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Efficiency of transpedicular reduction of intracanal bone fragments in comminuted fractures of L1 vertebra

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Abstract

The objective was to retrospectively review the relationship between the parameters and the position of intracanal bone fragments in comminuted fractures of L1 vertebra and the effect on neurological status and restoration of the anterior wall of the spinal canal using a transpedicular reduction device. **Material and methods** Spiral computed tomography (CT) scans and case histories of 45 patients with spinal cord injury at the level of L1 vertebra were reviewed. The study included patients with comminuted fractures including intracanal bone fragments from the posterior portion part of L1 vertebra. Bone fragments were relocated from the spinal canal to varying degrees in patients who underwent procedure using the posterior access and transpedicular reduction system. Two groups of patients were identified with regard to displacement: the bone could be shifted by 50 % and over in the first group (n = 25) and less than 50 % in the second group (n = 20). **Results** Preoperative time was shorter in the first group: 6.7 ± 3 versus 15.5 ± 5.6 days in the second group. The bone width was statistically smaller in the first group with 18.2 ± 2.3 mm versus 22.3 ± 2.6 mm in the second group. Deficient lumen and deficient area of the spinal canal were significantly greater in the first group. **Discussion** Prediction of the effective ligamentotaxis is essential for optimal surgical strategy. Bone parameters and position, performance of distraction and correction of angulation of injured vertebral segment play a role in the effectiveness of indirect reduction of bone fragments protruding into the spinal canal. **Conclusion** Deficient lumen and deficient area of the spinal canal, the length and width of the intracanal bone fragment were not associated with neurological disorders ASIA C, D and E types in case of comminuted fractures of L1 vertebra. The effectiveness of closed decompression of the spinal cord in spinal cord injury at L1 level was dependent on the width of intracanal bone fragments and the preoperative time.

Keywords: spinal cord injury, intracanal bone fragment, transpedicular reduction

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INTRODUCTION

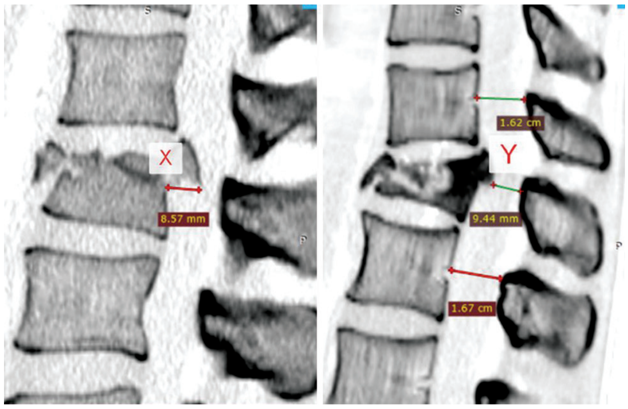
Injury to the spine and spinal cord has devastating consequences for the physical, social and vocational well-being of patients, family and society [1, 2]. There is an increased number of injuries associated with road accidents (22-70 %), falls from a height (18-61 %) [3, 4]. Injuries complicated by neurological disorders account for 39.2 % in the lower thoracic and 48.5 % in the lumbar spine [5]. The spinal cord suffers from both primary and secondary injury after an accident. If the primary injury to the spinal cord has already occurred, therapeutic strategies are aimed at reducing the severity of the secondary injury. Secondary injury mechanisms can be caused by impaired blood supply [6, 7], electrolyte imbalance [8, 9] and cell apoptosis [10]. Spinal cord decompression with reconstruction of the anterior and intermediate sections of the spine through the posterior median approach with transpedicular fixation is a safe and effective method in the treatment of fractures of the thoracolumbar spine [11-13]. Restoration of the shape of the spinal canal can be achieved by direct removal of bone fragments [14, 15] and by reduction due to the “effect” of ligamentotaxis [16, 17]. There is an opinion that displacement of a fragment into the spinal canal is not a reason for surgical treatment with a combination of factors including deformity and stability being relevant for the choice of treatment strategy to ensure spontaneous remodeling of the spinal canal during vertebral consolidation [18]. Distraction is the main factor contributing to the reduction of the fragment from the spinal canal leading to tension of the posterior longitudinal ligament and the posterior annulus fibrosus [19]. However, not all bone fragments can be removed from the spinal canal using ligamentotaxis [20, 21]. There are few studies evaluating the effectiveness of spinal reposition depending on the size and position of intracanal bone fragments.

