

Factors affecting neurological deficits and intractable back pain in patients with insufficient bone union following osteoporotic vertebral fracture

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Abstract The purpose of this study was to examine factors affecting the severity of neurological deficits and intractable back pain in patients with insufficient bone union following osteoporotic vertebral fracture (OVF). Reports of insufficient union following OVF have recently increased. Patients with this lesion have various degrees of neurological deficits and back pain. However, the factors contributing to the severity of these are still unknown. A total of 45 patients with insufficient union following OVF were included in this study. Insufficient union was diagnosed based on the findings of vertebral cleft on plain radiography or CT, as well as fluid collection indicating high-intensity change on T2-weighted MRI. Multivariate logistic regression analysis was performed to determine the factors contributing to the severity of neurological deficits and back pain in the patients. Age, sex, level of fracture, duration after onset of symptoms, degree of local kyphosis, degree of angular instability, ratio of occupation by bony fragments, presence or absence of protrusion of flavum, and presence or absence of ossification of the anterior longitudinal ligament (OALL) in the adjacent level were used as explanatory variables, while severity of neurological

deficits and back pain were response variables. On multivariate analysis, factors significantly affecting the severity of neurological deficits were angular instability of more than 15° [adjusted odds ratio (OR), 9.24 (95% confidence interval, CI 1.49–57.2); $P < 0.05$] and ratio of occupation by bony fragments in the spinal canal of more than 42% [adjusted OR 9.23 (95%CI 1.15–74.1); $P < 0.05$]. The factor significantly affecting the severity of back pain was angular instability of more than 15° [adjusted OR 14.9 (95%CI 2.11–105); $P < 0.01$]. On the other hand, presence of OALL in the adjacent level reduced degree of back pain [adjusted OR 0.14 (95%CI 0.03–0.76); $P < 0.05$]. In this study, pronounced angular instability and marked posterior protrusion of bony fragments in the canal were factors affecting neurological deficits. In addition, marked angular instability was a factor affecting back pain. These findings are useful in determining treatment options for patients with insufficient union following OVF.

Keywords Osteoporotic vertebral fracture · Risk factor · Insufficient bone union · Neurological deficit · Back pain

Introduction

Fracture associated with osteoporosis has become a major problem because the population of elderly individuals has been increasing [4]. Modalities efficacious in preventing as well as treating osteoporosis-associated fractures are thus desired in the coming decade [7]. Among such fractures, vertebral fractures are the most common type with significant morbidity [24].

The osteoporotic vertebral fracture (OVF) has severe impact on activities of daily living and quality of life in

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elderly patients and is the beginning of a long-lasting deterioration of the patient's health [28]. Some patients present with intractable back pain for prolonged periods of time, while others suffer from neurological deficits within a few months' time after fracture. In such cases, insufficient union is often noted on plain radiography and/or MRI [18, 22]. Recently, reports of insufficient union have been increasing [13, 15, 19, 26, 29].

Patients with insufficient union following OVF have been treated with various methods conservatively and surgically. Surgical procedures include resection of fractured vertebral bodies and grafting of autologous bone with implants through the anterior approach are one option, while closed-wedge osteotomy through the posterior approach has been another [9, 20, 25, 27]. However, considering the age and comorbidities of affected patients, each of these procedures is rather invasive. Vertebroplasty is less invasive and has been reported to yield successful clinical results for painful OVF, but not to yield satisfactory results for patients with neurological deficits [2, 8, 14, 23].

Thus, the treatment options for insufficient union following OVF have not been clearly established. To elucidate useful treatment options for this lesion, it is necessary to evaluate the factors contributing to neurological deficits and/or intractable back pain associated with this type of fracture. The purpose of this study was to elucidate the factors affecting the severity of neurological deficits and intractable back pain in patients with insufficient bone union following OVF.

Material and methods

Patient population

A total of 45 patients treated for insufficient union following OVF were retrospectively reviewed. Insufficient union was diagnosed based on intravertebral vacuum cleft on plain radiography or CT, as well as fluid collection on T2-weighted MRI within the vertebral body. All patients had continued to complain of symptoms for longer than 2 months (range 2–36 months, mean 6.9 months).

