Factors associated with Hydronephrosis and Vesicoureteral Reflux in Spinal Cord Injured Patients

Suksathien R,[1] Ingkasuthi K[1] and Bumrungna S[2]

[1]Department of Rehabilitation Medicine, [2]Department of Radiology, Maharat Nakhon Ratchasima Hospital, Nakhon Ratchasima, Thailand

ABSTRACT

Objectives: To investigate factors associated with hydronephrosis and vesicoureteral reflux (VUR) indicating upper urinary tract deterioration in spinal cord injured (SCI) patients.

Setting: Maharat Nakhon Ratchasima Hospital.

Study design: Retrospective study.

Subjects: SCI patients admitted at Rehabilitation ward between August 2008 and July 2018.

Methods: Medical records of the subjects were reviewed retrospectively. General demographic, urological and imaging data including hydronephrosis, VUR and bladder deformity were reviewed. Associations between all variables and hydronephrosis/VUR were identified.

Results: Of 278 patients, 30.2% had hydronephrosis and 24% had VUR. The most common bladder management was indwelling catheterization (66.5%) followed by triggered reflex voiding and incontinence (16.5%), voluntary voiding and continence (10%), combined voiding with clean intermittent catheterization (CIC) (4%), and CIC alone (2.9%). Bivariate analysis showed that age at onset, completeness of lesion, time interval from onset of injury, bladder management, antimuscarinic medication used, detrusor hyperreflexia, poor bladder compliance, small bladder volume, detrusor pressure (Pdet) > 40 mmHg and high grade bladder deformity were significantly associated with hydronephrosis and VUR. Five variables correlated with hydronephrosis/VUR were triggered reflex voiding with incontinence, indwelling catheterization, anticholinergic medication, high grade bladder deformity and time interval from onset of injury with adjusted odds ratios (95%CI) of 8.90 (2.50, 31.62), 7.23 (2.22, 23.59), 2.00 (1.11, 3.70), 1.82 (1.01, 3.30) and 1.01 (1.00, 1.02) respectively in multivariate analysis.

Conclusion: Triggered reflex voiding with incontinence and indwelling catheter were a strong predictor of hydronephrosis and/or vesicouretheral reflux whereas other factors such as taking antimuscarinic medication, high grade bladder deformity and time interval from onset of injury could be a less predictive factor in patients with spinal cord injury. Therefore, trigger reflex voiding and long-term indwelling catheter should not be recommended, and thus, those using such bladder emptying techniques should be closely monitored with regular urological check-up to early detection of upper urinary tract deterioration.

Keywords: spinal cord injury, vesico-ureteral reflux, hydronephrosis, urodynamics, neurogenic lower urinary tract dysfunction

ASEAN J Rehabil Med. 2019; 29(2): 51-57.

Introduction

Neurogenic lower urinary tract dysfunction (NLUTD) is a common impairment in spinal cord injured (SCI) patients leading to many serious complications such as urinary tract infection (UTI), renal calculi, vesicoureteral reflux (VUR) and hydronephrosis. Hydronephrosis and VUR cause renal deterioration which is one of the most common causes of death in SCI patients. Several studies investigated the risk factors of renal deterioration in patients after SCI[1-6] and spina bifida[6,7]. Many physicians and nurses recommended trigger reflex voiding without urodynamic data and long-term complications were commonly found. The objective of this study was to investigate factors associated with hydronephrosis and VUR which might be useful for the prediction of renal deterioration in spinal cord injured patients.

Materials and Methods

Electronic medical records and imaging results of all patients with SCI admitted at the Rehabilitation ward, Maharat Nakhon Ratchasima Hospital between August 2008 and July 2018 for urological check-up were reviewed retrospectively. The data from each check-up were divided into 3 parts: general demographic, urological and imaging data.

