Urodynamic patterns after traumatic spinal cord injury

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Objectives: To study the correlation between neurological level of spinal injury and bladder functions as detected by urodynamic study.

Study design: Analytical study.

Setting and participants: Seventy individuals with traumatic spinal cord injury (SCI) admitted to the Department of Physical Medicine and Rehabilitation, S.M.S. Medical College and Hospital, Jaipur. Detailed clinical, neurological evaluation as per American Spinal Injury Association Classification and radiological assessment were done along with clinical examination of bladder and urodynamic study.

Results: Out of 65 patients with suprasacral injuries, 53 (81.5%) demonstrated hyperreflexia with or without detrusor sphincter dyssynergia, 6 (9.2%) detrusor areflexia, and 6 (9.2%) had normal bladders, 41 (59.4%) low compliance (<20 ml/cmH₂O), and 47 (72.30%) had high detrusor leak point pressures (>40 cmH₂O). Of the five patients with sacral injuries, one (20%) showed detrusor hyperreflexia, four (80%) detrusor areflexia, and one (20%) had low bladder compliance; all five (100%) had high detrusor leak point pressures.

Conclusions: The correlation between somatic neurologic findings, spinal imaging studies, and urodynamic findings in patients with SCI is not exact. Therefore, bladder management should not completely rely only on clinical bladder evaluation or neurological examination alone, but should always include urodynamic studies.

Keywords: Dyssynergia, Detrusor, Bladder, Neurogenic, Spinal cord injuries, Bladder management, Urodynamic study

Introduction

Urologic complications continue to be an important reason for high morbidity in long-term spinal cord injury (SCI) survivors. Longitudinal studies compiled from rehabilitation centers reveal a high incidence of urinary tract infection (UTI; incidence of bacteremia at least one episode in 1 year being 66.7–100% depending on various bladder management methods),¹ renal stones (8%), bladder stones (36%), vesicoureteral reflux, hydronephrosis, and renal deterioration.^{2–4} These complications can lead to renal failure, death rate from which reported in the 1960s was between 37 and 76%.⁵

According to the International Standards for neurological classification of SCI, completeness or incompleteness of a lesion is determined clinically by the presence or absence of motor and/or sensory function below the lesion at sacral dermatomes or the presence or absence

of deep anal pressure.⁶ The aim of classifying neurogenic bladder is to facilitate better understanding of the pathophysiology behind the clinical findings, and to help in better management. One such classification was given by Bors and Comarr,⁷ who used the site of neurological lesion as the core factor for their classification scheme. It is simple, but it cannot explain the clinical and urodynamic variability exhibited by patients who by neurologic examination alone, seem to have similar lesions. Also changes in bladder or outlet activity may make it impossible to always predict accurately lower urinary tract activity solely on the basis of neurologic lesion. This has led the International Continence Society (1988, 1992, 2002) to classify neurogenic bladder by two functions, as difficulty to store and difficulty to void which gives a broad functional perspective to the classification system.⁸

On a teleological and anatomical basis, when there is injury proximal to the sacral spinal cord, one expects a voiding pattern consistent with upper motor neuron

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injury. In contrast, injury to either the sacral cord or cauda equina segment should result in lower motor neuron injury. To our knowledge, four studies have so far noted an inexact correlation between somatic neurologic findings and characteristic urodynamic findings.^{9–12}

Several factors merit consideration as contribution to this inexact correlation. First, degeneration and reorganization of crucial neural pathways distal to the lesion with or without neural sprouting at the level of injury may affect the neurologic and urodynamic findings. Second, SCI may be incomplete, thereby partially allowing the integration and modulation of complex micturition signals at multiple levels of the nervous system. Multiple injuries coexisting at different levels can result in unpredictable mixed voiding dysfunction. In fact, the multiplicity of levels of injury is occasionally unrecognized when based solely on urologic history and evaluation in patients with new SCI.¹³

The history and physical examination alone cannot determine the type of bladder and sphincter function in a person with SCI. It is imperative that complete urodynamic study be undertaken for specific identification. We evaluated 70 patients with SCI at various levels and tried to find the underlying neurogenic bladder type in these cases.