There were 10 men and 35 women whose mean age at the time of diagnosis was 74 years (range 56–87 years). The fracture levels were T9 in 1 patient, T11 in 3, T12 in 22, L1 in 11, L2 in 6, L4 in 1, and L5 in 1. In these patients, bone mineral density was measured in the lumbar spine or femoral neck, and the diagnosis of osteoporosis was confirmed. Other metabolic bone diseases and malignancies such as myeloma and metastatic cancer were excluded. The precipitating events leading to the fracture were fall in 22 patients (48.8%), lift of a heavy object in 5 (11.1%), and no recollection of trauma in 18 (40%). The comorbidities were diabetes in 12 patients (26.7%) and rheumatoid arthritis in four (8.9%) take steroids. For neurological findings, 10 patients had a hyperreflexia and 19 a hyporeflexia in their legs. Twenty-five patients had a sensory deficit. Eleven patients had a sphincter dysfunction.

Eight patients were managed with posterior decompression and fusion surgery, 23 vertebroplasty, and 14 conservative therapies such as a corset or analgesics.

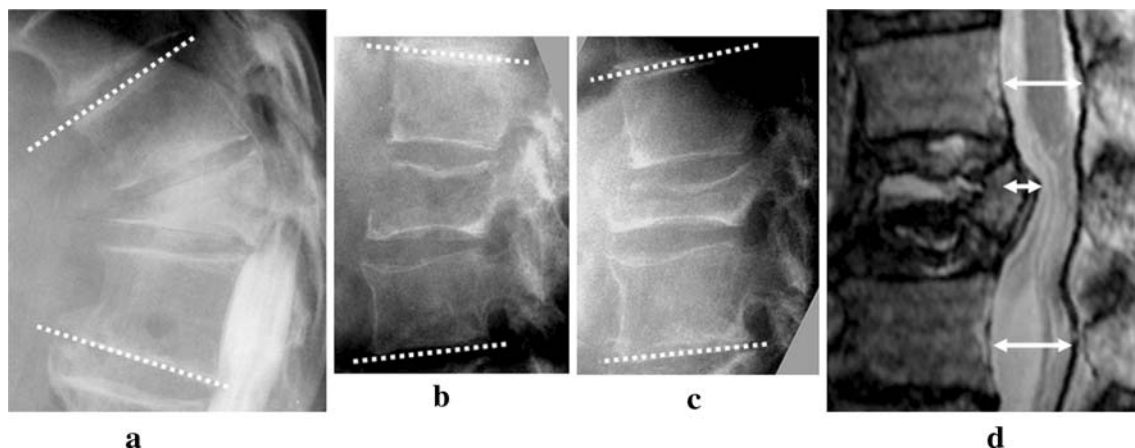


Fig. 1 Radiological assessment. **a** Local kyphosis was calculated as Cobb angle between the upper endplate of the vertebra just cranial to the fractured vertebra and the inferior endplate of the vertebra just caudal to the fractured vertebra on lateral plain X-ray films. **b, c** Angular instability was calculated as change in Cobb angle on

lateral dynamic radiography (**b** extension, **c** flexion). **d** Ratio of occupation by bony fragments of the spinal canal was calculated as ratio in percentage of bony fragment diameter to adjacent normal canal diameter on mid-sagittal MR images

Table 1 Patient demographics

Characteristics	No. of subjects	Mean or percent
Mean age (years)		74.0
Mean duration of fracture (months)		6.9
Sex		
Male	10	22.2%
Female	35	77.8%
Level of injury		
L2 or below	8	17.8%
L1	11	24.4%
T12 or above	26	57.8%
Mean local kyphosis (°)		24.8
Mean angular instability (°)		11.4
Mean ratio of occupation by bony fragments (%)		38.1
Protrusion of flavum		
Absent	36	80%
Present	9	20%
Adjacent OALL		
Absent	31	71.1%
Present	13	28.9%
Neurological deficits		
None	24	53.3%
Mild	10	22.2%
Severe	11	24.5%
Back pain		
None or mild	7	15.6%
Moderate	16	35.6%
Severe	22	48.8%

Radiological assessment

Local kyphosis was calculated as the Cobb angle between the cranial endplate of the vertebra just cranial to the fracture and the caudal endplate of the vertebra just caudal to the fractured vertebra on lateral plain X-ray films (Fig. 1). Angular instability was calculated as change in vertebral wedge angle on lateral dynamic radiography. Ratio of occupation by bony fragments of the spinal canal was calculated as the ratio in percentage of sagittal diameter of bony fragments to sagittal diameter of the spinal canal on mid-sagittal MR images.