The general demography including gender, age at onset, level of injury, completeness and cause of SCI, time interval from onset to urological evaluation and ambulatory level were recorded. A previous study) stated that injury at T10-L2, where the sympathetic intermediolateral nuclei that mediate sphincter relaxation during voiding are located, is associated with the highest incidence of VUR.[2] The authors categorized level of SCI into 3 groups: cervical to thoracic 9, thoracic 10 to lumbar 2[2] and lumbar 3 to sacral. The ambulatory level was classified into ambulatory (score of 3 or greater) and non ambulatory (score less than 3) according to mobility for moderate distance subscale of Spinal Cord Independence Measure version III.[8]

The urological data, including type of bladder management: indwelling catheterization, clean intermittent catheterization (CIC), voluntary voiding with continence, triggered reflex voiding with incontinence and voiding with CIC; antimuscarinic medication used and dosage, a history of UTI and upper tract calculi (renal and ureteric calculi) were reviewed. Urodynamic test or cystometry were performed in some patients. The para-

Correspondence to: Rachawan Suksathien, MD, FRCPhysiatrT; Department of Rehabilitation Medicine, Maharat Nakhon Ratchasima Hospital, Chang Phrueak Rd, Mueang District, Nakhon Ratchasima 30000, Thailand; E-mail:rachawan.su@cpird.in.th

Received: 28th June 2019 Revised: 5th August 2019 Accepted: 9th August 2019

meters such as detrusor overactivity, bladder compliance, maximum cystometric capacity and detrusor pressure were recorded. Bladder compliance was defined by dividing the change in volume by the change in detrusor pressure, where <20 mL/cmH_2O indicated low compliance.[9] Bladder capacity less than 250 mL[10] was identified as small bladder capacity. Detrusor pressure more than 40 cmH_2O during filling phase defined as high detrusor pressure (Pdet >40).[6,7]

The imaging data including hydronephrosis, VUR and bladder deformity were assessed by radiologists. Hydronephrosis was detected from ultrasonography or IVP whereas VUR was detected from voiding cystourethrography (VCUG) or cystography. All patients with both hydronephrosis and VUR were included. Those with only a single imaging data were excluded. Bladder deformity was evaluated by a radiologist, and classified into 4 grades (grade 0 to 3) according to Ogawa T.[11] For statistical analysis, bladder deformity was grouped into low (grade 0-1) and high grade (grade 2-3) deformity. Hydronephrosis and VUR were selected as an indication of renal deterioration.

To be noted, there was more than one annual urological check-up in most of the patients, there were different data from every check-up. Only one data set was selected for analysis with the same criteria. Because the objective of this study was to investigate factors associated with hydronephrosis and VUR, the authors chose the data from first time detected hydronephrosis or VUR whereas the complete or the latest data were selected for those with negative results.

Statistical analyses were studied using Student's t-test, Fisher's exact test and Mann-Whitney U test. Variables with p <0.25 bivariate evidence of association with hydronephrosis/VUR were then evaluated using backward stepwise logistic regression and area under curve. p< 0.05 was considered to indicate significance. The odds ratio (OR) shows the magnitude of association between variables and hydronephrosis or VUR. Because some patients did not have urodynamics data, 2 models of multivariate analysis were investigated: model A excluded the urodynamics data whereas model B included the urodynamics data.

Remark: The present study was approved by the Ethics Committee of Maharat Nakhon Ratchasima Hospital (No.114/2018).

Results

Three hundred and twenty-eight SCI patients with 856 ultrasonographs, 682 cystograms or VCUG and 148 urodynamic studies were evaluated. After selection according to the criteria, 278 patients were included in this study. The clinical characteristics of the studied population are shown in Table 1. The mean age at onset of injury was 40.7 years (range, 3-78). Seventy-nine percent were males. Forty-five percent of the patients had SCI at cervical to T9, 41% T10 to L2 and 14% L3 to sacral level. Half of them had complete lesion, and 73% of injuries were caused by trauma. There were 31% who could walk for a moderate distance.

There were 5 types of bladder management in this population. The most common bladder management was indwelling catheterization (66.5%) followed by triggered reflex voiding with incontinence (16.5%), voluntary voiding with continence (10%), voiding with CIC (4%) and CIC (2.9%). Antimuscarinics were prescribed to 34.5% of the patients and Oxybutynin was the most prescribed medication in this study. Of 278 patients, 104 (37.4%) had urodynamic/cystometry results. Just over half of this group (52.9%) had detrusor overactivity, 61.5% had low bladder compliance, 40.4% had Pdet >40 and 14.39% had small bladder capacity (<250 mL).

Hydronephrosis and VUR were found in 84 (30%) and 63 (24%) patients. When categorizing bladder deformity into 2 groups, there were 40% and 60% with high and low grade deformity. Twenty-seven percent had a history of UTI. Seven patients (2.52%) had upper tract calculi.

