

The Role of Sonography Videourodynamic Studies in Differentiation of Patients with Lower Motor Neuron Lesion from Upper Motor Neuron Lesion

Gaoyu Pan^{1,2#}, Jiacong Chen^{1,2#}, Yanbin Luo^{3,4*}, Ning Xiao^{1,2,3,4*}

¹Department of Urology, The Second Affiliated Hospital of Guilin Medical University, Guilin, China

²The Laboratory of Urodynamic Technology and Basic Research, The Second Affiliated Hospital of Guilin Medical University, Guilin, China

³Continence Research Clinic, Shaoyang Central Hospital, Shaoyang, China

⁴Department of Urology, Shaoyang Hospital Affiliated to University of South China, Shaoyang, China

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#These authors contributed equally.

*Corresponding author: Yanbin Luo (Shaoyang Central Hospital, Shaoyang, China)

Ning Xiao (The Second Affiliated Hospital of Guilin Medical University, Guilin, China)

Abstract

Original Research Article

Objectives: Differentiation of upper motor neuron lesion (UMNL) from lower motor neuron lesion (LMNL) has been proven to play an important role in decision of therapy protocol for patients with spinal cord injury (SCI). This study was to explore the capacity of sonography videourodynamic studies (SVUDS) for detecting difference between UMNL and LMNL. **Methods and Patients:** Clinical data of male patients with SCI and NLUTD underwent SVUDS from Oct 2020 to Oct 2024 in the Second Affiliated Hospital of Guilin Medical University were retrospectively reviewed. All patients recruited were divided into two groups, in which SCI patients with lesion above S1 were grouped into UMNL and below S1 into LMNL. Age, Parameters of SVUDS, including maximum detrusor pressure (Pdet.max), post-voiding residual (PVR), bladder outlet obstruction index (BOOI), and bladder wall thick (BWT), and incidence of detrusor sphincter dyssynergia (DSD) and flaccid bladder (FB), presenting hypotonic areflexic detrusor, were compared between UMNL and LMNL. **Results:** Although age, Pdet.max, BOOI, and BWT of UMNL group was not respectively statistically different from that of LMNL, significant larger PVR was found in LMNL compared to UMNL. A higher incidence of DSD and a lower prevalence of FB in male patients with UMNL were discerned compared to that of LMNL. **Conclusions:** Only PVR rather than age, Pdet.max, BOOI, and BWT was found to be a non-invasive parameter of SVUDS to differentiate UMNL from LMNL in this study. All DSD presented in UMNL patients and hypotonic areflexic detrusor was mostly derived from LMNL, which could be detected conveniently using SVUDS without fear of radiological risk.

Keywords: Spinal cord injury; neurogenic lower urinary tract dysfunction; upper motor neuron lesion; lower motor neuron lesions; sonography videourodynamic studies.

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INTRODUCTION

Pelvic organ dysfunction, encompassing lower urinary tract dysfunction (LUDT), sexual and bowel dysfunction, is a common sequel of spinal cord injury (SCI) patients [1]. Neurogenic LUDT (NLUTD) has been regarded as a life-threatening risk for SCI patients in chronic phase due to recurrent urinary tract infection (UTI) and chronic kidney impairment (CKI) attributed to a high intravesical pressure and vesicoureteral reflex (VUR) during storage and micturition [2]. However, formulation of an individual therapy protocol becomes

an arduous task on account of the limitation in standard treatment modalities due to varied presentation of NLUTD in SCI patients depending on the level of SCI and times gone by [3].

Different clinical condition of NLUTD has been proved based on lesions of nervous systems, including suprapontine, infrapontine-suprasacral and sacral cord. SCI patients with lesions of suprapontine often complained urgency, frequency, and urgent urinary incontinence due to reduced bladder capacity and detrusor overactivity (DO) although coordination

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between detrusor contraction and external urethral sphincter still was normal during voiding phase [4]. There will be a high intravesical pressure, a high detrusor leak point pressure (DLPP), and VUR, which may lead to intractable UTI, hydronephrosis and eventually end-stage renal disease, due to detrusor sphincter dyssynergia (DSD) during micturition in SCI patients with infrapontine-suprasacral lesion. Voiding dysfunction, such as detrusor underactivity (DO), acontractile detrusor, and flaccid bladder (FB), is not uncommon in SCI patients with lesions of sacral cord or infrasacral pathways. [5, 6].

