One-stage posterior instrumentation surgery for the treatment of osteoporotic vertebral collapse with neurological deficits

Hideki Sudo · Manabu Ito · Kuniyoshi Abumi ·
Yoshihisa Kotani · Masahiko Takahata ·
Yoshihiro Hojo · Akio Minami

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Abstract The number of reports describing osteoporotic vertebral fracture has increased as the number of elderly people has grown. Anterior decompression and fusion alone for the treatment of vertebral collapse is not easy for patients with comorbid medical problems and severe bone fragility. The purpose of the present study was to evaluate the efficacy of one-stage posterior instrumentation surgery for the treatment of osteoporotic vertebral collapse with neurological deficits. A consecutive series of 21 patients who sustained osteoporotic vertebral collapse with neurological deficits were managed with posterior decompression and short-segmental pedicle screw instrumentation augmented with ultra-high molecular weight polyethylene (UHMWP) cables with or without vertebroplasty using calcium phosphate cement. The mean follow-up was 42 months. All patients showed neurologic recovery. Segmental kyphotic angle at the instrumented level was significantly improved from an average preoperative kyphosis of 22.8–14.7 at a final follow-up. Spinal canal occupation was significantly reduced from an average before surgery of 40.4–19.1% at the final follow-up. Two patients experienced loosening of pedicle screws and three patients developed subsequent vertebral compression fractures within adjacent segments. However, these patients were effectively treated in a conservative fashion

without any additional surgery. Our results indicated that one-stage posterior instrumentation surgery augmented with UHMWP cables could provide significant neurological improvement in the treatment of osteoporotic vertebral collapse.

Keywords Osteoporosis · Vertebral collapse · Ultra-high molecular weight polyethylene cable · Posterior instrumentation

Introduction

The number of reports describing osteoporotic vertebral fracture has been steadily increasing as the number of elderly people has been growing in developed countries [14, 19]. Usually, vertebral fracture in osteoporosis is treated conservatively with rest and a brace. In spite of such treatment, some patients fail to improve their neurological deficits brought about by neural compression [11, 14, 19]. This compression is due to retropulsed bony fragments from the delayed collapsed vertebral body, restricting the spinal canal and causing kyphosis with instability, which severely reduce the patient's quality of life and independence [7].

The advantages of anterior instrumentation surgery with a vertebral spacer are the direct resection of the retropulsed bony fragment and the reconstruction of the stable anterior spinal column [7, 8, 19]. However, some patients who underwent anterior surgery for the treatment of osteoporotic vertebral collapse required additional posterior reinforcement because of multiple-level vertebral collapse, steroid-induced osteoporosis or low bone mineral density [5]. Moreover, an anterior approach was not easy to achieve in elderly patients with more severe comorbid

H. Sudo (✉)
Department of Advanced Medicine for Spine and Spinal Cord Disorders, Graduate School of Medicine, Hokkaido University, North-15, West-7, Kita-ku, Sapporo, Hokkaido 060-8638, Japan
e-mail: hidekisudo@yahoo.co.jp

M. Ito · K. Abumi · Y. Kotani · M. Takahata · Y. Hojo · A. Minami
Department of Orthopaedic Surgery, Graduate School of Medicine, Hokkaido University, Sapporo, Japan

medical problems [10, 11, 14]. Adjacent segment disease also increased in such patients who underwent additional long posterior fusion during long-term follow-up periods [3, 5].

Since 2003, we have performed one-stage posterior neural decompression and pedicle screw fixation augmented with ultra-high molecular weight polyethylene (UHMWP) cable with or without vertebroplasty to minimize the length of fusion segment and to reduce the risk of pedicle screw loosening. The objective of this study was to investigate the outcomes in the treatment of osteoporotic vertebral collapse with neurological deficits.