Bivariate associations between variables and hydronephrosis/VUR are shown in Table 2. Age at onset, completeness of lesion, time interval from onset of injury, bladder management, antimuscarinic medication, detrusor overactivity, low bladder compliance, cystometric capacity, Pdet >40 and high grade bladder deformity showed a significant association with hydronephrosis/VUR.

Unadjusted and adjusted odds ratio of hydronephrosis/VUR are shown in Table 3. Because of low numbers for urodynamic data (104 patients or 37.4%), 2 models of multivariate analysis were investigated. Model A (n=174) excluded the urodynamic/cystometric data whereas model B (n=104) included the urodynamic/cystometric data. Five variables correlated with hydronephrosis/VUR were triggered reflex voiding with incontinence, indwelling catheterization, taking antimuscarinic medication, high grade bladder deformity and time interval from onset of injury with adjusted odds ratios of 8.9 (2.5, 31.62), 7.23 (2.22, 23.59), 2 (1.11, 3.7), 1.82 (1.01, 3.3) and 1.01 (1, 1.02) respectively in model A (Pseudo R2=11.05%). Three variables correlated with hydronephrosis/VUR were triggered reflex voiding with incontinence, high grade bladder deformity and time interval from onset of injury with adjusted odds ratios of 17.49 (1.03, 296.14), 4.18 (1.12, 15.60) and 1.02 (1.00, 1.03) respectively in model B (Pseudo R2=26.66%).

Discussion

Neurogenic lower urinary tract dysfunction (NLUTD) is a common impairment in SCI patients and leads to upper urinary tract (UUT) deterioration. In the present study, hydronephrosis and/or VUR (hydronephrosis/VUR) were used as indicators for UUT deterioration and considered general demographic and urological data such as bladder management, antimuscarinic

Table 1. Clinical characteristics of the stuided population (n=278)

Characteristics	
Mean age at onset (year)[1]	40.7 (16.4)
Gender: male, n (%)	219 (78.8)
Level of spinal cord injury[2]	
Cervical – T9	125 (45)
T10 – L2	115 (41.4)
L3 – Sacral	38 (13.7)
Cause of spinal cord injury: Trauma[2]	204 (73.4)
Complete lesion[2]	140 (50.4)
Ambulatory level: ambulatory[2]	86 (30.9)
Hydronephrosis[2]	84 (30.2)
Vesicoureteral reflux (VUR)[2]	63 (24.0)
Time interval from onset of injury (month)[3]	5 (37, 1-265)

T, thoracic; L, lumbar

[1]Mean (SD), [2]number (%), and [3]median (IQR, range)

Table 2. Bivariate associations with hydronephrosis/VUR

Factors	Hydronephrosis/VUR (-) (n=152)	Hydronephrosis/VUR (+) (n=126)	*p* value
Age at onset (years)[1]	42.8 (15.8)	38.2 (16.9)	0.021*
Gender[2]			
Female	38 (25)	21 (16)	0.106
Male	114 (75)	105 (83.3)	
Level of spinal cord injury[2]			
Cervical – T9	72 (47.4)	53 (42.1)	0.659
T10 – L2	61 (40.1)	54 (42.9)	
L3 – Sacral	19 (12.5)	19 (15.1)	
Cause of spinal cord injury[2]			
Non-trauma	42 (27.6)	32 (25.4)	0.685
Trauma	110 (72.4)	94 (74.6)	
Completeness of lesion[2]			
Incomplete	87 (57.2)	51 (40.5)	0.006*
Complete	65 (42.8)	75 (59.5)	
Ambulatory[2]			
Yes	51 (33.6)	35 (27.8)	0.362
No	101 (66.5)	91 (72.2)	
Time interval from onset of injury (months)[3]	32 (4)	52 (9)	0.002*
Bladder management[2]			
Indwelling catheterization	98 (64.5)	87 (69.1)	0.006*
CIC	4 (2.6)	4 (3.2)	
Void with CIC	8 (5.3)	3 (2.4)	
Triggered reflex voiding with incontinence	19 (12.5)	27 (21.4)	
Voluntary voiding with continence	23 (15.1)	5 (4)	
Antimuscarinic medication[2]			
Yes	39 (25.7)	57 (45.2)	0.001*
No	113 (74.3)	69 (54.8)	
Dosage of Oxybutynin[1]	10.26 (1)	12.63 (1)	0.11
UTI[2]			
Yes	39 (25.7)	36 (28.6)	0.590
No	113 (74.3)	90 (71.4)	
Upper tract calculi[2]			
Yes	5 (3.3)	2 (1.6)	0.462
No	147 (96.7)	124 (98.4)	
Bladder deformity grading[2]			
Low grade (grade 0-1)	91 (65.5)	60 (52.6)	0.040*
High grade (grade 2-3)	48 (34.5)	54 (47.4)	
Detrusor hyperreflexia[2] (n=104)			
Yes	12 (32.4)	43 (64.2)	0.002*
No	25 (67.6)	24 (35.8)	
Low bladder compliance[2] (n=104)			
Yes	15 (40.5)	49 (73.1)	0.002*
No	22 (59.5)	18 (26.9)	
Cystometric capacity (n=104)			
≤ 250 mL	9 (24.3)	31 (46.3)	0.036*
> 250 mL	28 (75.7)	36 (53.7)	
Pdet ≥ 40 cmH_2O[2] (n=104)			
Yes	8 (21.6)	34 (50.8)	0.006*
No	29 (78.4)	33 (49.3)	