Statistical analysis

As response variables, severities of neurological deficits and of back pain were each graded using three levels. For neurological deficits, “none” was defined as no neurological deficit, “mild” as Manual Muscle Test (MMT) score of grade 4, and “severe” as MMT score of grade 3 or

less. For back pain, “none or mild” was defined as VAS score less than 30, “moderate” as VAS score from 30 to 70, and “severe” as VAS score above 70.

Explanatory variables included age, sex, level of fracture, duration of symptoms after onset, angle of local kyphosis, degree of angular instability, ratio of occupation by bony fragments of the spinal canal, presence or absence of protrusion of flavum in the spinal canal, and presence or absence of ossification of the anterior longitudinal ligament (OALL) in the adjacent level. Continuous variables (angle of local kyphosis, degree of angular instability, and ratio of occupation by bony fragments of the spinal canal) were categorized by approximate tertile, except for age, and duration of fracture. Levels of fracture were divided into three categories. The first category was the level cranial to T12, that of the spinal cord. The second category was the level of L1, that of the conus medullaris. The third category was L2 and caudal to L2, the levels of the cauda equina. Other characteristics were treated as dichotomous variables (male/female for sex, present/absent for protrusion of flavum, or adjacent OALL).

To express the associations between severe neurological deficits or severe back pain and explanatory variables, odds ratio (ORs) and their 95% confidence intervals (CIs) were computed using the proportional odds model in logistic regression. We calculated *P* values for the scores to test the proportional odds assumption in order to confirm that use of the proportional odds model would be appropriate for these models.

All analyses were performed using Statistical Analysis System Version 9.1 (SAS Institute, Inc., Cary, NC, USA). Findings of *P* < 0.05 were considered significant.

Results

Patient demographic variables are shown in Table 1. Overall, 46.7% of patients exhibited mild or severe neurological deficits, with findings of less than grade 4 on MMT, and 84.4% had moderate or severe back pain, with a VAS score of more than 30.

Neurological deficits

Table 2 shows the associations between explanatory variables and severity of neurological deficits. Univariate analysis showed that pronounced angular instability within the affected vertebral body of more than 15° significantly increased the OR of neurological deficits [crude OR, 10.0 (95%CI 2.12–47.7); *P* < 0.01]. Marked spinal canal encroachment by protruding bony fragments was also a factor significantly contributing to neurological deficits. ORs were 6.74 (95%CI 1.11–40.9, *P* < 0.05) for moderate protrusion from 33 to 41% and 11.9 [(95%CI 2.11–68.0);

Table 2 Univariate and multivariate ORs for severity of neurological deficits

Characteristics	Neurological deficits			Univariate		Multivariate	
	None <i>n</i>	Mild <i>n</i>	Severe <i>n</i>	OR (95%CI)	<i>P</i> value	OR (95%CI)	<i>P</i> value
Age (year) per 1 year				0.96 (0.89–1.04)	0.341	0.91 (0.82–1.02)	0.106
Sex							
Male	5	4	1	1		1	
Female	19	6	10	1.20 (0.31–4.66)	0.794	0.66 (0.12–3.69)	0.634
Level of injury							
L2 or below	4	4	0	1		1	
L1	6	2	3	2.09 (0.37–11.8)	0.406	3.31 (0.31–35.6)	0.323
T12 or above	14	4	8	2.06 (0.47–9.05)	0.341	4.87 (0.66–35.9)	0.120
Duration of fracture (month) per 1 month				0.96 (0.85–1.08)	0.532	0.99 (0.84–1.18)	0.932
Local kyphosis (°)							
–21	5	6	2	1		1	
22–28	10	3	2	0.45 (0.10–1.94)	0.281	0.31 (0.04–2.44)	0.263
29+	9	1	7	1.14 (0.29–4.37)	0.855	0.62 (0.08–4.97)	0.653
Angular instability(°)							
–7	12	1	2	1		1	
8–14	7	5	1	2.59 (0.51–13.2)	0.252	2.20 (0.25–19.6)	0.479
15+	5	4	8	10.0 (2.12–47.7)	0.004	9.24 (1.49–57.2)	0.017
Ratio of occupation by bony fragments (%)							
–32	12	1	1	1		1	
33–41	5	6	2	6.74 (1.11–40.9)	0.038	2.35 (0.29–18.9)	0.421
42+	7	3	8	11.9 (2.11–68.0)	0.005	9.23 (1.15–74.1)	0.037
Protrusion of flavum							
Absent	21	7	8	1		1	
Present	3	3	3	2.27 (0.58–8.94)	0.241	0.56 (0.08–4.06)	0.569
Adjacent OALL							
Absent	17	8	7	1		1	
Present	7	2	4	1.15 (0.34–3.89)	0.828	1.79 (0.35–9.26)	0.483