Materials and methods

A total of 21 consecutive patients (15 women and 6 men) with neurological deficits because of osteoporotic vertebral collapse were surgically managed and their outcomes retrospectively investigated (Table 1). All patients initially underwent conservative treatment, such as bed rest and thoracolumbosacral orthosis for several months, but vertebral collapse progressed gradually and neurological symptoms subsequently developed. Due to the delayed onset of symptoms, the estimated interval between back trauma or spontaneous compression fracture and hospital administration ranged from 4 to 10 months (average, 5.2 months). At the time the patients came to our hospital, it was difficult to treat with simple interventions such as vertebro- or kyphoplasty alone. Surgical indication for this study was vertebral collapse in the thoracic or lumbar spine with neurological problems and difficulty in walking due to paralysis of the lower extremities, which decreased the patients' quality of daily life. The patients were followed up for a minimum of 2 years after surgical treatment at our hospital. The average age at surgery was 69.3 (54–83) years. The causes of osteoporosis were primary osteoporosis in 8 patients and secondary osteoporosis in 13. DXA measurements of bone mineral density at the lumbar spine (L1–L4, if the collapsed vertebra was involved, the data of the vertebra were excluded) were made using a Hologic QDR1000 (Hologic, Bedford, MA, USA). The bone volume was $0.632 \pm 0.078 \text{ g/cm}^2$.

The compromised collapsed spinal vertebral levels were as follows: a single vertebra was collapsed in 17 patients. T8 was collapsed in one patient, T9 in one, T10 in one, T11 in three, T12 in five, L1 in two, L2 in two, and L3 in two. Two vertebrae were collapsed in three patients, T5 and 6 in one patient, T5 and 8 in one, and T12 and L2 in one. The remaining patient had four-level collapse of the T7–10 vertebral bodies.

Pre- and postoperative neurological status was assessed using a rating system based on the Japanese Orthopaedic

Association score (JOA score; the highest possible score for normal, well-being; 11 points for myelopathy by thoracic or thoracolumbar lesion; and 29 points for lumbar lesion below L2) and American Spine Injury Association (ASIA) impairment scale [4, 6].

Supine anteroposterior and lateral X-ray films of the thoracic and lumbar spine were taken before and immediately after surgery, and then radiographs obtained with the patient upright were available at the final follow-up. On X-ray films, the sagittal Cobb angle was measured as the angle between the upper endplate of the uppermost vertebra and the lower endplate of the lowest vertebra at the instrumented fusion levels before and after surgery [8, 10, 19, 21]. The anterior (AVBhr) and posterior vertebral body height ratio (PVBhr) was also measured as the percentage of the anterior and posterior vertebral height of the collapsed vertebra with respect to average heights of two adjacent vertebrae [10, 21]. When collapsed vertebra existed within more than two levels, the average vertebral body height ratio including adjacent uncollapsed vertebrae was calculated. Spinal canal occupation due to retropulsed bony fragments was measured on CT images before surgery and at the final follow-up (Fig. 1) [1].

Surgical procedures

Positioning on an operation table

The surgical procedures were performed in a prone position. Spontaneous reposition of the local kyphosis after lying on the frame was expected.

Decompression procedures

For the patients who showed recovery of neurological function during bed rest and whose radiological assessment showed mild spinal canal compromise, only posterior fixation without laminectomy was conducted because it was thought that the main factor causing delayed neurological deficits following vertebral collapse in the osteoporotic spine was instability of the spinal column at the fracture site rather than mechanical compression of the spinal cord by the bone fragments [1]. Otherwise, laminectomy of the affected vertebra was performed. When the affected vertebra were severely collapsed and neural compression due to retropulsed bony fragments remained severe even after laminectomy, the collapsed vertebral body was decancelled through the pedicles [9, 15]. After the medial part of both pedicles was resected using an air drill and rongeurs, cancellous bone and soft tissue within the vertebral body was removed. Then the cancellous bone of the body was pushed anteriorly into the body to create a cavity in the vertebra. After thinning the posterior wall, the remaining

Table 1 Summary of data obtained from 21 patients with osteoporotic vertebral collapse with neurological deficits