T, thoracic; L, lumbar; CIC, clean intermittent catheterization; VUR, vesicoureteral reflux; UTI, urinary tract infection
[1]Mean (SD), [2]number (%), and [3]median (IQR); *statistical significance

Table 3. Unadjusted and adjusted odds ratios of hydronephrosis/VUR

Factors	Unadjusted odds ratio (95% CI)	Adjusted odds ratio (95%CI) of model A (n=174)	Adjusted odds ratio (95%CI) of model B (n=104)
Age at onset			
< 20 years	2.53 (1.19, 5.40)		
20-40 years	1.15 (0.69, 1.93)		
> 40 years (reference)	1		
Level of spinal cord injury			
Cervical – T9	0.74 (0.36, 1.52)		
T10 – L2	0.89 (0.42, 1.84)		
L3 – Sacral (reference)	1		
Complete lesion	1.97 (1.22, 3.18)		
Non-ambulatory	1.31 (0.78, 2.20)		
Time interval from onset of injury (months)	1.01 (1.00, 1.02)	1.01 (1.00, 1.02)*	1.02 (1.00, 1.03)*
Bladder management:			
Indwelling catheterization	4.08 (1.49, 11.20)	7.23 (2.22, 23.59)*	7.77 (0.80, 75.11)
CIC	4.60 (0.85, 24.93)	3.14 (0.49, 20.10)	1.15 (0.06, 21.54)
Void + CIC	1.72 (0.33, 8.91)	1.77 (0.30, 10.53)	3.9 (0.23, 65.32)
Triggered reflex voiding with incontinence	6.54 (2.11, 20.26)	8.90 (2.50, 31.62)*	17.49 (1.03, 296.14)*
Voluntary voiding with continence (reference)	1		
Antimuscarinic medication	2.38 (1.45, 4.00)	2.00 (1.11, 3.70)*	1.22 (0.35, 4.19)
UTI	1.16 (0.68, 1.97)		
High grade bladder deformity	1.71 (1.03, 2.83)	1.82 (1.01, 3.30)*	4.18 (1.12, 15.60)*
Detrusor overactivity	3.73 (1.59, 8.74)		2.95 (0.53, 16.29)
Low bladder compliance	3.99 (1.71, 9.34)		3.53 (0.56, 22.22)
Bladder volume < 250 mL	2.68 (1.10, 6.53)		0.63 (0.13, 2.98)
Pdet ≥ 40 cmH_2O	3.73 (1.49, 9.35)		0.71 (0.10, 5.01)

Model A, without urodynamic/cystometric data; model B, with urodynamic/cystometric data

T, thoracic; L, lumbar; CIC, clean intermittent catheterization; UTI, urinary tract infection; Pdet, detrusor pressure

*Statistical significance at $p<0.05$

medication, a history of UTI, abnormal urodynamic findings and bladder deformity as potential risk factors for hydronephrosis/VUR. From the results, there were 5 variables correlated with hydronephrosis/VUR: triggered reflex voiding with incontinence, indwelling catheterization, antimuscarinic medication, high grade bladder deformity and time interval from onset of injury.