Since the negative effects of NLUTD on patients' health and quality of life (QOL), there has been great investigation in early diagnosis and individual management of NLUTD to achieve none other than realization of safe urinary continence, improvement of QOL, avoidance of UTI and preservation of upper urinary function. Given that variable presentation of NLUTD with time gone by in SCI patients, in whom urological complication, such as high intravesical pressure, VUR, recurrent UTI, urolithiasis, hydronephrosis, and chronic renal failure, has been reported to occur in no less than nearly half of SCI patients, regular following-up for NLUTD and adequate treatment should be accessed in these patients with high-risk of deterioration in upper urinary tract function [5]. Although physical examination, urinalysis, blood chemistry, and morphological detection of urinary tract could be used to discern these urological complications, videourodynamic studies (VUDS) has still been regarded as "the golden standard" of diagnosis for high-risk patients [7].

Beside brain injury, upper motor neuron lesion (UMNL), which is defined as a spinal cord injury above the S1 level, and lower motor neuron lesion (LMNL), in which the S1-S4 were involved in the sacral paths, have been used to classify the SCI depending on the level of lesions. Detection of varied characterization of VUDS between UMNL and LMNL is crucial for individual therapy protocol and following-up plan in SCI patients [8]. However, only the leading neurourological centers could conduct VUDS due to high investment in VUDS facility and radiological risk during VUDS for patients and physicians, therefore easy accession of VUDS was not common in developing countries across world.

In our previous studies, sonography videourodynamic studies (SVUDS), which could integrate synchronically sonographic images with data of urodynamic studies (UDS), was used to differentiate SCI patients with neurogenic lower urinary tract symptoms (NLUTS) from patients with benign prostate hyperplasia (BPH) and non-neurogenic lower urinary tract symptoms (NNLUTS) [9]. The difference of SVUDS between UMNL and LMNL was explored in this study due to paucity of reports focusing this field in the literature.

METHODS AND PATIENTS

Clinical data of male patients with SCI and NLUTD underwent SVUDS from Oct 2020 to Oct 2024 in the Second Affiliated Hospital of Guilin Medical University were retrospectively reviewed. The exclusion criterions of male patients with NNLUTS were voiding volume <150 mL, previous prostatic surgery, urethral stenosis, utilization of medication that affects micturition, proven prostate or bladder carcinoma, and pelvic radiotherapy. Patients with abnormal urinalysis received treatment prior to SVUDS.

Urodynamic data and sonographic images could be integrated synchronically by a software (UDS.V14) using SVUDS, combining a sonography scan with multichannel UDS (Aquarius XT, Laborie, USA). Bladder wall thick (BWT) was detected using transabdominal ultrasound scan (DC-65, Mindray, China) at a bladder volume of about 150-200 mL during SVUDS and post-voiding residual (PVR) was measured after voiding using 4-MHz curvilinear probe according to our previous reports [9-11]. Peak flow rate (Qmax), detrusor pressure at Qmax (Pdet.Qmax), BOOI index (BOOI; calculated as $Pdet.Qmax - 2Qmax$) and maximum detrusor pressure (Pdet.max) were obtained during pressure-flow study of SVUDS. All SVUDS were undergone by a urologist in according with the Good urodynamic Practices of International Continence Society [12].

All patients recruited were divided into two groups, in which SCI patients with lesion above S1 were grouped into UMNL and below S1 into LMNL, and normality was analyzed in all parameters, including age, Pdet.max, BOOI, PVR, and BWT. Shapiro-wilk test was used to analyze normality and data was presented as mean \pm standard deviation if normality distribution or median (25th percentile, 75th percentile) if non-normality. Mann-Whitney U test if non-normality or T test if normality was adopted to discern the difference of parameters. The difference in the incidence of DSD or FB between UMNL and LMNL groups was compared using square-chi method. All statistical analyses were performed using SPSS for Windows (version 27.0, IBM Corp., Chicago, IL, USA). A p-value of <0.05 was considered statistically significant.

