408

Joussain C[1], Levy J[1], Charlanes A[2], Even Schneider A[2], Falcou L[2], Chartier Kastler E[3], Denys P[4]
1. *Medical School Paris Île-de-France Ouest, Inserm U1179, Versailles Saint-Quentin University, Versailles, France.,* ***2.*** *Department of Physical Medicine and Rehabilitation, Hôpital Raymond-Poincaré AP-HP, Garches, France,* ***3.*** *Pitié-Salpêtrière Academic Hospital, Department of Urology, Assistance Publique-Hôpitaux de Paris, Pierre and Marie Curie Medical School, Paris 6 University, Paris, France,* ***4.*** *Medical School Paris Île-de-France Ouest, Inserm U1179, Versailles Saint-Quentin University, Versailles, France. Department of Physical Medicine and Rehabilitation, Hôpital Raymond-Poincaré AP-HP, Garches, France*

NEUROGENIC DETRUSOR OVERACTIVITY IN PATIENTS WITH HEREDITARY SPASTIC PARAPLEGIAS

Hypothesis / aims of study

Hereditary spastic paraplegia (HSP) represent a clinically and genetically heterogeneous group of neurodegenerative diseases, with a worldwide estimated prevalence of 1.3/100,000 (1). The “pure” form of HSP is a characterized by a progressive length dependent distal axonal degeneration of the corticospinal tract responsible for a progressive spastic paraplegia. HSP clinical presentation also often include lower urinary tract symptoms (LUTS) with an estimated prevalence ranging from 72.4% to 77.6% (2,3). However, clinical and urodynamic evaluation of HSP patient are rarely reported as well as urologic complications. Thus, the aim of this study was to characterize clinical and urodynamic aspects of LUTS following HSP, and to describe treatment and urological complications of LUTS in a large series of HSP patients, in order to improve management of these patients.

Study design, materials and methods

We performed a monocentric retrospective evaluation based on review of medical records of HSP patients admitted in the department of Physical Medicine and Rehabilitation from January 1999 to January 2016. Inclusion criteria were patients older than eighteen years old presenting HSP. Clinical, urodynamic and radiologic (ultrasonography) data were recorded. Patients with any other documented neurological or urological disorder were excluded from the study. Statistical analysis were performed using Microsoft Excel software.

Results

We have conducted a retrospective study of 33 HSP patients between 1999 and 2015. Mean age of the patients was 62 ±14 years with 70% of men. The mean follow-up time was 8.1 ± 5 years.
The initial complaint of the patients was lower urinary tract symptoms including urgency, hesitancy, nocturia and urinary incontinence (Table1). The PVR was over 100ml in 70%, leading to the diagnosis of chronic urinary retention with an indication for clean intermittent self-catheterizations (CISC). Bowel disorders were present in 45% of the patients and only one patient was treated for erectile dysfunction.

Symptoms	%
Urgency	85
Hesitancy	85
Chronic urinary retention (post-void residual volume was over 100ml)	70
Urinary incontinence	69
Nocturia	56
Frequency	54

Table 1: Lower urinary tract symptoms in HSP patients

Urodynamic findings	%
DO	83
DSD	76
PDetMax > 40cmH2O	68
BH (< 20cmH2O)	10

Table 2: Urodynamic findings in HSP patients
DO: detrusor overactivity; DSD: detrusor- sphincter-dyssynergia; PDetMax: Maximal detrusor pressure; BH: bladder hypo-compliance

NDO was diagnosed according to urodynamic testing in 83% of patients, associated in 76% of them with a DSD and in 85.5% of them with a PVR > 100ml. DSD was associated with urinary retention in 75% of cases. Functional bladder capacity was 328+/-198 ml and urethral closure pressure was 95+/-50 cmH_2O. In all patients with a functional bladder capacity below 300ml (n=13), both DO and DSD were present. (Table 2).

HSP patients with NDO were exposed to urologic and nephrologic complications. Indeed, we found 25% of febrile infections, 21% of urolithiasis and 8% of hydronephrosis (assessed by bladder and kidney ultrasonography). Seventeen percent of patients suffered from renal failure (defined by a creatinine clearance below 60 ml/min). Furthermore, this figure could be under-estimated because only 54% of the patients performed their analysis (Table 3).

Uro-nephrologic complications of NDO	%	Treatment	%
Febrile urinary infection	25	Anticholinergics	69
Renal failure	17	Intradetrusor botulinum toxin	10
Uro-lithiasis	21	Clean intermittent catheterization	29
Hydronephrosis	8	Non continent urinary diversion (Bricker)	13
Bladder cancer	3		

Table 3: NDO complications and treatment in HSP patients

HSP patients have been mainly treated with anticholinergics with 10% being switched to intradetrusor botulinum toxin. These treatments were combined with clean intermittent bladder catheterization, often associated with spontaneous micturition, in only 30% of patients despite the prevalence of PVR > 100 ml was high. Three patients underwent a non-continent surgical urinary diversion (Bricker's technique), one for bladder cancer and two for severe urinary incontinence refractory to proper medical management (Table 3).

Interpretation of results

Our clinical and urodynamic results show a significant percentage of HSP patients presenting LUTS mainly resulting of the association of PVR, DO and DSD (2,3). However, for the first time, our study underlines uro-nephrologic complications following LUTS related to HSP. Thus, we may consider, that HSP patients present a NDO comparable to SCI patients. They are exposed to the same uro-nephrologic complications, which could be prevent by the same management proposed to SCI, including intradetrusor botulinum toxin A delivery and surgery to prevent deleterious high detrusor pressure.

Concluding message

Owing to their hight prevalence and their potential complications, LUTS in HSP patients should be assessed, such as in SCI patients, in order to improve our management allowing an improvement of HSP patients' quality of life and a decrease of uro-nephrologic complications.

References

1. 1. Lo Giudice T, Lombardi F, Santorelli FM, Kawarai T, Orlacchio A. Hereditary spastic paraplegia: Clinical-genetic characteristics and evolving molecular mechanisms. Experimental Neurology. nov 2014;261:51839.
2. 2. Braschinsky M, Zopp I, Kals M, Haldre S, Gross-Paju K. Bladder dysfunction in hereditary spastic paraplegia: what to expect? J Neurol Neurosurg Psychiatr.2010;81(3):2636.
3. 3. Fourtassi M, Jacquin-Courtois S, Scheiber-Nogueira MC, Hajjioui A, Luaute J, Charvier K, et al. Bladder dysfunction in hereditary spastic paraplegia: a clinical and urodynamic evaluation. Spinal Cord. 2012;50(7):55862.

Disclosures

Funding: NONE **Clinical Trial:** No **Subjects:** HUMAN **Ethics not Req'd:** Retrospective study **Helsinki:** Yes **Informed Consent:** No