Original Article Correlations between posterior longitudinal ligament status and size of bone fragment in thoracolumbar burst fractures

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Abstract: This study aim to determine the correlation between the size of bone fragment and injury of posterior longitudinal ligament (PLL). In this study retrospectively analyze medical chart of patients with thoracolumbar burst fractures from June 2010 to December 2012. Patients were divided into two groups (Intact group and Disrupted group) according to the result of MRI assessing status of PLL. All the fractures were classified according to the Arbeit Fuer Osteoosynthese (AO) classification system. Neurological status was classified according to American Spinal Injury Association (ASIA). Mimics measured the height and width of bone fragment (HBF and WBF), transverse canal diameter (TCD) and calculate the height of posterior wall of the injury vertebrae, ratio of height of bone fragment occupying height of posterior wall of vertebrae body (RHBF) and ratio of width of bone fragment occupying transverse canal diameter (RWBF). The results indicated that 52 patients were included in the study. There are 31 patients with intact PLL and 21 patients with disrupted PLL. There was significant difference on the HBF (t = -3.646, P = 0.001), WBF (t = -3.615, P = 0.001), RHBF (t = -4.124, P = 0.000) and RWBF (t = -3.305, P = 0.002) between the intact group and injury group. There was a significant correlation between injury of PLL and ASIA grade (OR = 7.851, P = 0.005), and AO classification (OR = 6.401, P = 0.011), and RHBF (OR = 6.455, P = 0.011), and HBF (OR = 5.208, P = 0.022). In conclusion, the results of this study indicate that AO classification, ASIA grade, HBF and RHBF could act as the predictors of injury of PLL.

Keywords: Thoracolumbar burst fractures, bone fragments, posterior longitudinal ligament

Introduction

Ninety percent of all spinal fractures occur in the thoracolumbar region, and burst fractures contribute to approximately 10-20% of such injuries [1-4]. It is one of the most common causes for spinal cord injury, and the frequency of neurological deficits in all thoracolumbar burst fractures can reach up to 50-60% [4-6]. Spinal cord injury includes both primary and secondary injury mechanisms [2, 7]. Secondary injury because of compression of bone fragments lead to a series of pathophysiologic changes such as 1) vascular changes including reduction in blood flow, loss of auto-regulation, neurogenic shock, hemorrhage, loss of microcirculation, vasospasm and thrombosis [7, 8]; 2) electrolyte shifts including increased intracellular calcium, increased extracellular potassium, and increased sodium permeability [9, 10]; 3) neurotransmitter accumulation such as

serotonin or catecholamines [11] and extracellular glutamate [12], the latter producing excitotoxicity [13]; 4) arachidonic acid release, free radical production especially oxygen-free radicals [14], eicosanoid production, especially prostaglandins, and lipid per-oxidation [15, 16]; 5) endogenous opioids [17, 18]; 6) edema formation [19]; 7) inflammation; and 8) loss of energy metabolism, especially decreased adenosine triphosphate production [20]. So reposition of bone fragments is benefit to recovery of neurological function.

In the spinal surgery there are two ways including anterior and posterior approaches. Posterior surgery can be recommended in emergency neurodecompression and fixation of unstable thoracolumbar fractures because of the shorter operation time and smaller blood loss versus anterior surgery [21-25]. In the posterior surgery lordosation and distraction with the inter-



Figure 1. WBF, TCD and HBF measurement. A. The width of bone fragment was 17.04 mm on the axial plane of CT image. B. The TCD was 26.77 mm on the axial plane of CT image. C. The height of bone fragment, posterior wall of vertebras above and below injury vertebra were 12.81 mm, 30.76 mm and 30.54 mm on the sagittal plane of CT image.

nal fixator lead to the restoration of height, kyphosis correction and in many cases to canal widening by the phenomenon of ligamentotaxis. Ligamentotaxis is primarily induced by increased tension on the PLL during lordosation and distraction. The rapid volume increase of the fractured vertebra during this procedure may contribute to the effect of ligamentotaxis by creating an area of under pressure, inducing suction on the dislocated bone fragments. But if the PLL was injury it is difficult to reposition the bone fragments by the posterior surgery. This study discriminated if multiple radio-graphical parameters correspond to injury of PLL.