Case no.	Age (years)/sex	FU (mo)	Cause of osteoporosis	BMD	Vertebral level	Instrumented level	Pedicle screws (diameter × length) mm	Decompression	Vertebroplasty
1	67/F	69	Secondary	0.46	T12, L2	T10-L4	T10, 11 (5.5 × 35), L3, 4 (6.5 × 40)	LT (L1, 2)	None
2	76/F	59	Secondary (steroid)	0.68	T11	T10-12	T10 (5.5 × 35), T12 (5.5 × 35)	LT (T11)	T11
3	66/F	63	Secondary	0.53	T12	T10-L1	T10, 11, L1 (5.5 × 35)	LT (T12) and AD (T12)	None
4	72/F	58	Primary (senile)	0.69	T9	T8-10	T8, 10 (5.5 × 35)	LT (T9) and AD (T9)	None
5	75/F	58	Primary (senile)	0.70	T12	T10-L2	T10 (4.75 × 35), T11 (6.25 × 35), L2 (5.5 × 40)	LT (L1, 2)	T12
6	54/M	57	Secondary (steroid)	0.63	T5, 8	T3-10	T3, 4 (4.75 × 30), T9, 10 (4.75 × 35)	LT (T5, 8) and AD (T5, 8)	None
7	71/M	47	Primary (senile)	0.68	T11	T8-L2	T8, 9, 10 (5.5 × 35), L1, 2 (6.5 × 40)	LT (T11, 12) and AD (T11)	None
8	73/F	46	Secondary	0.57	L1	T11-L3	L11, L2 (6.25 × 40), T12 (5.5 × 35), L3 (6.25 × 40)	LT (T12, L1) and AD (L1)	None
9	74/F	45	Primary (senile)	0.59	T12	T11-L1	T11 (6.25 × 35), L1 (6.25 × 40)	None	T12
10	66/F	44	Secondary (steroid)	0.67	L2	L1-3	L1 (4.5 × 35), L3 (6.5 × 35)	None	L2
11	75/F	43	Primary (senile)	0.62	L2	L1-3	L1 (5.5 × 40), L3 (6.5 × 40)	None	L2
12	54/F	43	Secondary	0.58	T8	T7-9	T7, 9 (5.5 × 35)	LT (T8) and AD (T8)	None
13	70/F	35	Secondary	0.57	L3	L2-4	L2, 4 (6.0 × 40)	LT (L3) and AD (L3)	None
14	81/F	33	Secondary (steroid)	0.76	T5, 6	T3-9	T3, 4 (4.75 × 35), T7, 8, 9 (5.5 × 35)	LT (T5, 6) and AD (T5, 6)	None
15	67/F	31	Secondary	0.49	T7–10	T5-12	T5, 6 (5.5 × 30), T7 (5.5 × 35), T11, 12 (6.5 × 35)	LT (T9, 10)	None
16	59/M	27	Secondary	0.62	L3	L2-4	L2, 4 (6.0 × 40)	LT (L3)	None
17	64/F	26	Secondary (steroid)	0.65	T10	T8-12	T8, 9, 11, 12 (6.0 × 35)	LT (T9, 10)	None
18	65/F	25	Secondary (steroid)	0.67	T11	T10-L2	T10 (4.5 × 35), T12 (6.5 × 35)	LT (T11)	T11
19	76/M	24	Primary (senile)	0.73	L1	T12-L2	T12 (6.2 × 40), L2 (6.2 × 45)	LT (L1)	L1
20	67/M	24	Primary (senile)	0.68	T12	T10-L1	T10, 11, L1 (6.2 × 40)	LT (T12)	None
21	83/F	24	Primary (senile)	0.70	T12	T11-L1	T11 (5.5 × 35), L1 (5.5 × 40)	LT (T12)	T12

FU follow-up, BMD bone mineral density, LT laminectomy, AD anterior decompression through a posterior approach

posterior cortex was pushed into the body and dissected from the adherent dura so as to decompress the spinal canal.

Vertebroplasty

Vertebroplasty using calcium phosphate cement (CPC) was considered in all cases where intervertebral vacuum cleft

was identified within the collapsed vertebrae by preoperative flexion and extension radiographs [11, 20]. We use CPC for vertebroplasty instead of polymethylmethacrylate (PMMA), because CPC is a biocompatible self-hardening material with less local heating or toxic effect on surrounding bone tissue, which is degraded over time by creeping substitution, and can stimulate formation of new bone substrate at the bone–cement interface [10]. After

Fig. 1 Spinal canal occupation due to retropulsed bony fragments. As measured on CT images ($b/a \times 100$). Preoperative (*left*) and final follow-up (case 14)

necrotic tissue in the vertebral cavity was removed through the pedicles and flushed out by normal saline, contrast medium was introduced into the vertebral body to confirm that the medium had not leaked into the vessels or around the collapsed vertebra. Then, CPC was slowly applied to fill up the cavity. When the medium was seen to leak, vertebroplasty was not performed to prevent cement extravasation that is a well-known complication of vertebral body augmentation with cement.