The objective of the study was a retrospective analysis of the relationship between the parameters and position of intracanal bone fragments in comminuted fractures of the L1 vertebra and the effect on the neurological status and restoration of the anterior wall of the spinal canal using a transpedicular repositioning device.

MATERIAL AND METHODS

Pre- and postoperative SCT scans of 45 patients (25 males, 20 females) with spinal cord injury at the level of the L1 vertebra were evaluated. The study included patients with multicomminuted fractures, including intracanal bone fragments from the posterior upper body of the L1 vertebra. Exclusion criteria were multiple vertebral fractures, non-traumatic fractures. The study was performed in compliance with the Declaration of Helsinki of the World Medical Association "Ethical principles for conducting scientific medical research involving human subjects" as amended in 2000. The average age of the patents was 38.2 ± 3.9 years. Patients were diagnosed with AO type A3 injuries (n = 3), type A4 (n = 39), type B2 (n = 3). The neurological status, the severity of spinal cord injury was identified using the ASIA scale as type C (n = 20), type D (n = 13) and type E (n = 12). Posterior approach was used for the patients and a 5- or 6-screw transpedicular construct mounted. Transpedicular screws were implanted in the bodies of Th12, L1, L2 vertebrae. The angular deformity of the involved spinal segment was corrected using the Sintez repositioning device for transosseous transpedicular osteosynthesis, the height of the vertebra restored and the spinal cord decompressed in a closed manner due to ligamentotaxis. Displacement of bone fragments from the spinal canal to varying degrees was achieved in the ventral direction in all cases. Two groups of patients were identified according to the displaced intracanal bone fragment (X) after the operation. Bone fragments were displaced from the spinal canal by 50 % or more of the initial displacement in group 1 (n = 25) and by less than 50 % in the second group (n = 20). Measurement X is shown in Figure 1a. The characteristics of the groups by types of spinal injury, gender, age and neurological status are presented in Table 1.

Ha Multiplanar reconstruction (DICOM format) was performed using preoperative and postoperative SCT scans and the RadiAnt program. The lumen deficit and the area deficit of the spinal canal at the level of damage were calculated; the length and width of the bone fragments, the posterior height of the damaged and adjacent vertebral bodies (PVH), the inversion angle of the bone fragment (β), and the angle between the lower cortical plate of the Th7 vertebral body and the cortical part of the fragment (λ) were measured; the transverse diameter of the spinal canal (L), the width of the bone fragment relative to the transverse diameter of the spinal canal were measured and calculated. To reduce measurement errors, all measurements were repeated twice and averaged.



a b

Fig. 1 Displacement of the bone fragment towards the spinal canal (a) and lumen of the spinal canal measured (b)

Measurement of the lumen of the spinal canal is shown in Figure 1b. The diameter of the spinal canal (Y) calculated at the level of injury was determined by averaging the diameters of the spinal canals of neighboring vertebrae above and below the injury level. Lumen deficit of the spinal canal was calculated using the formula (Y-Y1) / Y × 100 %, where Y1 was the size of the spinal canal at the level of the L1 vertebra.

The measurement of the transverse diameter of the spinal canal (L) is shown in Figure 2a. The calculation of the deficit of the area of the spinal canal was performed by analogy with the deficit of the lumen of the spinal canal. The area of the spinal canal was measured (Fig. 2b) using axial SCT scans at the level of injury and adjacent levels. The area deficit was calculated using the formula (S-S1)/S × 100 %, where S1 was the area of the spinal canal at the level of L1 vertebra. The posterior vertebral height (PVH) at the level of injury was calculated as a percentage of the normal height. The average height of the posterior wall above and below the vertebra was recorded as the normal height of the posterior wall of the damaged vertebra (Fig. 2c). The kyphotic deformity angle α was measured between the lower endplate of the Th12 body and the upper endplate of the L2 vertebral body. Measurement of the length and width of the bone fragment is shown in Figure 3.