Materials and methods

We retrospectively reviewed consecutive patients with a thoracolumbar $(T_{11}-L_2)$ burst fracture from a single center. Inclusion criteria include consecutive patients with single vertebrae thoracolumbar burst fractures because of trauma. Exclusion criteria include pathological fractures, multiple vertebras thoracolumbar burst fractures were examined by multi-planar computed tomography (CT) scan and an MRI of the injured segment before surgery. All the patients underwent spinal surgery with the same posterior instrumentation.

Axial-plane central canal measurements

The width of bone fragment (WBF, **Figure 1A**) was defined as width of bone fragment at the vertebral pedicle level of CT image. Transverse

canal diameter (TCD, **Figure 1B**) was defined as distance between the medial borders of the pedicles at the mid-pedicle level. The ratio of width of bone fragment occupying transverse canal diameter (RWBF) was calculated according to formula V2/ (V1 + V3)/2 [26]. V1 indicates the TCD above the injury vertebra. V2 indicates width of bone fragment. V3 indicates TCD below the injured vertebra.

Sagittal-plane central canal measurements

The height of bone fragment (HBF, **Figure 1C**) was defined as height of bone fragment at the mid-sagittal plane of the CT image. Height of posterior wall of injury vertebral body was calculated according to formula (V1 + V3)/2 [26, 27]. The ratio of height of bone fragment occupying posterior wall of injury vertebral body (RHBF) was calculated according to formula V2 / (V1 + V3)/2 [26, 27]. V1 indicates the height of vertebra at the level above the injury vertebra. V2 indicates height of bone fragment. V3 indicates height of vertebra at the level below the injured vertebra. V1, V2 and V3 were measured directly.

Fracture pattern and neurological injury

All the fractures were classified according to the AO classification system. A1 is compression fracture. A3 is burst fracture. A3.1 is wedge compression fracture. A3.2 is sagittal or coronal split fracture in the vertebral body. A3.3 is comminuted and displacement fracture.

Characteristics	Number
Gender	
Male	34
Female	18
Mean age (yr)	37.1
thoracolumbar burst fracture position	
T11	4
T12	11
L1	22
L2	15
ASIA grade	
ASIA A	10
ASIA B	9
ASIA C	8
ASIA D	7
ASIA E	18
Fall	32
AO Classification	
A3.1	18
A3.2	16
A3.3	18
MVA	20
PLL intact	31
PLL disrupted	21

Table	1.	Demographics	of the	patients
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ASIA: American Spinal Injury Association; PLL: posterior longitudinal ligament.

The neurological status was classified according to American Spinal Injury Association's modified Frankel's grading of traumatic paraplegia [28]: A, No sensory or motor function is preserved in the sacral segments S4-S5; B, Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-S5; C. Motor function is preserved below the neurological level, and more than half of key muscles below the neurological level have a muscle grade less than 3; D, Motor function is preserved below the neurological level, and at least half of key muscles below the neurological level have a muscle grade greater than or equal to 3; and E. Sensory and motor function is normal. As the fractures pattern is sequentially classified into three subgroups and neurological injury is classified into five types, the values are added to provide a comprehensive severity score. A3.1 is assigned 1 point, A3.2 is assigned 2 points and A3.3 is assigned 3 points. ASIA A is assigned 0 point, ASIA B is assigned 1 point, ASIA C is assigned 2

points, ASIA D is assigned 3 points and ASIA E is assigned 4 points.

Assessment of injury of PLL

MRI assessed injury of PLL according to Grenier's study [29]. Intact PLL is assigned 1 point. Disrupted PLL is assigned 2 point.

Statistical analysis

We used SPSS 12.0 for windows (SPSS Inc, Chicago, Illinois) for statistical analysis. All data were presented as mean \pm standard deviation (SD) or frequency. Logistic Regression correlates different parameters, AO classification, ASIA grade to PLL status. All tests were set as two sides and a *P* value of < 0.05 was considered statistical significant.

Results

Included patients

A total of 52 patients formed the study population. All of these patients were divided into two group randomly, including intact group (31 patients) and disrupted group (21 patients). Also, there were no significant differences between the two groups for the clinical characteristics. The demographics of the patients are presented in **Table 1**. Mean age was 37.1 years. There were 34 men and 18 women.