OR odds ratio, CI confidence interval

$P < 0.01$] for severe protrusion of more than 42%. No other explanatory variables examined significantly contributed to neurological deficits on univariate analysis.

Multivariate analysis showed that pronounced angular instability in the upper tertile, of more than 15°, and high ratio of occupation by bony fragments, in the upper tertile of more than 42%, were factors significantly contributing to the degree of neurological deficit [adjusted OR 9.24 (95%CI 1.49–57.2); $P < 0.05$ and adjusted OR 9.23 (95%CI 1.15–74.1); $P < 0.05$, respectively]. No other variables examined were significant factors on multivariate analysis (Fig. 2).

Intractable back pain

Table 3 shows the associations between explanatory variables and severity of back pain in patients with

insufficient union following OVF. Univariate analysis revealed that moderate angular instability ranging from 8° to 14° had an OR of 4.45 [(95%CI 1.01–19.6); $P < 0.05$] while marked angular instability of more than 15° had an OR of 9.83 [(95%CI 2.19–44.1); $P < 0.01$]. No other variables examined were significant factors on univariate analysis.

Multivariate analysis showed that pronounced angular instability of more than 15° was a factor significantly contributing to intractable pain [adjusted OR 14.9 (95%CI 2.11–105); $P < 0.01$]. On the other hand, the existence of OALL in the level adjacent to the fracture was a factor reducing the degree of back pain [adjusted OR 0.14 (95%CI 0.03–0.76); $P < 0.05$]. No other variables examined were significant factors on multivariate analysis.