Methods and materials

Evaluation of the study subjects

All patients underwent routine history and physical examination, including evaluation of perianal sensation, anal sphincter tone, and sacral reflexes along with bladder diary and urodynamic study. The patients were divided into neuroanatomical groups based on the clinical neurological level. Patients were also categorized as complete or incomplete, based on American Spinal Injury Association Classification (ASIA), in which subgroups based on the integrity of the sacral dermatomes were made. Those with abnormalities of the sacral reflex arc (absent bulbocavernosus reflex, and lax anal sphincter tone) were deemed positive for the presence of sacral cord lesion and those with intact sacral reflexes were deemed negative for sacral cord lesion. All the patients considered for the study underwent routine testing including blood counts, renal function tests, urine microscopy, urine culture and sensitivity, radiographs, and diagnostic ultrasound of the urinary tract.

Urodynamic evaluation

Urodynamic evaluation on each patient was performed using Ellipse, which is a Danish-designed German

urodynamic device from Andromeda (Buckinghamshire, UK, established as Mediplus in 1986) placed in our department. It consisted of a filling phase and a voiding phase cystometrogram along with perineal muscle electromyography (EMG). A 9F double-lumen catheter was introduced transurethra-ly into the bladder. One lumen was used for bladder filling at an average flow rate of 21 ml/min (according to ICS guidelines) and another was used to record intravesical pressure. Intra-abdominal pressure was recorded by a 12F rectal catheter. Through multichannel pressure transduction, intravesical and intra-abdominal pressures were simultaneously transduced on a strip chart recorder. Sphincter EMG was performed using patch electrodes (Ambu blue sensor NF) by an experienced electromyographer.

Inclusion criteria

Patients with SCI of more than 3 months duration who were willing to undergo urodynamic evaluation.

Exclusion criteria

All those who were diagnosed with urinary tract stones, foreign body, symptomatic UTI, or any other bladder abnormality were excluded from the study.

Standard definitions

Detrusor overactivity (DO) is a urodynamic observation characterized by involuntary detrusor contractions during the filling phase which may be spontaneous or provoked.¹⁴ Detrusor-external sphincter dyssynergia was defined as the intermittent or continuous involuntary contraction of the urethral sphincter during detrusor contraction.⁸ Bladder compliance was defined as ratio of a change in bladder volume to the associated change in intravesical pressure and is usually obtained from urodynamic study.¹⁵ Low compliance was defined as <20 ml/cmH₂O.¹⁶ Patients who had absence of urodynamic and neurourological abnormalities were further classified as normal. Detrusor leak pressure was defined as the bladder pressure at which there was leakage from urethra without increasing abdominal pressure due to either phasic or tonic muscular activity.¹⁰ Urodynamic terms used in this study conform to the International Continence Society definitions and to the above-mentioned reports.¹⁷

Results

There was a total of 70 individuals with traumatic SCI, of whom 35 (50%) had cervical injury, 16 (23%) had dorsal injury, 14 (20%) had lumbar injury, and 5 (7%) had sacral vertebral injury. Among these individuals, 39 had neurologically complete injuries and 31

Table 1 Neurological level of injury

Neurological level	Complete	Incomplete
Cervical	22	13
Thoracic	12	4
Lumbar	3	11
Sacral	2	3

Figure 1 Urodynamic findings at each level of spinal cord injury. DH, Detrusor overactivity; DSD, Detrusor sphincter dyssynergia; DA, Detrusor areflexia.

incomplete (according to ASIA) at the time of examination (Table 1). The overall relationship between the clinical neurological level and urodynamic findings is noted in Fig. 1. As noted in Table 2, both the individuals with detrusor areflexia had signs of sacral cord involvement. All but one cervical patient with DO and detrusor sphincter dyssynergia were negative for sacral cord lesion. Only one of the 16 patients with thoracic-level injuries had normal urodynamic findings and sacral cord lesion, whereas none of the patients had detrusor areflexia. None of the five patients with lumbar cord injuries with DO and detrusor sphincter dyssynergia had positive sacral cord lesion, whereas all four patients with detrusor areflexia were positive for sacral cord lesion. Of the four patients with sacral cord lesion, one had DO and three (75%) had detrusor areflexia.