Measurements of parameters

Summary of CT measurements (mean, standard deviation) is displayed in Table 2 for each measurement. The minimal HBF was 5 mm in the intact group, while 7 mm in the disrupted group. The minimal WBF was 10 mm in the intact group, while 12 mm in the disrupted n group. The minimal RHBF was 26.7% in the intact group, while 33.5% in the disrupted group. The minimal RWBF was 43.5% in the intact group, while 58.5% in the disrupted group. There was significant difference on the HBF (t = -3.646, P = 0.001 < 0.05), WBF (t = -3.615, P = 0.001 < 0.05), RHBF (t = -4.124, P = 0.000 < 0.05) and RWBF (t = -3.305, P = 0.002 < 0.05) between the intact group and injury group.

PLL status and AO classification, ASIA grade

The numbers of PLL status in different AO classification and ASIA grade are displayed in **Table**

	Intact group (n = 31)		Disrupted group (n = 21)		
Characteristics	Mean	SD	Mean	SD	- P
Height of bone fragments (mm)	10.17	3.11	13.15	2.53	0.001
Width of bone fragments (mm)	12.97	3.58	16.88	4.17	0.001
RHBF (%)	0.358	0.127	0.498	0.109	0
RWBF (%)	0.549	0.177	0.734	0.226	0.002
AO Classification					
A3.1	15		3		0.001
A3.2	13		3		0.001
A3.3	3		15		0.013
ASIA Grade					
ASIA A	3		7		0.042
ASIA B	3		6		0.045
ASIA C	4		4		-
ASIA D	6		1		0.001
ASIA E	15		3		0.001

Table 2. Summary of Mimics10.0 Measurements and different AO classification and ASIA Grades

RHBF: ratio of height of bone fragment; RWBF: ratio of width of bone fragment; SD: standard deviation. ASIA: American Spinal Injury Association; AO: Arbeit Fuer Osteoosynthese.

Table 3. Correlation between Reposition of Bone Fragments andParameters

Characteristics	OR (95% CI)	β-Coefficient	Р
Height of bone fragments (mm)	5.208 (0.87-7.15)	0.54	0.022
Width of bone fragments (mm)	2.847 (1.05-5.68)	0.37	0.092
RHBF (%)	6.455 (1.31-9.04)	0.65	0.011
RWBF (%)	0.992 (0.64-5.63)	0.04	0.319
AO Classification	6.401 (2.05-8.04)	0.54	0.011
ASIA Grade	7.851 (2.51-10.14)	0.73	0.005

RHBF: ratio of height of bone fragment; RWBF: ratio of width of bone fragment; ASIA: American Spinal Injury Association; AO: Arbeit Fuer Osteoosynthese; OR: odds ratio.

2. There were 31 patients with intact PLL and 21 patients with disrupted PLL.

Correlations between different parameters measurements, AO classification, ASIA grade and reposition of bone fragments

Table 3 displays coefficients between different parameters measurements with Mimics assistance, AO classification, ASIA grade and PLL status. There was a significant correlation between injury of PLL and ASIA grade (OR = 7.851, P = 0.005), and AO classification (OR = 6.401, P = 0.011), and RHBF (OR = 6.455, P = 0.011), and HBF (OR = 5.208, P = 0.022). There was no significant correlation between WBF, RWBF and PLL status (**Table 3**).

Discussion

These results demonstrate that HBF, RHBF, AO classification and ASIA grade were found to be related to injury of PLL. Reposition of bone fragments has been established to be associated with ligamentotaxis by intact PLL and ligamentotaxis can reduce only those retropulsed fragments that are still attached to a ligamentous structure [30]. According to

Muller's study [31], the large trapezoid-shaped fragments were considered to be difficult to reposition. But it is not clear that what the size of bone fragment was the large trapezoidshaped fragments and the correlation was between injury of PLL and size of bone fragment. MRI is currently considered the "gold standard" for determination of a PLL injury in thoracolumbar burst fracture and has been found to be reasonably sensitive to PLL injury, although it may not discriminate all the PLL status [29]. So bony threshold parameters should be correspond to PLL injury for indirect assessment of injury of PLL.