Type of bladder management was the most important factor associated with UUT deterioration. Many studies demonstrated that indwelling catheterization was associated with urinary tract complications such as UTI,[12] low creatinine clearance,[3] proteinuria,[3] VUR,[3,13] hydronephrosis,[13,14] and renal calculi.[14] Consistent with our study, we found that indwelling catheterization was the second most important risk factor associated with UUT deterioration in model A with the odds ratio of 7.23. In our service, indwelling catheterization remains the most common form of bladder management in our patients (66.5%). According to a study of quality of life (QoL) of SCI patients done in Thailand, Pongboriboon P et al. reported no difference in QoL between those using indwelling catheter and those using CIC; and the only reason of choosing indwelling catheter was convenience.[15] On the other hand, CIC was the bladder emptying method that had the lowest complications,[1,4,16] but it has been the least popular bladder management in our setting (2.9%). There are many factors affecting CIC in SCI patients[17-19] such as incontinence between catheterization, autonomic dysreflexia, poor hand function or obesity interfering with the ability to catheterize, patients' knowledge and attitude, and environmental barriers.

Trigger reflex voiding or urinary incontinence was the method that is most controversial. According to a review of conservative bladder management in SCI, trigger reflex voiding is not recommended as it is unphysiologic, causes detrusor-sphincter dyssynergia; and few patients had a balanced voiding with post-void residue less than 100 mL or less than 25% of bladder volume; if used, neuropathic patients should be closely observed and monitored with urodynamic test; and nowadays trigger reflex voiding has been replaced with antimuscarinics and CIC.[20] On the contrary, Ma Y et al. found that urinary incontinence with detrusor overactivity were protective factors for hydronephrosis in spina bifida.[7] DeLair SM et al. and Yildiz N et al. showed that urinary incontinence was not a significant risk factor for upper urinary tract complications[21,22] whereas Gao Y et al. found that condom catheter with reflex voiding was closely related to upper urinary tract complication, consistent with the present study.[1] In this study, triggered reflex voiding with incontinence was the most important risk factor associated with hydronephrosis/VUR from both models of multivariate analysis with the odds ratio of 8.9 in model A and 17.49 in model B. Unfortunately, this bladder emtpying technique was the second most popular in our patients (16.5%).

Antimuscarinic treatment has been widely used for SCI patients with neurogenic detrusor overactivity (NDO). It decreases involuntary detrusor contraction,[23] increases bladder capacity,[23]and compliance[24] and lessens occurrence of hydro-

nephrosis,[24] Vaidyanathan S et al. reported that SCI patients, who did not receive antimuscarinic drugs to reduce intravesical pressure, were at high risk for developing reflux nephropathy.[5] The clinical practice guideline 2006 recommended the use of such medication in those with suprasacral lesion, doing CIC and having urine leakage between catheterization, and also in those having long-term use of indwelling catheter.[25] Surprisingly, the results for taking antimuscarinics in this study were not consistent with the previous studies.[5,23-25] Those taking such medication had a higher risk of hydronephrosis/VUR with an odds ratio of 2. In our opinion it was a coincidence and due to the criteria for data collection. The data set that was included in this retrospective study might not be the first time that such urological abnormalities were detected, and such medications were prescribed because of abnormal urological findings during previous visits.

Bladder trabeculation and deformity of bladder shape were classified into 4 grades from cystography by Ogawa T.[11] Previous studies reported that trabeculation and deformity of bladder were associated with upper urinary tract deterioration (hydronephrosis/VUR).[11,14] Consistent with the present study, high grade bladder deformity was a predictive factor for hydronephrosis/VUR in both models with an odds ratio of 1.82 in model A and 4.18 in model B.

Time interval from onset of injury is one factor correlated with hydronephrosis/VUR in this study with an odds ratio around 1 in both models of multivariate studies. Previous studies showed that injury duration was associated with complications and probability of indwelling catheterization.[1,17-19] Shin JC et al. demonstrated that time interval from injury and time interval between injury and rehabilitative treatment were significantly longer in low bladder compliance patients compared with normal compliance group which may be correlated with inappropriate bladder management before admission.[26] Interestingly, unfavorable urodynamic parameters were detected very early within the first 40 days after SCI.[27] Therefore an early and appropriate bladder management was suggested in SCI patients to prevent upper urinary tract deterioration.