Table 1

Characteristics of comparison groups

Group	Type of injury (AO classification)			Gender		Age, years	Neurological status ASIA		
	A3	A4	B2	M	F		C	D	E
1	1	22	2	15	10	39.6 ± 5.4	13	9	3
2	2	17	1	10	10	36.5 ± 5.8	7	4	9

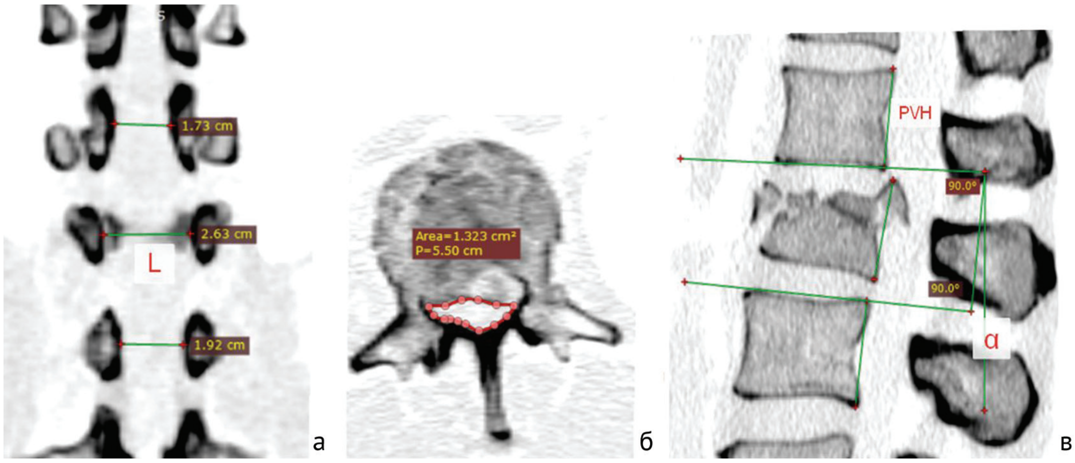


Fig. 2 Transverse diameter of the spinal canal (L) (a); areas of the spinal canal (S) (b); posterior vertebral height (PVH) and segmental deformity angle α (c) measured

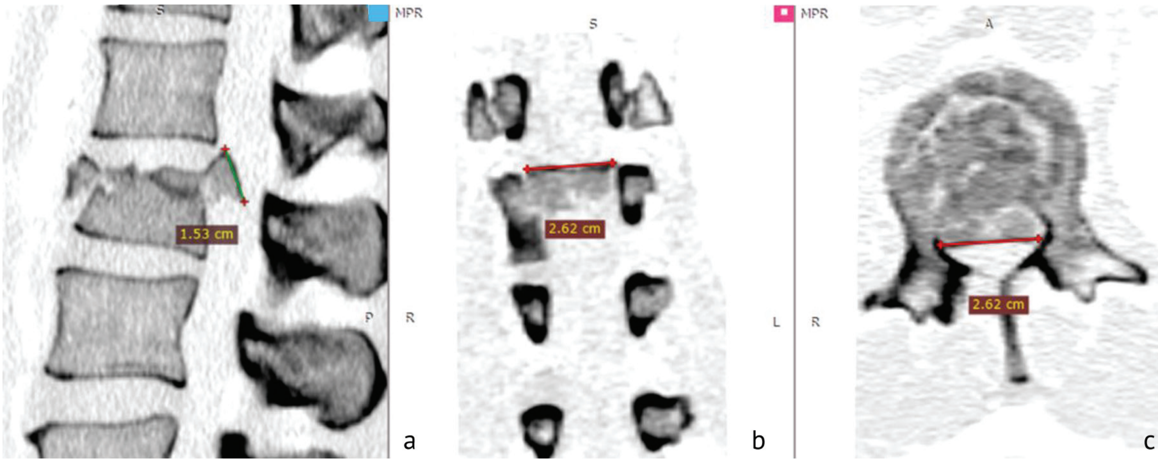


Fig. 3 Bone fragment length; the width of the bone fragment(a); measured in the frontal plane (b); in the horizontal plane (c)