All patients with thoracic cord lesions had either DO or detrusor sphincter dyssynergia and no sacral cord lesion. As seen in Table 3, low bladder compliance was seen in 19 (73%) cases with DO, 18 (60%) cases with detrusor sphincter dyssynergia, while in only four (44.4%) cases with detrusor areflexia and one (17%) with normal bladder. Overall, 47 (72.30%) patients

Table 2 Urodynamic findings in spinal cord injury at each level in accordance with sacral cord lesion (SCL)

Neurological level	Detrusor overactivity		Detrusor sphincter dyssynergia		Detrusor areflexia		Normal	
	SCL +	SCL –	SCL +	SCL –	SCL +	SCL –	SCL +	SCL –
Cervical	1	12	0	20	2	0	0	0
Thoracic	0	7	0	8	0	0	1	0
Lumbar	0	3	0	2	4	0	0	5
Sacral	1	1	0	0	3	0	0	0

Table 3 Bladder compliance at various levels of spinal cord injury

	Low bladder compliance			
	Detrusor overactivity	Detrusor sphincter dyssynergia	Detrusor areflexia	Normal
Cervical	13	13	1	0
Thoracic	3	5	0	1
Lumbar	2	0	3	0
Sacral	1	0	0	0

Table 4 Leak point pressure at various levels of spinal cord injury (mmHg)

	Leak point pressures			
	Detrusor hyperreflexia	Detrusor sphincter dyssynergia	Detrusor areflexia	Normal
>40	19	24	7	2
<40	6	6	3	4

with suprasacral cord lesions had high detrusor leak point pressures (Table 4).

Discussion

Subramonian *et al.*,⁴ Bradley and Conway,¹⁸ Fletcher and Bradley¹⁹ stated that normal micturition is a complex neurophysiological event that is dependent on a variety of integrated neuronal pathways connecting the cerebral cortex, brain stem nuclei in the pons, and the sacral spinal cord. They also enumerated that the centers for volitional control of voiding are located in the cerebral cortex and therefore, pathological entities that affect this area of the brain should theoretically result in an overactive bladder, that is DO with loss of voluntary control.^{4,18,19} Reflex detrusor activity is dependent on the integrity of the sacral micturition reflex arc. After recovery from spinal shock, patients with suprasacral injuries should theoretically retain reflex detrusor activity. In contrast, injury to or below the sacral cord should lead to detrusor areflexia.

Our understanding of the overall neural control of micturition can only be advanced if objective assessment of detrusor sphincter dysfunction is combined with accurate neurologic diagnosis maintaining a careful distinction between observed functional derangements and our concepts of the mechanisms involved.

Past classification systems and terminologies used to define neurogenic bladder dysfunction have usually been based on the site and degree of neurologic damage. As urodynamic techniques are no longer regarded as merely a research tool in the investigation of patients with neurogenic bladder dysfunction, the pendulum now appears to be swinging away from such an approach and attention is being focused on the actual detrusor and/or sphincter dysfunction, regardless of the site and degree of neurologic damage. However, the findings of urodynamic studies can be influenced by various factors like UTI, stones etc. We administered appropriate antibiotics to all the individuals suffering from UTI until the final urine was sterile. Medications known to interfere with urodynamic results were stopped for adequate time before carrying out the urodynamic evaluation. Neurourological and urodynamic evaluation can help to objectively define the neurologic deficit of the bladder. Patients with severe spasticity in the lower extremities, invariably, also present with a spastic pelvic floor and external urethral sphincter along with severe detrusor sphincter dyssynergia. Understanding of the basic neurologic lesion and bladder dysfunction is vital to bladder retraining or transurethral surgery to provide adequate voiding.

A poorly compliant bladder distends with high intravesical pressure at relatively low volumes, and may lead to vesicoureteral reflux and places the upper urinary tract at even greater risk for deterioration, as has been explained by Weld *et al.*,²⁰ Hackler *et al.*,²¹ and McGurie *et al.*²² Samson *et al.*¹⁵ said that a highly compliant bladder is associated with hyporeflexive or areflexive bladder as seen in lower motor neuron injuries, which is in close agreement to the findings in our study in which 37 (88%) patients with low bladder compliance had either DO or detrusor sphincter dyssynergia. But our findings do not match with the findings of Weld and Dmochowski, who demonstrated a higher frequency of impaired compliance in the sacral injury group, which can be explained on the basis of a relatively smaller sample size in our study.¹⁰ Hackler *et al.*²¹ and McGurie *et al.*²² have correlated low bladder compliance with upper tract complications and decreased upper tract function. A poorly compliant bladder increases the ureteral workload and compromises upper tract drainage. High detrusor leak point pressures are known to correlate with low bladder compliance as noted by Ghoniem *et al.*¹³ in their study. This is in close agreement to our study in which 34 (85%) out of a total of 42 patients with low compliance had high detrusor leak point pressures. All complications of untreated external detrusor sphincter dyssynergia result from high intravesical pressure prior to urinary leakage. As is recommended by McGurie *et al.*²² the pressure must be <40 cmH₂O in a neurogenic bladder to minimize upper tract damage.

