Motor and bladder dysfunctions in patients with vertebral fractures at the thoracolumbar junction

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Abstract

Objective To present the motor deficits and type of neurogenic bladder dysfunction (NBD) in patients with vertebral fractures at thoracolumbar junction.

Methods Fifty-two patients with single level vertebra fracture over T11–L2 with onset duration of longer than 3 years were enrolled. All participants provided basic demographic data, ambulatory status and received neurologic examination and urodynamic studies. The differences in distribution of NBD types, neurologic injury sites and functional walkers in patients with different levels of vertebral injury were analyzed. Receiver operating characteristic curve analysis was used to define the cutoff value of lower extremities motor score (LEMS) in functional walker and non-walker.

Results Of the 52 patients, the injured levels were 3 (5.8%) in T11, 21 (40.4%) in T12, 22 (42.3%) in L1, and 6 (11.5%) in L2 vertebrae. Eight (15.4%) patients had upper

lumbar cord lesions, 26 (50.0%) had epiconus and lumbar roots lesions, 18 (34.6%) had conus medullaris or/and cauda equina lesions. Mean LEMS was 0 ± 0 , 5.4 ± 7.7 , 11.1 ± 10.2 , and 28.0 ± 11.0 for patients with T11, T12, L1, and L2 fractures, respectively. Patients with L2 fractures had higher LEMS than other levels ($p < 0.001$). The cutoff value of LEMS for functional walking was set at 20, and both the sensitivity and specificity was 100%. Thirty-one (59.6%) patients had spastic NBD, 18 (35.6%) had flaccid NBD, and 3 (5.8%) had mixed type NBD. Positive prediction value of ankle spasticity for bladder and sphincter spasticity was 95.2 and 100%, respectively.

Conclusion Half of the patients had epiconus lesion following thoracolumbar junction fracture, and they had a clinical presentation of flaccid legs and spastic NBD. Patients with L2 fracture had higher LEMS than patients with T11, T12, and L1 fracture. Patients whose LEMS was higher than 20 could all walk functionally. Fracture at the thoracolumbar junction may cause spastic, flaccid, or mixed type NBD, and urodynamic study is an essential tool for the correct diagnosis and management. Ankle spasticity has a high positive predictive value for spastic bladder or sphincter.

Keywords Spinal cord injury · Thoracolumbar junction fracture · Functional walking · Neurogenic bladder dysfunction

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Abbreviations

NBD	Neurogenic bladder dysfunction
LEMS	Lower extremity motor score
T	Thoracic
L	Lumbar
S	Sacral
SCI	Spinal cord injury

MRI Magnetic resonance imaging
 ASIA American Spinal Injury Association

Introduction

The most commonly injured area of the thoracic (T) and lumbar (L) spine is the thoracolumbar junction [1, 2]. The thoracolumbar junction represents a transitional area in relation to anatomy and stress, rendering it particularly vulnerable to fracture and dislocation. The thoracic vertebrae (T1–T10) above the thoracolumbar junction are stabilized by the rib cage and intercostal muscles. The lumbar vertebrae (L3–L5) below the thoracolumbar junction are increasingly more stable because they are larger, stronger and have substantial muscular support. Thus, the majority of thoracic and lumbar spine injuries occur in T11–L2 as a result of the fact that it forms a transitional zone between the very long lever arm of the chest and the very mobile lumbar spine [3, 4].

It is widely accepted that the conus medullaris terminates in the lower third of the L1 vertebra. However, the position of the termination varies in an adult population, and the range span extends from the lower third of the T11 to the upper third of L3 [5, 6]. Hence, the fracture at the thoracolumbar junction may cause damage to cord, conus medullaris or cauda equina (spinal roots) and result in upper motor neuron (UMN), or lower motor neuron (LMN) or mixed lesions over legs and bladder/bowel [7].

The primary concern of spinal cord injured (SCI) patients and their families is whether they will be able to walk following an injury. Among professional staff, walking is also of high priority [8–10]. Previous studies revealed that the total lower extremity motor score (LEMS) was significantly related to both walking level and walking performances. Greater muscle strength produces better endurance, higher walking speed, and lower oxygen consumption per meter [11, 12].

The purpose of this study was to present the motor deficits and type of neurogenic bladder dysfunction (NBD) in patients with fractures at the thoracolumbar junction, and to analyze the requirements of LEMS for functional walking in community.