The angle of rotation of the bone fragment (β) was formed by the intersection of the line along the posterior wall of the damaged vertebra and the line on the bone fragment as a continuation of the posterior wall of the vertebra (Fig. 4a). An angle (λ) formed by the lower cortical plate of the body of the overlying vertebra and a part of the upper cortical plate of the injured vertebra located on the bone fragment was included in the study (Fig. 4b). The angle was not shown to change during vertebral reduction.

Statistical analysis was performed using the SPSS Statistic ver. 23. Descriptive statistics included calculation of mean values with 95 % confidence intervals. A cross-sectional statistical analysis of the parameters measured in two groups was produced using a t-test for independent samples and one-way analysis of variance ANOVA. The difference was considered statistically significant at $p < 0.05$.

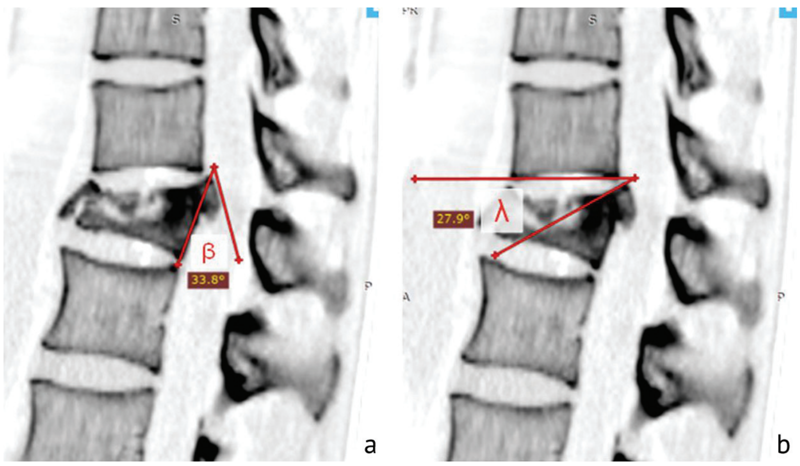


Fig. 4 Angle of turn of the bone fragment β (a); angle λ (b) measured

RESULTS

The neurological status of patients was not dependent on the deficiency of the lumen of the spinal canal, which is shown in the box diagram (Fig. 5a). Analysis of variance revealed no statistically significant differences between the degree of neurological disorders and the deficiency of the spinal lumen (p = 0.27). There was a significantly greater deficit in the lumen of the spinal canal (p = 0.018) in the first group, but this did not affect the effectiveness of the reformation.

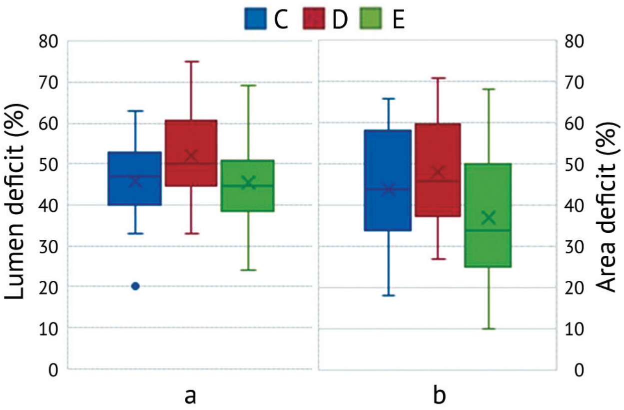


Fig. 5 Lumen deficit (a) and area deficit (b) of the spinal canal in % and neurological status according to ASIA

The box diagram (Fig. 5b) shows the dependence of neurological disorders on the deficit of the area of the spinal canal. The greater deficit of the spinal canal area is seen with grade C neurological disorders with no statistical confirmation received (p = 0.17). The deficit of the spinal canal area prevailed in group 1 measuring 47.2 ± 5.8 % versus 38.4 ± 6.7 % in group 2 (p = 0.05). Table 2 presents the statistical analysis of the parameters compared. Patients with two bone fragments were seen in two groups. There were more of the patients in the first group: 1.5 ± 0.2 versus 1.2 ± 0.2. The neurological status was not affected by the length (p = 0.5), width (p = 0.6) and the number of bone fragments (p = 0.48), which is shown in Figure 6.