RESULTS

7 male patients with UMNL and 3 male patients with LMNL were recruited into the present study and the difference of parameters were analyzed using T-test since normality distribution was proved. Although age (52.3 ± 4.5 year), Pdet.max (63.1 ± 24.8 cmH₂O), BOOI (30.4 ± 17.1), and BWT (6.69 ± 3.82 cm) of UMNL group was not respectively statistically different from that of LMNL (53.0 ± 3.0 year; 39.9 ± 9.0 cmH₂O; 26.1 ± 9.7 ; 3.77 ± 1.50) (all $P > 0.05$), significant larger PVR was found in LMNL (633.3 ± 57.7 ml) compared to UMNL (148.6 ± 128.0 ml) ($P < 0.01$) (Table 1). A higher incidence of DSD (100%, 7/7) and a lower prevalence of

FB (0%, 0/7) in male patients with UMNL were discerned compared to that of LMNL (DSD: 33.3%, 1/3; FB: 66.7%, 2/3) (all $P=0.022$) (Table 2). It was suggested that SVUDS has a capacity of differentiation of UMNL

from LMNL. Lower radiological risk for patients and physicians and reduced setup investigation would facilitate generalization of SVUDS in the developing countries.

Table 1: Comparison of age, Pdet.max, BOOI, PVR, and BWT between UMNL and LMNL patients

	UMNL(n=7)	LMNL(n=3)	t	P
Age(year)	52.3±4.5	53.0±3.0	0.246	0.812
Pdet.max(cmH ₂ O)	63.1±24.8	39.9±9.0	1.530	0.165
BOOI	30.4±17.1	26.1±9.7	0.399	0.700
PVR(ml)	148.6±128.0	633.3±57.7	6.133	<0.01
BWT(cm)	6.69±3.82	3.77±1.50	1.246	0.248

Abbreviation: UMNL: upper motor neuron lesion; LMNL: lower motor neuron lesion; Pdet.max: maximum detrusor pressure; BOOI: bladder outlet obstruction index; PVR: post-voiding residual; BWT: bladder wall thick

Table 2: Comparison of incidence of DSD or FB between UMNL and LMNL patients

	DSD	FB
UMNL	7/7	0/7
LMNL	1/3	2/3
χ^2	5.25	5.25
P	0.022	0.022

Abbreviation: UMNL: upper motor neuron lesion; LMNL: lower motor neuron lesion; DSD: detrusor sphincter dyssynergia; FB: flaccid bladder

DISCUSSION

There has been “three high”, including a high incidence, a high cost, and a high disability rate, and “one low”, namely a low age of onset, in the SCI patients derived from high-intensity injuries, infections, tumors, vertebral column degenerative diseases, ischemia-reperfusion injuries, and vascular malformation. 66,374 new cases of SCI annually and 759,302 patients totally in China were reported and patients and their families bear a heavy burden physically psychologically financially [13]. Given that NLUTD during storage phase (detrusor overactivity, DO) and voiding phase (DSD or UD or FB) was the focus of therapy protocol following SCI, discernment of DSD or/and FB in UMNL or LMNL has played an important role in offering the individual treatment due to the fact that although neurological evaluation has been proven to play an important role and urological techniques have been deemed as a research tool in characterizing NLUTD, the pendulum is swinging to diagnosis of actual DSD using VUDS disregard of the site and degree of neurogenic damage [5]. In this study, characterization of UMNL or LMNL was evaluated using SVUDS to discover some non-invasive parameters to differentiate UMNL or LMNL and to explore the prevalence of DSD or/and FB in UMNL or LMNL in real world.

Most of our patients in this study were suffering from high-intensity injuries, such as high fall and traffic accidents, and the youngest patient is 32 years comparable to 39.4±14.3 years reported in Wuhan of a central metropolis in China [14]. Therefore, it was not surprise for similar age between two groups. Although varied presentations of UDS with different levels of SCI and time gone by, an increased Pdet.max might be

resulted from a poor compliance of detrusor or/and bladder obstruction outlet (BOO) and could be a simple surrogate of bladder safety in SCI patients [3]. There was not a statistically higher Pdet.max in UMNL patients compared to LMNL, but numerically increased Pdet.max of UMNL patients to some extent indicated that there may be a higher prevalence of poor detrusor compliance in SCI patients above S1 compared to below S1 based on the finding of analogous BOOI between two groups in this study.