Many variables that showed a significant association with hydronephrosis/VUR in bivariate analysis but not in multivariate analysis such as age at onset, completeness of lesion, detrusor overactivity, low bladder compliance, small bladder volume, and Pdet >40 cmH_2O. Lower age at onset may be accompanied with time interval from onset of injury which has been discussed before. Moreover, when VUR is present, it lowers intravesical pressure; and this might be one reason why Pdet >40 cmH_2O was not associated with hydronephrosis/VUR in this study.

Severity of SCI was an important clinical evaluation in predicting functional outcomes. However, the correlation between severity of injury and upper urinary tract deterioration in NLUTD had conflicting results. Devivo MJ et al. studied the risk of renal calculi in SCI patients and found that complete lesion was a factor associated with renal calculi[28] whereas other studies showed that the severity of lesion failed to determine the bladder type and urological complications.[13,29] Similarly, we found that complete and incomplete lesions were not associated with hydronephrosis/VUR in multivariate analysis. However, whether lesions are complete or incomplete, every SCI with NLUTD should have a regular urological check-up to early detect UUT deterioration.[29]

Many physicians usually consider that those having an incomplete SCI and being able to walk independently could void normally or nearly normal. They were rarely considered to have unfavorable urodynamics or complications, implying that urological evaluation would not be necessary. But Bellucci CHS et al. reported that ambulatory and nonambulatory SCI patients have similar risk of unfavorable urodynamics.[30] This is consistent with the present study, which did not show a significant association between ambulatory level and hydronephrosis/VUR. The same assessment was recommended in all SCI patients independent of the ability to walk.[30]

Detrusor overactivity, low bladder compliance, small bladder volume and Pdet >40 cmH_2O were the urodynamic parameters detected in 55 (52.88%), 64 (61.54%), 40 (38.46%) and 42 (40.38%) patients respectively. Because of the low numbers of urodynamic data, the correlation analysis failed to demonstrate that these urodynamic parameters were the predictors of UUT deterioration in this study. However, many other studies have shown detrusor overactivity and bladder compliance associated with UUT deterioration,[2,6,7,31,32] whether in suprasacral or sacral lesions.[24] There were 3 possible mechanisms for development of a low compliance bladder proposed by the previous studies. Firstly, changes to passive properties of the bladder wall from fibrosis may be caused by prolonged infection.[33] Secondly, the denervated muscles changed into increased collagen fibers, and increased thickness and stiffness of smooth muscle.[34] Lastly, hypertrophy of the detrusor muscle induced nerve and muscle cells to be hyperactive.[35] In sacral and subsacral lesions, changes to passive properties of the detrusor muscle were believed to be the main etiology of low bladder compliance[26] and lead to hydronephrosis/VUR.

Level of SCI may be another factor associated with renal deterioration[1,2,13,17] but the details are still controversial. Hu HZ et al. stated that injury at T10-L2, where the sympathetic intermediolateral nuclei that mediate sphincter relaxation during voiding are located, is associated with the highest incidence of VUR[2] whereas cervical and lumbosacral lesions showed contradictory results. Gao Y et al.[1] and DeVivo MJ et al.[28] reported that cervical level lesions had the highest risk of complications but Zhang Z et al. demonstrated that the lumbosacral level had the highest risk.[13] Patients with lumbosacral lesions were presumed to have a higher incidence of low bladder compliance according to previous studies,[4,26] although some studies did not find a correlation between level of injury and UUT complications.[3,12,16] In the present study, the association between level of injury and hydronephrosis/VUR was not found in both bivariate and multivariate analyses. The pathology of NLUTD in SCI patients was classified into suprasacral and sacral lesions. Suprasacral lesions were associated with detrusor overactivity so called neurogenic detrusor overactivity (NDO) and detrusor-sphincter dyssynergia (DSD) in 94.9%[36] which led to hydronephrosis/VUR. In sacral lesions, the cause of hydronephrosis/VUR may be due to low bladder compliance following recurrent bladder overdistension and UTI.