Subjects and methods

Patients with traumatic SCI around the thoracolumbar junction (T11–L2), who consecutively underwent annual urological survey in our outpatient clinics from January 2007 to December 2009, were recruited for this study. The inclusion criteria were single level vertebra fracture over

T11–L2 with onset duration of longer than 3 years. All of them received internal fixation for spinal injuries, and were confined to wheel chairs when they were transferred to the rehabilitation ward. Patients with multiple level fractures, other neurologic lesions or diseases of musculoskeletal and urological systems were excluded. Patients who ever experienced over distension of the bladder beyond 600 ml were not enrolled, because the results of urodynamic studies could be inaccurate.

The study was approved by the local ethics committee; all subjects gave their informed consent to participate in this study.

All participants provided basic demographic data and received further neurologic examination and urodynamic studies (water cystometry and external sphincter electromyography). The fracture sites were decided according to X-ray, magnetic resonance imaging (MRI) or computed tomography (CT) taken at the acute stage of injury. Neurologic examinations were taken according to the standards from American Spinal Injury Association (ASIA) and included deep tendon reflexes, muscle power and sensation of lower extremities [10]. We also examined the sacral reflex including anal tone and anal reflex. All of the participants reported their ambulatory status at home and in community. A functional walker was defined as someone who could walk independently in the community, with or without the use of devices and braces [10].

According to sacral examinations and urodynamic studies, bladder dysfunction was defined as (1) spastic NBD, with hyperreflexic bladder and sphincter, (2) flaccid NBD, with hyporeflexic or areflexic bladder and sphincter, and (3) mixed type NBD with areflexic bladder and hyperreflexic sphincter.

According to neurological examinations and results of urodynamic studies, the patients were further classified into three groups with different neurologic injury sites. Group A included the patients with upper lumbar cord lesions, who had increased knee and ankle jerks and spastic bladder and urethral sphincter. Group B included patients with epiconus lesions with or without other lumbar root injuries, who had decreased knee jerks and spastic bladder and/or urethral sphincter. Group C included patients with conus medullaris and/or cauda equina lesions, who had decreased knee jerks and flaccid bladder and urethral sphincter.

Statistical analysis

Kruskal–Wallis test was used to compare the LEMS scores in different groups of vertebral fracture levels, and neurologic injury sites. Mann–Whitney *U* test with Bonferroni correction was used for post hoc analysis. The differences in the distribution of NBD types, neurologic injury sites and functional walkers in patients with different levels of

vertebral injury were analyzed by Chi-square test. This test was also used to analyze the association of spasticity in knee/ankle and spasticity in bladder/sphincter. Receiver operating characteristic (ROC) curve analysis was used to define the cutoff value of LEMS in functional walker and non-walker.

Results

We enrolled 52 SCI patients; including 38 men and 14 women, with a mean age of 44.4 ± 12.7 years and mean onset duration of 9.3 ± 6.5 years. The levels of vertebral fracture were T11 in 3 (5.8%), T12 in 21 (40.4%), L1 in 22 (42.3%), and L2 in 6 (11.5%) patients. There were 8 patients that had lumbar cord lesions (group A), 26 that had epiconus lesions with or without other lumbar root injuries (group B) and 18 that had conus medullaris and/or cauda equina lesions (group C). The distributions of bladder dysfunction were spastic in 31 (59.6%), flaccid in 18 (35.6%), and mixed type in 3 patients (5.8%). The levels of vertebral fracture were not associated with different neurologic lesions or NBD types (Table 1).

The role of leg spasticity to predict spasticity of bladder and sphincter is shown in Table 2. Sensitivity, specificity, PPV, and NPV of increased knee jerk on bladder/sphincter spasticity were 27.6/25, 100/100, 100/100, and 48.8%/41.5%, respectively. Sensitivity, specificity, PPV, and NPV of increased ankle jerk on bladder/sphincter spasticity were 64.5/61.8, 95.2/100, 95.2/100, 64.5%/58.1%, respectively.

Among these 52 patients, 42 patients required the use of a wheelchair for ambulation most of the time, and their mean LEMS was 5.9 ± 6.9 . The remaining 10 (19.2%) patients could walk independently in community, and their LEMS was 27.8 ± 6.2 . There was a significant difference ($p < 0.001$) with regards to LEMS between them. The cutoff value of LEMS for functional walking was 20 and both the sensitivity and specificity were 100%. The average LEMS scores and the distribution of walker/non-walker in groups with different fracture levels or different neurologic

injury sites are summarized in Table 3. There was a significant association of fracture levels and ability of walking with phi coefficient of 0.749 ($p < 0.001$, Chi-square test) and the mean LEMS score of patients with L2 fracture was significantly higher than the other three groups. The LEMS of patients with different neurologic lesions was not different from each other.

