivity. *J Urol* 2012; **187**: 2131–2139.
- 11 Karsenty G, Denys P, Amarengo G, De Seze M, Gamé X, Haab F *et al*. (BotoxW) Intradetrusor injections in adults with neurogenic detrusor overactivity/neurogenic overactive bladder: a systematic literature review. *Eur Urol* 2008; **53**: 275–287.
- 12 Von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP. STROBE initiative. The strengthening the reporting of observational studies in epidemiology (STROBE) statement: guidelines for reporting observational studies. *J Clin Epidemiol* 2008; **61**: 344–349.
- 13 Reitz A, Stohrer M, Kramer G, Del Popolo G, Chartier-Kastler E, Pannek J *et al*. European experience of 200 cases treated with botulinum-A toxin injections into the detrusor muscle for urinary incontinence due to neurogenic detrusor overactivity. *Eur Urol* 2004; **45**: 510–515.
- 14 Popat R, Apostolidis A, Kalsi V, Gonzales G, Fowler CJ, Dasgupta P. A comparison between the response of patients with idiopathic detrusor overactivity and neurogenic detrusor overactivity to the first intradetrusor injection of botulinum-A toxin. *J Urol* 2005; **174**: 984–989.
- 15 Kennelly MJ, Kang J. Botulinum-A toxin injections as a treatment for refractory detrusor hyperreflexia. *Top Spinal Cord Inj Rehabil* 2003; **8**: 46–53.
- 16 Schurch B, Stohrer M, Kramer G, Schmid DM, Gaul G, Hauri D. Botulinum-A toxin for treating detrusor hyperreflexia in spinal cord injured patients: a new alternative to anticholinergic drugs? Preliminary results. *J Urol* 2000; **164**: 692–697.
- 17 Mangera A, Andersson KE, Apostolidis A, Chapple C, Dasgupta P, Giannantoni A *et al*. Contemporary management of lower urinary tract disease with botulinum toxin A: a systematic review of botox (onabotulinumtoxin A) and dysport (abobotulinumtoxin A). *Eur Urol* 2011; **60**: 784–795.