As mentioned-above, there were some limitations of this study. Firstly, this study was retrospective and the collected data might be inadequate. Secondly, less than 40% of the patients had urodynamic results, making correlation analysis between urodynamic parameters and upper UUT unreliable. Thirdly, urological check-up protocols might not be the same in every

SCI patient, and it might be influenced by data selection and analyses. However, the criteria for data selection were clarified and have been used in all patients to diminish these problems. Forthly, due to a retrospective study, it was difficult to make a strong conclusion whether some associated factors found in this study were really a cause of UUT deterioration, not an effect of such deterioration. Lastly, radiological abnormalities concerned in this study reflected only anatomical deterioration of the UUT, but not renal function deterioration which has been a major cause of death among patients with chronic SCI.[3,5,13]

In conclusion, triggered reflex voiding with incontinence and indwelling catheterization could be two main predictive factors of hydronephrosis and/or vesico-ureteral reflux in patients with spinal cord injury and neurogenic lower urinary tract dysfunction regardless of level or severity of the lesion. Antimuscarinic medication and high grade bladder deformity could be counted as an associated factor with such deterioration. Early and appropriate bladder management is suggested in spinal cord injured patients to prevent upper urinary tract deterioration.

Acknowledgements

The authors wish to thank Dr. Kanyalak Na Rungsri for help with statistical analysis and Mr. Jason Cullen for help with English correction.

Disclosure

The authors declare no conflicts of interest.

Funding

This research received no grant.

References

1. Gao Y, Damforth T, Ginsberg DA. Urologic management and complications in spinal cord injury patients: a 40-to 50-year follow-up study. Urology. 2017;104:52-8.
2. Hu HZ, Granger N, Jeffery ND. Pathophysiology, clinical importance, and management of neurogenic lower urinary tract dysfunction caused by suprasacral spinal cord injury. J Vet Intern Med. 2016;30:1575-88.
3. Weld KJ, Wall BM, Mangold TA, Steere EL, Dmochowski RR. Influences on renal function in chronic spinal cord injured patients. J Urol. 2000;164:1490-3.
4. Weld KJ, Graney MJ, Dmochowski RR. Differences in bladder compliance with time and associations of bladder management with compliance in spinal cord injured patients. J Urol. 2000;163: 1228-33.
5. Vaidyanathan S, Abraham KA, Singh G, Soni B, Hughes P. Screening for proteinuria in 'at-risk' patients with spinal cord injuries: lessons learnt from failure. Patient Saf Surg. 2014;8:25-32.
6. Musco S, Padilla-Fernández B, Del Popolo G, Bonifazi M, Blok BFM, et al. Value of urodynamic findings in predicting upper urinary tract damage in neuro-urological patients: a systematic review. Neurourol Urodyn. 2018;37:1522-40.
7. Ma Y, Li B, Wang L, Han X. The predictive factors of hydronephrosis in patients with spina bifida: reports from China. Int Urol Nephrol. 2013;45:687-93.
8. Itzkovich M, Gelernter I, Biering-Sorensen F, Weeks C, Laramee MT, et al. The spinal cord independence measure (SCIM) version III: reliability and validity in a multi-center international study. Disabil Rehabil. 2007;29:1926-33.
9. Pannek J, Kennelly M, Kessler TM, Linsenmeyer T, Wyndaele JJ, et al. International spinal cord injury urodynamic basic data set (version 2.0). Spinal Cord Ser Cases. 2018;4:98-102.
10. Suvichai J, Kovindha A. A study of normal uroflow in Thais. Thai J Urol. 2000;21:533-40.
11. Ogawa T. Bladder deformities in patients with neurogenic bladder dysfunction. Urol Int. 1991;47:59-62.
12. Krebs J, Wöllner J, Pannek J. Risk factors for symptomatic urinary tract infections in individuals with chronic neurogenic lower urinary tract dysfunction. Spinal Cord. 2016;54:682-6.
13. Zhang Z, Liao L. Risk factors predicting upper urinary tract deterioration in patients with spinal cord injury: a prospective study. Spinal Cord. 2014;52:468-71.
14. Güzelküçük Ü, Demir Y, Kesikburun S, Aras B, Yaşar E, Tan AK. Ultrasound findings of the urinary tract in patients with spinal cord injury: a study of 1005 cases. Spinal Cord. 2015;53:139-44.
15. Pongboriboon P, Tongprasert S, Kovindha A. Quality of life in persons with spinal cord injury: a comparative study between those with indwelling catheterization and intermittent catheterization. J Thai Rehabil Med. 2011;21:13-20.
16. Rabidi MH, Aston C. Complications and urologic risks of neurogenic bladder in veterans with traumatic spinal cord injury. Spinal Cord. 2015;53:200-3.
17. Cameron AP, Wallner LP, Tate DG, Sarma AV, Rodriguez GM, et al. Bladder management after spinal cord injury in the United States. 1972 to 2005. J Urol. 2010;184:213-7.
18. Chen SF, Jiang YH, Jhang JF, Lee CL, Kuo HC. Bladder management and urological complications in patients with chronic spinal cord injuries in Taiwan. Tzu Chi Med J. 2014;26:25-8.
19. Krebs J, Wöllner J, Pannek J. Bladder management in individuals with chronic neurogenic lower urinary tract dysfunction. Spinal Cord. 2016;54:609-13.
20. Wyndaele JJ, Madersbacher H, Kovindha A. Conservative treatment of the neuropathic bladder in spinal cord injured patients. Spinal Cord. 2001;39:294-300.
21. DeLair SM, Eandi J, White MJ, Nguyen T, Stone AR, Kurzrock EA. Renal cortical deterioration in children with spinal dysraphism: analysis of risk factors. J Spinal Cord Med. 2007;30:S30-4.
22. Yildiz N, Akkoç Y, Erhan B, Gündüz B, Yılmaz B, et al. Neurogenic bladder in patients with traumatic spinal cord injury: treatment and follow-up. Spinal Cord. 2014;52:462-7.
23. Hadiji N, Previnaire JG, Benbouzid R, Robain G, Leblond C, et al. Are oxybutynin and trospium efficacious in the treatment of detrusor overactivity in spinal cord injury patients?. Spinal Cord. 2014; 52:701-5.
24. Kim YH, Bird ET, Priebe M, Boone TB. The role of oxybutynin in spinal cord injured patients with indwelling catheters. J Urol. 1997; 158:2083-6.
25. Consortium of Spinal Cord Medicine. Bladder management for adults with spinal cord injury: a clinical practice guideline for health-care providers. J Spinal Cord Med. 2006;29:527-73.
26. Shin JC, Park C-i, Kim HJ, Lee IY. Significance of low compliance bladder in cauda equina injury. Spinal Cord. 2002:40:650-5.
27. Bywater MB, Tornic J, Mehnert U, Kessler TM. Detrusor acontractility after acute spinal cord injury-myth or reality?. J Urol. 2018; 199:1565-70.
28. DeVivo MJ, Fine PR, Cutter GR, Maetz HM. The risk of renal calculi in spinal cord injury patients. J Urol. 1984;131:857-60.