Discussion

The spinal cord transitions to the cauda equina in the thoracolumbar junction and vertebral fractures in this area would result in variable clinical features including UMN and LMN signs. The conus medullaris, which is the terminal segment of the adult spinal cord, lies at the L1 vertebra for most adults. The segment above the conus medullaris is termed the epiconus and consists of spinal cord segments L4 through S1 [13, 14]. Lesions of the epiconus affect the lumbar cord and the roots supplying muscles of legs and feet, with sparing of reflex function of sacral segments. Conus medullaris lesions, which affect neural segments S2 and below, present with LMN deficits of the bladder and sphincter. Injuries of cauda equina, which includes lumbar and sacral root, will cause LMN deficits of legs, feet, bladder, and sphincter [14].

According to UMN or LMN deficits over legs and bladder/sphincter, the neuropathologic damage from vertebral fractures of thoracolumbar junction can be classified into (a) upper lumbar cord, (b) epiconus and lumbar roots, and (c) conus medullaris and/or cauda equina (Fig. 1). It seemed reasonable that higher vertebral levels should result in higher neurologic lesions, but it is not the case in our study. The possible causes might be that the position of cord termination varies in adult populations (extending from T11 to L3) [6] and fractures were caused by different injury mechanisms. On the other hand, we found that half of our patients in the whole group or in each single fracture group, had epiconus and lumbar roots lesions. Patients who have this kind of injuries showed flaccidity of quadriceps

Table 1 Distribution of groups A, B, C and NBD types in patients with different fracture levels

Vertebral fracture (N)	Neurologic injury sites			NBD types		
	Group A n (%)	Group B n (%)	Group C n (%)	Spastic n (%)	Flaccid n (%)	Mixed n (%)
T11 (3)	2 (66.7)	1 (33.3)	0	3 (100)	0	0
T12 (21)	4 (19.0)	11 (52.4)	6 (29.0)	15 (71.4)	6 (28.6)	0
L1 (22)	2 (9.1)	11 (50.0)	9 (41.0)	10 (45.4)	9 (40.9)	3 (13.6)
L2 (6)	0	3 (50.0)	3 (50.0)	3 (50.0)	3 (50.0)	0
Total (52)	8 (15.4)	26 (50.0)	18 (34.6)	31 (59.6)	18 (34.6)	3 (5.8)

Group A cord lesions, group B epiconus lesions, group C cauda equina lesions, NBD neurogenic bladder dysfunction

Table 2 The predictive value of leg spasticity on spasticity of bladder and sphincter

	Bladder		Sphincter	
	Spastic	Flaccid	Spastic	Flaccid
Knee jerk				
Increased	8	0	8	0
Normal or decreased	21	20	24	17
Ankle jerk				
Increased	20	1	21	0
Normal or decreased	11	20	13	18

Sensitivity, specificity, PPV, and NPV of increased knee jerk on detrusor/sphincter spasticity were 27.6/25, 100/100, 100/100, 48.8%/41.5%
 Sensitivity, specificity, PPV, and NPV of increased ankle jerk on detrusor/sphincter spasticity were 64.5/61.8, 95.2/100, 95.2/100, 64.5%/58.1%

Table 3 Lower extremity motor score (LEMS) and ambulatory status of patients with different levels of vertebral fracture and sites of neurologic injury

	Vertebral fracture levels				Neurologic injury sites		
	T11	T12	L1	L2	Group A	Group B	Group C
LEMS	0 ± 0*	5.4 ± 7.7*	11.1 ± 10.2*	28.0 ± 3.6*	4.4 ± 8.1	10.7 ± 10.8	11.8 ± 12.0
Walker	0 (0%) [†]	1 (5%) [†]	3 (13.6%) [†]	6 (100%) [†]	0	6 (30.0%)	4 (28.5%)
Non-walker	3 [†]	20 [†]	19 [†]	0 [†]	8	20	14

* Higher score in patients with L2 fracture as compared to each of the other three levels ($p < 0.001$, Kruskal–Wallis Test and Mann–Whitney U test with Bonferroni correction)

[†] Significant association of fracture levels and ability of walking with phi coefficient of 0.749 ($p < 0.001$, Chi-square test)

(L3) and ankle dorsiflexors (L4, L5), however, their bladder and/or sphincter (S2–4) are spastic. To the best of our knowledge, there have been few reports in the related literature that mention these clinical events [14].