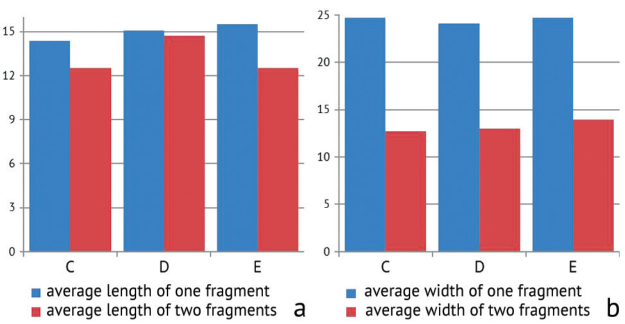


Fig. 6 Average length (a) and average width (b) of one or two fragments in mm and neurological status according to ASIA

The change in the position of bone fragments displaced into the spinal canal was also affected by the time from injury to surgery. Earlier terms of surgical interventions in group 1 allowed to achieve better results (p = 0.01). The PVH was comparable both before and after surgery in the two groups. Restored PVH did not statistically significantly affect the position of the intracanal bone fragment (p = 0.31). The PVH was almost 100 % restored in two groups. The average width of the spinal canal at the L1 level was 22.2 ± 0.34 mm in the first group, and 22.2 ± 0.38 mm in the second group. With the divergent pedicles of the arches seen in the majority of patients, the actual dimensions of the spinal canal at the L1 level measured 22.2 ± 0.34 mm in the first group and 22.1 ± 0.38 mm in the second group.

The depth of bone fragments displaced into the spinal canal (X) did not affect the effectiveness of the closed decompression. Preoperative X value was statistically higher (p = 0.006) and statistically lower (p = 0.0001) postoperatively in group 1. It could be associated with the operating time, because reparative processes in the spinal canal placed limit to the displacement of bone fragments at a long term. Differences in the angles of kyphotic deformity (α) before and after surgery were not statistically significant. before surgery, the average angle measured preoperatively 5.9 ± 1.6 degrees in the first group and 6.6 ± 2.3 degrees in the second group; 6.1 ± 1.3 and 3.8 ± 2.0 degrees, respectively, postoperatively. The mean preoperative angles of rotation of bone fragments (β) were close in the two groups (p = 0.38). The average angle of rotation of bone fragments with a 95 % confidence interval ranged 26.9-35.8 in the first group and 25.2-34.4 degrees in the second group. Postoperative angle of rotation of the bone fragments significantly decreased in the first group and led to better reformation of the spinal canal.

The angles between the lower cortical plate of the overlying vertebral body and the cortical plate of the bone fragment (λ) were close in the two groups (p = 0.28). The angle did not change postoperatively in the first group with a slight, statistically insignificant increase from 38.9 ± 3.2 to 41.1 ± 4.0 degrees in the second group. We found no explanation to this. There was no statistically significant difference in the height of bone fragments between the groups (p = 0.56). There was a significant difference in the width of the fragment (p = 0.03) between the groups. The average width of the fragment was 22.3 ± 2.6 mm in the second group, versus 18.2 ± 2.3 mm in the first. We calculated the ratio of the width of the bone fragment to the true transverse diameter of the spinal canal and obtained a statistically higher percentage in the second group (p = 0.015). If the ratio of the width of the bone fragment to the true transverse diameter of the spinal canal was more than 86.2 ± 9.6 % the fragment could be displaced from the spinal canal by more than 50 %. Figure 7 shows a clinical example of the effective transpedicular reposition in a comminuted fracture of the L1 vertebral body. The shape and size of the damaged vertebral body could be restored with transpedicular repositioning system and closed decompression of the spinal cord could be produced.