BWT has been used to evaluate the degree of BOO in patients with benign prostate hyperplasia (BPH) but controversial results existed across the literature [15]. Increased BWT is to overcome urethral resistance due to BOO in BPH patients, whereas it is on account of BOO due to DSD and immune response to neurological lesion in SCI patients. In line with most of researches, in which BWT could not classify the grade of BOO, it had not a capacity of distinguishing BOO due to DSD from immunologically changes of bladder wall in this study. However, larger PVR was discerned in patients with LMNL compared to UMNL. Although PVR could be generated by BOO, detrusor underactivity (DU) has been deemed to play a more important role than BOO in pathogenesis of large PVR of more than 200ml. Hence, 633.3±57.7ml of PVR in LMNL patients indicated that significantly higher prevalence of DU might present in SCI patients below S1 than above S1. It was suggested that PVR could be used as a non-invasive parameter of SVUDS for differentiation of UMNL from LMNL.

It has come to a consensus that there would be DSD if injury above S1 or DU if below S1 in complete SCI patients, but no agreement has reached about presentation of DSD or/and DU in incomplete SCI

patients [3]. DSD presentation of increased urethral sphincter activity during detrusor contraction was detected if lesion of SCI was between sacral and pontine level using electromyography (EMG) or VUDS. Additionally, the dyssynergia between smooth muscular of bladder neck and detrusor, namely smooth muscular dyssynergia, would be combined with detrusor external sphincter dyssynergia (DESD) in UMNL patients above T10 level given the sympathetic system of T10-L2 spinal cord segments has been considered to control smooth muscle of bladder neck and proximal urethra [7]. Therefore, the “funnel formation” of bladder neck and proximal urethra could be used to evaluate the completeness of sympathetic regulation in T10-L2 in patients with complete UMNL. In this study, DSD was discerned in all 7 patients with UMNL using SVUDS, in which 3 patients with lesion above T10 presented smooth muscular dyssynergia.

Different from presentation of DSD or/and smooth muscular dyssynergia, there were uncertainties about detrusor compliance, relaxation of urethral sphincter, and bladder neck opening based on completeness of LMNL. In the present study, one patient with complete LMNL presented a decreased detrusor compliance with opening in bladder neck and was not diagnosed with FB. It may be explained that complete denervation of parasympathetic system between S1 and S4 could cause adrenergic innervation to the detrusor conversing β -adrenoreceptor-mediated relaxation to α -adrenoreceptor-mediated contraction due to outgrowth of sympathetic fibers in detrusor. FB, presenting hypotonic areflexic detrusor, was diagnosed in the other two patients with incomplete LMNL. However, the role of sympathetic system in parasympathetically denervation bladder detrusor is still unclear.

There were several limitations in this study. Firstly, the medical data were retrospectively reviewed in this study, hence it made evidence weak than prospective research. Secondly, only male SCI patients were recruited into the present study and the sample was 10 cases, therefore we should be cautious for generalization of the conclusion of this study in clinical practice. In the future, female and male patients with other neurological diseases rather than SCI alone would be included to facilitate generalization of research findings.

CONCLUSIONS

The presentation of neurogenic lower urinary tract dysfunction was on account of completeness, level of neurological lesions, and time gone by. Variable coordination between detrusor and urethral sphincter and inconsistent compliance and contractility of detrusor were found in SCI patients. Only PVR rather than age, Pdet.max, BOOI, and BWT was found to be a non-invasive parameter of SVUDS to differentiate UMNL from LMNL in this study. All DSD presented in UMNL patients and hypotonic areflexic detrusor was mostly

derived from LMNL, which could be detected conveniently using SVUDS without fear of radiological risk.

Declaration of interests

The authors declare no conflict of interest.

Author contributions

Pan GY and XN led in conceptualization and writing the original draft. All other authors contributed to reviewing and editing the manuscript.

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