Weld and Dmochowski¹⁰ and Kaplan *et al.*¹² concluded that detrusor sphincter dyssynergia is a common occurrence in supra sacral spinal cord lesions. It is clinically characterized by high detrusor pressure, vesicoureteral reflux, and upper tract deterioration.²³ Arnold *et al.*²⁴ reported that all patients with supranuclear injury developed features of detrusor-external sphincter dyssynergia. However, in our study, 28 out of 51 patients had detrusor sphincter dyssynergia. In that series, the magnitude of the detrusor contraction was related to the completeness of the lesion; individuals with a complete lesion had higher detrusor pressures than did those with an incomplete lesion. Other authors have noted that detrusor areflexia may occur with upper motor neuron lesions, presumably due to a coexistent clinical or subclinical spinal cord lesion. This was evident in our study, in which two (5.71%) of the patients with cervical cord lesions had persistent detrusor areflexia, but 8.57% had signs of a sacral cord lesion. Arnold *et al.* reported two cases of upper motor neuron lesions, presumably due to a coexistent

clinical or subclinical sacral spinal cord lesion. Seong *et al.*²⁵ enumerated that pelvic floor EMG has been extremely useful in detecting lower motor neuron lesions of the sacral cord when there is any doubt as to its presence. Arnold *et al.*²⁴ reported two cases of upper motor neuron lesions with detrusor areflexia. Light *et al.*²⁶ reported on 13 patients with suprasacral SCI and detrusor areflexia who were evaluated with somatosensory testing and sacral evoked potentials. In our series, since somatosensory testing and evoked potentials were not performed, one can only speculate on the incidence of subclinical sacral cord injury.

Most of the patients with traumatic thoracic cord lesions in our study had complete lesions. In fact, in our series, all patients with traumatic thoracic cord lesions had either DO or detrusor sphincter dyssynergia and negative sacral cord signs except one who had normal bladder function. Patients with mixed cord lesions or simultaneous thoraco-lumbar injury have less predictable voiding dysfunction, as noted by Yalla and Andriole.²⁷

Wyndaele¹¹ in their study of 92 patients with SCI concluded that clinical examination for detailed diagnosis of neurological bladder is insufficient and urodynamic tests are needed for a profound evaluation of the function of different parts of lower urinary tract. It is clear from urodynamic findings in our patients that many with spinal cord lesions do not have well defined, complete lesions. Although the majority of patients demonstrated consistent bladder and sphincter behavior based on the neurologic deficit, this is by no means absolute. In addition, the presence or absence of abnormalities on the neurourological examination was helpful but not always predictive. Thus, one should be wary of predicting urological dysfunction based solely on neurological injury. Thorough attempts to evaluate bladder and sphincter behavior should be done before appropriate therapy is instituted.

Conclusion

Bladder function differs according to the level of injury. It is, therefore, important to define the neurological lesion to appreciate the voiding dysfunction and thereby to develop an appropriate management plan for long-term urologic care. Bladder management should be directed towards protection of upper tracts, prevention of infection, autonomic dysreflexia, and encouraging bladder emptying at low pressures, as the first and foremost goal. Creating a system for bladder control, drainage, and maintenance of continence and convenience throughout patient's lifetime should be our additional goals. Inferences from neurologic

examination may be incorrect because of the superimposed complexity of multiple injury levels. Despite consistent data regarding classic voiding dysfunction with complete injuries, multiplicity of injury may contribute to complicated urodynamic findings. Therefore, urodynamic evaluation is crucial to correctly identify the type of voiding dysfunction and to optimize long-term management.

Disclaimer statements

Contributors No role has been played by any other person other than authors.

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