Table 2

Results of statistical analysis of pre- and postoperative parameters in two groups

Description (units)	Mean and 95 % C.I.		Significance (P)
	Group 1	Group 2	
Tme from injury to surgery (day)	6.7 ± 3 (3.6–9.8)	15.5 ± 5.6 (9.6–21.5)	0.01
Number of bone fragments (1 or 2)	1.5 ± 0.2	1.2 ± 0.2	0.01
Fragment height (mm)	14.4 ± 1.2 (13.2–15.5)	13.9 ± 1.1 (12.8–15.1)	0.56
Fragment width (mm)	18.2 ± 2.3 (15.5–20.9)	22.3 ± 2.6 (19.5–25.1)	0.03
Deficit of lumen of the spinal canal (%)	51.3 ± 4.6 (46.5–56.2)	42.9 ± 5.0 (37.7–48.1)	0.018
Deficit of area of the spinal canal (%)	47.2 ± 5.8 (41.2–53.3)	38.4 ± 6.7 (31.3–45.5)	0.05
Pre-op PVH (%)	92 ± 1.5 (90.4–93.5)	93.5 ± 1.5 (92.0–95.0)	0.15
Pre-op X (mm)	8.2 ± 0.7 (7.5 – 9.0)	6.7 ± 0.8 (5.8 – 7.5)	0.006
Pre-op α angle (degrees)	-5.9 ± 1.6 (-7.6 – - 4.2)	-6.6 ± 2.3 (-9.1 – -4.2)	0.6
Pre-op β angle (degrees)	31.2 ± 4.2 (26.9–35.8)	29.8 ± 4.2 (25.2–-34.4)	0.38
Pre-op λ angle (degrees)	42.1 ± 5.0 (36.9–47.4)	38.9 ± 3.2 (35.4–42.4)	0.28
The transverse diameter of the spinal canal calculated (mm)	22.2 ± 0.34 (21.8–22.5)	22.1 ± 0.38 (21.7–22.6)	0.81
True transverse diameter of the spinal canal (mm)	26.0 ± 0.9 (25.1–26.9)	25.2 ± 1.0 (24.2–26.2)	0.79
The width of the bone fragment relative to the true transverse diameter of the spinal canal (%)	69.2 ± 9.2 (59.5–78.9)	86.2 ± 9.6 (76.1–96.2)	0.015
Post-op X (mm)	3.2 ± 0.5 (2.7–3.8)	4.9 ± 0.5 (4.3–5.4)	0.0001
Post-op PVH (%)	97.7 ± 1.8 (95.7–99.7)	96.1 ± 2.0 (93.8–98.3)	0.31
Post-op α angle (degrees)	6.1 ± 1.3 (4.8–7.5)	3.8 ± 2.0 (1.6–6.0)	0.42
Post-op β angle (degrees)	14.8 ± 2.7 (12.0–17.6)	23.6 ± 4.1 (19.1–28.1)	0.002
Post-op λ angle (degrees)	42.6 ± 4.0 (38.5–-46.8)	41.1 ± 4.0 (36.6–45.5)	0.58
Average deformity correction angle (degrees)	11.7 ± 1.2 (10.4–13.1)	10.6 ± 2.8 (7.5–13.7)	0.85