The present study establishes that threshold bony parameters do not conform to reposition

of bone fragments, with the exception of > 67% compromise of the spinal canal [32, 33]. There was no significant association between threshold bony parameters and injury to the PLL. The bony parameters may be predictors of injury of PLL.

These findings suggest that AO classification and ASIA could be predictors of assessing reposition of bone fragments. Possible explanations for these findings include a relationship between bony size and injury of PLL. Patients with large bony fragment might be expected to have higher degree AO classification, ASIA grade because the vertebral body had a greater crush, the PLL may be disrupted. Conversely, patients with subtle bony fragments have lighter degree AO classification, ASIA grade, the PLL may be intact.

Strengths of this study include it analyze multiple parameters that are correlation to the injury of PLL and point out the most important referential parameters. At the same time this study reminds surgeon attention on necessary parameters about assessing status of PLL before operation. Limitations of this study include that sample is small and it did not acquire quantized numeric.

In conclusion, these results demonstrate that AO classification, ASIA grade, HBF and RHBF are correlations to injury of PLL in the thoracolumbar burst fracture. Especially PLL status is not clear on MRI scan, it is necessary that paying attention to AO classification, ASIA grade, HBF and RHBF. Certainly surgeons should consider direct assessment of PLL injury if there is clinical concern instead of indirect assessment from bony measurements.

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Disclosure of conflict of interest

None.

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References

- [1] Wilcox RK, Boerger TO, Allen DJ, Barton DC, Limb D, Dickson RA, Hall RM. A dynamic study of thoracolumbar burst fractures. J Bone Joint Surg Am 2003; 85: 2184-2189.
- [2] Niu JJ, Shen MJ, Meng B, Yang Y, Yang HL. Percutaneous kuphoplasty for the treatment of osteoporotic thoracolumbar fractures with neurological deficit: radicular pain can mimic disc herniation. Int J Clin Exp Med 2014; 7: 2360-2364.
- [3] Dai LY, Jiang SD, Wang XY, Jiang LS. A review of the management of thoracolumbar burst fractures. Surg Neurol 2007; 67: 221-231.
- [4] Qiu TX, Tan KW, Lee VS, Teo EC. Investigation of thoracolumbar T12-L1 burst fracture mechanism using finite element method. Med Eng Phys 2006; 28: 656-664.
- [5] Cho DY, Lee WY, Sheu PC. Treatment of thoracolumbar burst fractures with polymethyl methacrylate vertebroplasty and short-segment pedicle screw fixation. Neurosurgery 2003; 53: 1354-1601.
- [6] Willen J, Lindahl S, Nordwall A. Unstable thoracolumbar fractures. A comparative clinical study of conservative treatment and Harrington instrumentation. Spine 1985; 10: 111-122.
- [7] Tator CH. Review of experimental spinal cord injury with emphasis on the local and systemic circulatory effects. Neurochirurgie 1991; 37: 291-302.
- [8] Stripling TE. The cost of economic consequences of traumatic spinal cord injury. Paraplegia News 1990; 1: 50-54.
- [9] Agrawal SK, Fehlings MG. Mechanisms of secondary injury to spinal cord axons in vitro: role of Na+, Na(+)-K(+)-ATPase, the Na(+)-H+ exchanger, and the Na(+)-Ca++ exchanger. J Neurosci 1996; 16: 545-552.
- [10] Young W, Koreh I. Potassium and calcium changes in injured spinal cords. Brain Res 1986; 365: 42-53.
- [11] Osterholm JL, Mathews GJ. Altered norepinephrine metabolism following experimental spinal cord injury. Part 1: Relationship to hemorrhagic necrosis and post-wounding neurological deficits. J Neurosurg 1972; 36: 386-394.
- [12] Agrawal SK, Fehlings MG. Role of NMDA and non-NMDA ionotopic glutamate receptors in traumatic spinal cord axonal injury. J Neurosci 1997; 17: 1055-1063.
- [13] Faden AI, Simon RP. A potential role for excitotoxins in the pathophysiology of spinal cord injury. Ann Neurol 1988; 23: 623-626.
- [14] Demopoulos HB, Flamm ES, Pietronigro DD, Seligman ML. The free radical pathology and the microcirculation in the major central ner-

vous system disorders. Acta Physiol Scand (Suppl) 1980; 492: 91-119.

