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Abstract

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Reference

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Female or Male Lower Urinary Tract Symptoms

Can Dopamine Depletion at DAT Scan in a Non Parkinson Patient be the Cause a Refractory Overactive Bladder?

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A B S T R A C T
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Introduction

Overactive bladder is a urological syndrome which affects 13–16% of the population and focus point of the neuro-urological world due to its socially invalidating symptoms (urgency and incontinence). Nevertheless it remains a complex matter as its etiology is multifactorial (neurogenic, non neurogenic, idiopathic) and its physiopathological mechanism localized at various levels and yet to be clearly known. For this matter in the last 20 years it has been subject of various studies with following implications in treatment, advocated by guidelines, which goes from behavioral therapy, to oral drugs (anti-muscarinics and recently beta adrenergics) intracavitary drugs (vanilloids: non longer in use) intradetrusorial drugs (botox) and nerve stimulation (neuromodulation, PTNS).

Addressing this matter, Andersson introduced the concept of possible central nervous system targets by reviewing all neurotransmitters involved at a central level. He emphasized however the fact that a selective action on the lower urinary tract might be difficult to obtain and is in fact yet to demonstrate in non neurogenic patients.

We report here what we believe to be the first case of a patient treated successfully with dopamine for idiopathic refractory overactive bladder after evidencing depletion by DAT scan.

Material and methods

A 49 year old female patient come to our observation in 1989 for a symptomatic incontinence (more than 3 diapers/day wet) initially falsely labeled as stress incontinence, which persisted without any improvement nor modification of symptoms after colposuspension according to Burch. Our initial work-up included video-urodynamics which concluded at urge incontinence from detrusor overactivity. Patient subsequently underwent full neurological evaluation as well as urological work-up to rule out any neurogenic (Parkinson’s disease) or non neurogenic origin to finally conclude to urge incontinence in a context of idiopathic overactive bladder. Initial treatment included lifestyle treatment and adjunction of oxybutynin without any success. In 1990, patient underwent surgery for releasing of the colposuspension believing that this might have determined the non response to drugs, unfortunately again without success. Patient
subsequently underwent pharmacological modulation by intravesical instillation of vanilloids and chronic intravesical instillation of nociceptin\textsuperscript{3}. Due to symptom persistence electrical neuromodulation by stimulation of S3 sacral roots as well as experimental pudendal nerve stimulation were performed without any results. Detrusor botulinum toxin did not have any more luck. Molecules such as tolterodine and trospium chloride as well as mirabegron did not show any improvement in symptom control.

Due to a “therapeutical dead end, discussion with patient of a potential derivation associated to cystectomy was taken into account. In front of patients reluctance, inspired by Andersson’s work\textsuperscript{2} we decided to perform DAT scan. The exam showed a clear reduction in dopamine at level of the left putamen and at level the right caudate nucleus. (Fig. 1) although no neurological condition was ever diagnosed (full neurological work-up). In this context patient started retard levodopa (Madopar\textsuperscript{4}) treatment (100 mg per day) and was scheduled for follow-up controls.

Results

At first control at 1 month, patient already described an improvement with persistence of mild urgency and frequency at voiding diary but complete resolution of urge incontinence requiring only small pads which however were used more for the fear of an eventual leakage.

Patient continued treatment and was seen at 3 months from treatment start and still referred to be completely dry (one pad a day dry) with only the persistence of daytime frequency at bladder diary which however had improved since the last control.

Discussion

This case is an interesting case of refractory idiopathic overactive bladder. In our centers experience a few comparable situations have been noted. For each patient, all guidelines recommended treatments (Fig. 2) were used without any success, bringing up the idea that we do not know yet everything about the pathophysiology of overactive bladder. Andersson describes that central dopaminergic pathways may have facilitatory and inhibitory effects on micturition by actions through D1-like and D2 like dopaminergic receptors. This has already been described in Parkinson’s disease affected patients\textsuperscript{5} but not in “non neurological” patients, where an involvement of dopamine depletion has yet to be demonstrated. We believe in the importance of presenting this case for two reasons: it is the first description of dopamine use for successful treatment of refractory idiopathic overactive bladder. Second it introduces DAT scan imaging as a possible “diagnostic” tool in patients non responders to current treatment options. This case presentation does not claim to have found a new treatment option but opens the way for new research which might confirm the use of imaging for overactive bladder diagnosis.

Furthermore if a correlation between symptoms and imaging is found central nervous system target might finally be confirmed as treatment options in which could therefore be more defined as undisclosed neurogenic overactive bladder than idiopathic. It is important to emphasize once again that this report should be a preliminary for a structured randomized study.

Conclusion

This represents for us an interesting case as it shows for the first time a possible correlation between voiding symptoms and central nervous system deficit, demonstrated through imaging. If this is confirmed in other patients, central nervous system deficit may be recognized as a cause of bladder instability in non neurological patients and central nervous targets might be confirmed as treatment options. Furthermore this might lead to a modification of terminology switching from idiopathic overactive bladder to a more meaningful “undisclosed neurogenic” overactive bladder.

Conflicts of interest

There were no conflicts of interest.

References