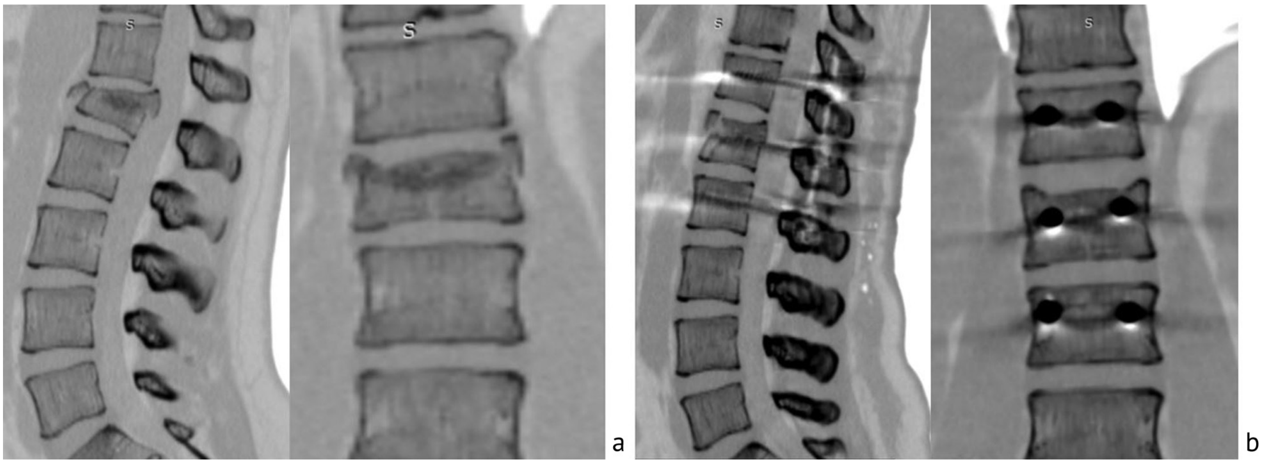


Fig. 7 SCT of the spine of a 38-year-old patient B. who sustained AO type A3 fracture of the body of the L1 vertebra: (a) preoperative scan; (b) postoperative scan

DISCUSSION

The process of destruction of the vertebral body develops in a certain sequence. The fracture can be caused by compression along the vertical axis with the initial rupture of the upper cortical plate and subsequent penetration of the nucleus pulposus into the vertebra breaking the body into separate fragments [22, 23]. The role of the nucleus pulposus in the mechanism of vertebral body fracture has been shown using dynamic loading with high-speed cineradiography [24]. The cortical plate broke with a load on the nucleus pulposus of up to 14,142 ± 486 N and the bone fragments were thrown into the spinal canal at a speed of about 2.9 m/s. Bone fragments protruding into the spinal canal remain a problem as they can cause neurological deficits after injury. The risk of neurological disorders significantly increases in stenosis: 35 % or more for the Th11-Th12 level, 45 % or more for the L1 level, 55 % or more for the L1-L3 level [25]. A scale for assessing the risk of neurological complications during surgical treatment of patients with post-traumatic deformity of the thoracic and lumbar spine was developed [26].

Neurological deficit in fractures of the thoracolumbar spine can also be assessed with computed tomography to evaluate the degree of stenosis of the spinal canal, the degree of compression of the anterior parts of the vertebral body measuring the distance from the intracanal bone fragment to the body of the overlying vertebra [27]. The effect of a bone fragment in the spinal canal on the recovery of neurological disorders remains unclear due to the fact that over time, bone fragments are resorbed and the spinal canal is remodeled [28].

Decompression of the spinal canal can be performed directly or indirectly. Indirect decompression of the spinal canal, the so-called ligamentotaxis, is closely associated with the posterior longitudinal ligament with the average width measuring 7.8 mm at the L1 level. The ratio of the width of the posterior longitudinal ligament to the width of the body of the L1 vertebra was 21 % [29]. Predicting the effective performance of ligamentotaxis is important for choosing the optimal surgical strategy. It is difficult to assess the integrity of the posterior longitudinal ligament using preoperative computed tomography or magnetic resonance imaging [30] and the expected effect of ligamentotaxis is difficult to accurately predict. The time from vertebral fracture to surgery is an important factor influencing the elimination of local post-traumatic deformity [31]. If the post-traumatic deformity is not addressed within 72 hours the malalignment is fixed, and scars develop in the spinal canal [32]. Closed repositioning decompression shows high efficiency in spinal cord injury in the lower thoracic and lumbar spine produced within 10 days [33].