Spinal root injuries have a better prognosis for recovery than spinal cord injuries. This is most likely because the rootlets are more mobile than cord, with nerve roots being more resilient to injury, and because many of the biochemical processes that occur in the spinal cord may

produce secondary damage [14]. The LEMS of group A patients was numerically lower than group B and C, however, this was not statistically significant. This was most likely caused by the low case numbers of group A (8).

Waters et al. found that patients with LEMS ≤ 20 were limited walkers with slow walking speed and great energy expenditure. Patients with LEMS ≥ 30 were functional walkers in community; their values regarding gait performance and energy expenditure were reasonably close to comparable values for able-bodied control subjects [15]. In our study, patients who had lower levels of vertebral fracture had higher mean LEMS as well as a higher possibility of functional walking. The cutoff value of LEMS for functional walking was 20 in this study with good sensitivity and specificity and this was compatible with the findings of Waters et al. [15]. In eight of ten functional walkers in this study, their LEMS were between 20 and 30. Although they walked with the help of some walking aids or ankle foot orthoses and had slower walking speed and higher energy expenditure than able-bodied subjects, it was practical and more convenient for them to walk in community than to use a wheelchair.

Spinal cord injuries are well known to cause NBD. The parasympathetic efferent supply of bladder originates from pelvic nerve nucleus located in the intermediolateral

Fig. 1 Areas of neuropathic damage produced by thoracolumbar junction fractures. **a** Destruction of upper lumbar cord. **b** Destruction of conus medullaris and surrounding lumbar roots. **c** Destruction of conus medullaris and cauda equina

gray matter of the S2–4 cord. Somatic efferent supply of external sphincter originates from pudendal nerve nucleus located in the anterior horn of S2–4 gray matter [16]. Suprasacral lesions frequently lead to spastic NBD with hyperreflexic bladder and sphincter. Sacral lesions cause flaccid NBD with areflexic bladder and atonic sphincter [16]. In this study, three patients of L1 fractures with epiconus lesions showed areflexic bladder and hyperreflexic sphincter. Bors [17] classified it as mixed type NBD caused by lower vesicomotor (pelvic nerve) neuron lesion and upper somatomotor (pudendal nerve) neuron lesion. This may happen, when the epiconus lesions extend to the pelvic nucleus with sparing of the pudendal nucleus because the pelvic nucleus is slightly higher and posterior to the pudendal nucleus [16, 17].

Previous studies showed that voiding dysfunction in patients with spinal cord lesions of the thoracolumbar junction could be hyperreflexic or areflexic. Because the sacral cord is located at thoracolumbar junction, it may be difficult to predict urodynamic dysfunctions merely on the basis of the vertebral body involved [18, 19]. In this study, distribution of NBD types in patients with different fracture levels revealed no statistical difference. We also concluded that urodynamic study is an essential tool in the correct diagnostic and therapeutic approach to the voiding dysfunction in these types of patients [18, 19].

The somatomotor nucleus of quadriceps and calf muscles is mainly located in L3 and S1 cord, respectively. Patients with increased knee or ankle jerks must have a suprasacral lesion. Ankle spasticity has high PPV and specificity for bladder and sphincter spasticity with acceptable sensitivity and NPV. Increased knee jerk indicated higher spinal cord lesions and even better PPV and specificity for predicting bladder and sphincter spasticity (all 100%), but the sensitivity and NPV were much lower than ankle spasticity. Thus, we concluded that after the acute stage of injury, if the ankle spasticity appeared, spastic NBD can be predicted with high accuracy.

This study has some limitations that are worth noting. The number of patients with T11 and L2 fractures is far less than those with T12 and L1 fractures, and the results of statistic analysis might therefore be influenced. Not all patients had MRI, the location of termination of conus medullaris and neural damage could not be defined to correlate their neurological deficits.

Conclusion

Thoracolumbar junction fracture may cause cord, epiconus and lumbar roots or conus medullaris and/or cauda equina lesions. In this study, half of the patients had epiconus

and/or cauda equina lesions, and they had a clinical presentation of flaccid legs but spastic NBD.

The patients with L2 fracture had higher LEMS and possibility of functional walking than patients with T11, T12, and L1 fractures. Patients whose LEMS were higher than 20, all could walk functionally.

Fracture at the thoracolumbar junction may cause spastic, flaccid, or mixed type NBD, and urodynamic study is an essential tool for the correct diagnosis and therapeutic approach to voiding dysfunction. Ankle spasticity has a high PPV for spastic bladder or sphincter.

Conflict of interest None.

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