- [15] Hall ED, Yonkers PA, Horan KL, Braughler JM. Correlation between attenuation of posttraumatic spinal cord ischemia and preservation of tissue vitamin E by the 21-aminosteroid U7-4006F: evidence for an in vivo antioxidant mechanism. J Neurotrauma 1989; 6: 169-176.
- [16] Hung TK, Albin MS, Brown TD, Bunegin L, Albin R, Jannetta PJ. Biomechanical responses to open experimental spinal cord injury. Surg Neurol 1975; 4: 271-276.
- [17] Faden AI, Jacobs TP, Holaday JW. Comparison of early and late naloxone treatment in experimental spinal injury. Neurology 1982; 32: 677-681.
- [18] Faden AI, Jacobs TP, Smith MT. Evaluation of the calcium channel antagonist nimodipine in experimental spinal cord ischemia. J Neurosurg 1984; 60: 796-799.
- [19] Wagner FC, Stewart WB. Effect of trauma dose on spinal cord edema. J Neurosurg 1981; 54: 802-806.
- [20] Anderson DK, Means ED, Waters TR. Spinal cord energy metabolism in normal and postlaminectomy cats. J Neurosurg 1980; 52: 387-391.
- [21] Haas N, Blauth M, Tscherne H. Anterior plating in thoracolumbar spine injuries. Indication, technique, and results. Spine 1991; 16: 100-111.
- [22] Oprel PP, Tuinebreijer WE, Patka P, den Hartog D. Combined anterior-posterior surgery versus posterior surgery for thoracolumbar burst fractures: a systematic review of the literature. Open Orthop J 2010; 4: 93-100.
- [23] Esses SI, Botsford DJ, Kostuik JP. Evaluation of surgical treatment for burst fractures. Spine 1990; 15: 667-673.
- [24] Chen F, Liu T, Li J, Xing Z, Huang S, Wen G. MRI characteristicvs and follow-up findings in patients with neurological complications of enterovirus 71-related hand, foot, and mouth disease. Int J Clin Exp Med 2014; 7: 2696-2704.

- [25] Stancic MF, Gregorovic E, Nozica E, Penezic L. Anterior decompression and fixation versus posterior reposition and semi-rigid fixation in the treatment of unstable burst thoracolumbar fracture: prospective clinical trial. Croat Med J 2001; 42: 49-53.
- [26] Hashimoto T, Kaneda K, Abumi K. Relationship between traumatic spinal canal stenosis and neurologic deficits in thoracolumbar burst fractures. Spine 1988; 13: 1268-1272.
- [27] Willen J, Anderson J, Toomoka K, Singer K. The natural history of burst fractures at the thoracolumbar junction. J Spinal Disord 1990; 3: 39-46.
- [28] Maynard FM, Bracken MB, Creasey G. International standards for neurological and functional classification of spinal cord injury. Spinal Cord 1997; 35: 266-274.
- [29] Grenier N, Greselle JF, Vital JM, Kien P, Baulny D, Broussin J, Senegas J, Calile JM. Normal and Disrupted Lumbar Longitudinal Ligaments: Correlative MR and Anatomic Study. Radiology 1989; 171: 197-205.
- [30] Vidal J, Buscayret C, Connes H. Treatment of articular fractures by 'Ligamentotaxis' with external fixation. In: Brooker AS, Edwards CC, editors. External fixation. Baltimore: Williams and Wilkins; 1979.
- [31] Mueller LA, Mueller LP, Schmidt R, Forst R, Rudig L. The phenomenon and efficiency of ligamentotaxis after dorsal stabilization of thoracolumbar burst fractures. Arch Orthop Trauma Surg 2006; 126: 364-368.
- [32] Whang PG, Vaccaro AR. Thoracolumbar Fracture: Posterior Instrumentation Using Distraction and Ligamentotaxis Reduction. J Am Acad Orthop Surg 2007; 15: 695-701.
- [33] Al-Daghri NM, Yakout S, Al-Shehri E, Al-Fawaz H, Aljohani N, Al-Saleh Y. Inflammatory and bone turnover markers in relation to PTH and vitamin D status among Saudi postmenopausal women with and without osteoporosis. Int J Clin Exp Med 2014; 7: 2812-2819.