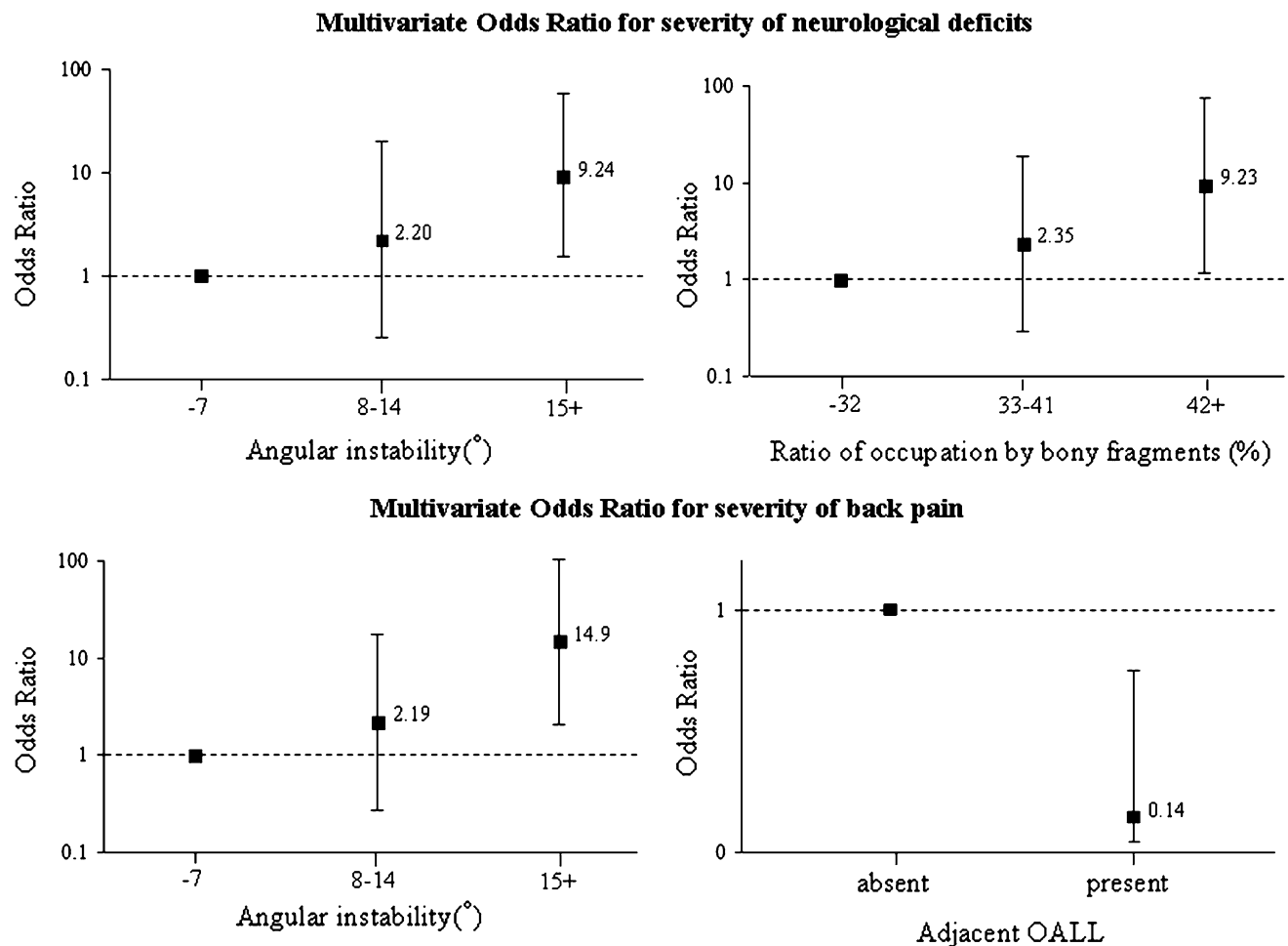


Fig. 2 Multivariate odds ratios for severity of neurological deficits and back pain. The y axis is on a log scale. The *I* bars denote 95% confidence intervals

Discussion

The pain associated with OVF usually improves if there is no severe residual deformity. Ettinger et al. reported that vertebral deformities cause marked back pain if vertebral height ratios fall four standard deviations below the normal mean [6]. However, pseudoarthrosis following OVF has recently come to be considered a poor prognostic factor for prolonged and intractable pain [10, 14, 16]. On the other hand, Hashidate et al. reported that in 66.7% of patients back pain decreased naturally despite the presence of vertebral instability in cases of vertebral pseudoarthrosis [11]. Thus, not all patients with non-union exhibited prolonged and intractable back pain. As noted above, the factors causing prolonged and intractable back pain in the case of insufficient bone union following OVF are still unknown. Toyone et al. reported a significant correlation between back pain and changes in wedging rate from supine to standing positions for OVF of recent onset [31]. In the present study, marked angular instability of the affected

vertebral body significantly contributed to severity of back pain.

The presence of an intravertebral cleft had been reported to be a risk factor for progressive vertebral collapse inducing neurological deficits [15, 17]. A few previous studies investigated the relationship between neurological deficits and radiological findings. Hashimoto et al. reported that ratios of occupation by bone fragments in the spinal canal of more than 35% at epiconus level, more than 45% at conus medullaris level, and more than 55% at cauda equina level were factors significantly associated with neurological impairment in traumatic burst fracture [12]. Trafton et al. reported that burst fracture at T12 or L1 with canal encroachment of more than 50% of mid-sagittal canal diameter was a significant risk factor for neurological impairment [32]. These reports were both on acute traumatic vertebral fracture. Nguyen et al. reported a series of ten patients with osteoporotic vertebral burst fracture, which led to varying degrees of neurologic compromise. In their cases, mean