Fractures in the thoracolumbar spine with incomplete neurological impairment can be effectively treated with indirect decompression without laminectomy [34]. Although indirect decompression of the spinal canal results in good remodeling of the spinal canal it may not improve neurological recovery [35]. Distraction and ligamentotaxis lead to restoration of the height of the body of the damaged vertebra, correction of kyphosis, displacement of bone fragments from the spinal canal, expansion of the canal and allow for indirect decompression of the spinal canal without resection of the compressing fragments [36]. The height of the involved vertebra can be restored during distraction. The L3 vertebral fracture model showed an increase d stress in the L2-L3 disc over the body of the involved vertebra by 154 % (from 0.93 to 2.37 MPa) in case of incompletely restored vertebral body height [37]. The posterior wall of the involved L1 vertebral body was almost 100 % restored in our series. Crutcher et al. reported almost 50 % reduction in spinal stenosis achieved through posterior distraction with ligamentotaxis [38]. Distraction that was applied before or after kyphosis correction demonstrated an effective mechanism for displacing bone fragments from the spinal canal [39]. However, excessive extension in the injured motor segment without distraction may compromise the displacement of the intracanal fragment [40]. Biomechanical studies of indirect reduction of bone fragments protruding into the spinal canal showed distraction as the determining factor in generating force in the posterior longitudinal ligament. Correction of the angulation prior to distraction significantly weakens the posterior longitudinal ligament and distraction is recommended to be performed prior to angulation [41]. The average force during distraction which led to rupture of the posterior longitudinal ligament, measured 48.3 N in the cervical spine, 61.3 N in the thoracic spine, and 48.8 N in the lumbar spine [42].

The intracanal bone fragment can turn up to 180° in comminuted fractures of the vertebral bodies so that the cancellous bone becomes posteriorly turned [43]. This indicates that the free bone fragment of the fracture is completely separated from the ligament. In this case, distraction can lead to displacement of the fragment towards the spinal cord, which is a contraindication for ligamentotaxis [44]. An intact posterior annulus, initially attached to the end plate of the bone fragment, prevents the fragment from turning more than 90°, and ligamentotaxis is indicated in the cases [45]. Rupture of the posterior longitudinal ligament can be assumed if the deficit of the lumen of the spinal canal is 52 % on CT scans, and the angle of rotation of the bone fragment is 33 degrees [46]. There is a correlation between the size of the bone fragment and injury to the posterior longitudinal ligament [47, 48]. Large bone fragments resist reduction with ligamentotaxis. With the width of the bone fragment being more than 75 % of the transverse diameter of the spinal canal and the height being more than 47 % of the height of the injured vertebrae closed decompression could not be performed due to ligamentotaxis [49].

In our series, the bone fragment could be displaced from the spinal canal by 50 % with a bone fragment width of 86.2 ± 9.6 % in the transverse diameter. The entrapped bone fragment in the rupture of the posterior longitudinal ligament is reported to cause ineffective ligamentotaxis. Tan et al. reported no correlation between posterior longitudinal ligament injury, local kyphosis, and the degree of vertebral body compression [50]. The displacement distance and the angle of rotation of the bone fragment were shown to be the most important parameters indicating the final position of the fragment after ligamentotaxis [51]. A displacement distance greater than 0.85 cm and a rotation angle greater than 55 degrees were the 2 criteria for treatment failure reported by Wang et al.

CONCLUSION

There was no significant correlation between the lumen deficit and the deficit of the area of the spinal canal, the length and width of the intracanal bone fragment and neurological disorders ASIA types C, D and E in comminuted fractures of the L1 vertebral body. The width of the intracanal bone fragments and the time from injury to surgery were the factors that made an impact on closed decompression of the spinal cord in spinal cord injury at the L1 level. The fragment could not be shifted from the spinal canal by more than 50 % with the ratio of the width of the bone fragment to the transverse diameter of the spinal canal being greater than 86.2 ± 9.6 %. The fragment was shifted from the spinal canal by greater than 50 % with the width of the bone fragment measuring less than 69.2 ± 9.2 % of the transverse diameter of the spinal canal.

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