29. Afsar SI, Sarifakioglu B, Yalbuzdağ ŞA, Saraçgil Coşar SN. An unresolved relationship: the relationship between lesion severity and neurogenic bladder in patients with spinal cord injury. J Spinal Cord Med. 2016;39:93-8.
30. Bellucci CHS, Wollner J, Gregorini F, Birnbock D, Kozomara M, Mehret U, et al. Acute spinal cord injury - do ambulatory patients need urodynamic investigations? J Urol. 2013;189:1369-73.
31. Cho SY, Yi JS, Oh SJ. The clinical significance of poor bladder compliance. Neurourol Urodyn. 2009;28:1010-4.
32. Lee JS, Koo BI, Shin MJ, Chang JH, Kim SY, et al. Differences in urodynamic variables for vesicoureteral reflux depending on the neurogenic bladder type. Ann Rehabil Med. 2014;38:347-52.
33. Macneil HF, Bradkinbg AF, Williams JH. Cause of low compliance in a guinea pig model of instability and low compliance. Neurourol Urodyn. 1992;11:47-52.
34. Ghoniem GM. Effect of bilateral sacral decentralization on detrusor contractility and passive properties in dogs. Neurourol Urodyn. 1984;3:23-30.
35. Levin RM, Longhurst PA, Barasha B, McGuire EJ, Elbadawi A, Wein AJ. Studies on experimental bladder outlet obstruction in the cat: long-term functional effects. J Urol. 1992;148:939-43.
36. Schurch B, Tawadros C, Carda S. Dysfucntion of lower urinary tract in patients with spinal cord injury. Handb Clin Neurol. 2015; 130:247-67.