Table 3 Univariate and multivariate ORs for severity of back pain

Characteristics	Back pain			Univariate		Multivariate	
	None or mild <i>n</i>	Moderate <i>n</i>	Severe <i>n</i>	OR (95%CI)	<i>P</i> value	OR (95%CI)	<i>P</i> value
Age (year) per 1 year				0.98 (0.91–1.06)	0.658	1.05 (0.94–1.18)	0.406
Sex							
Male	2	4	4	1		1	
Female	5	12	18	1.56 (0.42–5.84)	0.509	2.15 (0.43–10.6)	0.348
Level of injury							
L2 or below	0	2	6	1		1	
L1	1	7	3	0.24 (0.04–1.48)	0.125	0.15 (0.01–213)	0.160
T12 or above	6	7	13	0.30 (0.06–1.44)	0.133	0.20 (0.02–1.96)	0.165
Duration of fracture (month) per 1 month				0.96 (0.87–1.07)	0.459	0.97 (0.83–1.14)	0.715
Local kyphosis (°)							
–21	1	3	9	1		1	
22–28	3	5	7	0.36 (0.08–1.63)	0.186	0.72 (0.10–5.35)	0.751
29+	3	8	6	0.28 (0.06–1.20)	0.086	0.37 (0.04–3.36)	0.377
Angular instability(°)							
–7	6	5	4	1		1	
8–14	0	7	6	4.45 (1.01–19.6)	0.048	2.19 (0.28–17.2)	0.456
15+	1	4	12	9.83 (2.19–44.1)	0.003	14.9 (2.11–105)	0.007
Ratio of occupation by bony fragments (%)							
–32	3	5	6	1		1	
33–41	1	3	9	3.19 (0.69–14.7)	0.137	4.72 (0.65–34.2)	0.124
42+	3	8	7	0.99 (0.27–3.63)	0.983	0.47 (0.07–3.29)	0.445
Protrusion of flavum							
Absent	5	13	18	1		1	
Present	2	3	4	0.72 (0.18–2.84)	0.958	4.04 (0.47–34.6)	0.202
Adjacent OALL							
Absent	3	11	18	1		1	
Present	4	5	4	0.30 (0.09–1.05)	0.059	0.14 (0.03–0.76)	0.023

OR odds ratio, CI confidence interval

canal compromise was 41% [21]. No reports were found in our review of the literature on the relationship between neurological deficits and intravertebral instability. Baba et al. reported 27 patients who suffered osteoporotic vertebral collapse with late neurological complications. They considered the pathology in this lesion to involve abnormal hypermobility at the fractured spinal level with gradual retropulsion of fractured fragments into the spinal canal, resulting in late paralysis [1].

In the present study, it was found that intravertebral instability contributes to the degree of neurological deficits in cases of insufficient union following OVF. Stabilization of the lesion site without decompression is, thus, a treatment option for this condition. Some studies have reported that treatment with immobilization without decompression, such as conservative management using a plaster body cast, vertebroplasty,

or posterior in situ stabilization, could result in neurological improvement with progression of bone union [3, 5, 30]. These findings suggest that spinal instability is related to the pathogenesis of neurological deficits.

In this study, the factors significantly contributing to neurological deficits were angular instability of more than 15° and ratio of occupation by bony fragments of the spinal canal of more than 42%. The factors contributing to severity of back pain were angular instability of more than 15° and absence of OALL in the adjacent segment. As noted above, intravertebral angular instability was a factor common to neurological deficits and back pain. Stabilization of the fractured vertebra thus appears to be the most important treatment option for insufficient fracture following OVF. In patients with bony encroachment of higher percentage, decompression is needed to obtain satisfactory surgical results.

Conclusions

In conclusion, we investigated the factors contributing to the severity of symptoms in patients with insufficient union following OVF. Factors significantly contributing to the severity of neurological deficits were angular instability of more than 15° within the affected vertebral body and ratio of occupation by bony fragments in the spinal canal of more than 42%. Factors significantly contributing to the severity of back pain were angular instability of more than 15° and absence of adjacent OALL. Angular instability was thus a factor common to both neurological deficits and prolonged back pain.

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