Spinal instrumentation augmented with UHMWP cables

Principally, short segment pedicle screw instrumentation construct includes the adjacent vertebrae immediately above and below the collapsed vertebra [10, 21]. When the adjacent vertebra suffered from concomitant compression fracture, we extended the instrumentation to one further level above or below. When more than two vertebrae had collapsed, the fixation level included two above and two below the collapsed vertebrae. After assembly of the rods, augmentation with sublaminar cables using the NESPLON[®] cable system (Alfresa Pharma, Osaka, Japan) was performed within the instrumented levels to prevent the pullout of the screws. As previously reported in patients with adolescent idiopathic scoliosis, before placement of the cable, the ligamentum flavum was removed from the required interlaminar space at all levels that needed sublaminar fixation [18]. Then, the cables were bilaterally fixed to the rods by a double-loop sliding knot, which allowed further tightening without loosening (Fig. 2a, b). All the sublaminar cables were sequentially tightened twice at 20 kgf. Finally, local bone graft harvesting from the iliac crest was performed after decortication of the posterior lamina and transverse process of the fusion area (Figs. 3a, b; 4a, b). All the patients

who underwent reconstructive surgery were managed in a rigid thoracolumbosacral orthosis after surgery until bony fusion was completed, to prevent the loosening of screws and enhance the fusion rate, and prescribed anti-osteoporotic drugs such as bisphosphonates during the follow-up period.

Statistical analysis

Statistical analysis was performed using a paired *t* test for changes in each radiographic parameter and JOA scores. Statistical significance was set at $P < 0.05$.

Results

The mean operative time was 244 (170–380) min. The mean intraoperative blood loss was 478 (30–1,400) g. Among pedicle instrumentation surgery, vertebroplasty was added in eight cases and the retropulsed bony fragments in the spinal canal were resected through the pedicles in nine cases. Three patients underwent posterior fixation without neural decompression. The average fusion length was 3.4 segments. Fusion of two segments was performed in 11 cases, three segments in 2, four segments in 3, six segments in 3 and seven segments in 2 patients. All the patients were observed for a minimum of 24 months, with an average follow-up of 42 (24–69) months (Tables 1, 2, 3).

Preoperative neurologic deterioration and the course of postoperative development are shown in Table 2. All patients had lost the power of walking before surgery, but gained ambulatory ability at the final follow-up. Based on the ASIA neurologic grading system, five patients had grade B, ten had grade C, and six had grade D neurological status preoperatively. At the final follow-up, there were 14 patients whose neurological status had improved by one grade. Five patients had recovered by two grades. One patient recovered from grade B to grade E. One patient showed no change in ASIA neurologic grade (grade D). The average JOA score was significantly improved from 2.0 before surgery to 4.1 immediately after surgery in patients with myelopathy by thoracic or thoracolumbar lesion ($P < 0.0001$). The score at the final follow-up was 5.9, which showed significant improvement compared to the immediate postoperative result ($P < 0.0001$). For patients with lumbar lesion below L2, preoperative JOA score was significantly improved from 7.8 to 17.0 immediately after surgery ($P = 0.0201$) and the score at the final follow-up was 19.0, which did not show further improvement compared to the immediate postoperative result ($P > 0.05$).

Bone union of the collapsed vertebra and the instrumented fusion levels was assessed to be successful when

Fig. 2 Ultra-high molecular weight polyethylene cables are bilaterally fixed to the rods by a double-loop sliding knot, which allows further tightening without loosening (a). Intraoperative photograph. All the sublaminar cables are sequentially tightened twice at 20 kgf (b)

there was no change in the local kyphosis angle and sagittal Cobb angle on flexion and extension radiographs [10, 21]. Moreover, continuous bony bridging within the lamina and facet joint was confirmed at all levels of instrumentation by assessment using AP and oblique views [10, 21]. The diagnosis of solid spinal fusion was definitively confirmed with CT scans [10]. All the patients showed solid fusion 6–10 months after surgery.

The mean kyphotic angle at the instrumented spinal level was 22.8 before surgery, 9.6 immediately after surgery and 14.7 at the final follow-up. The average correction angle at the final follow-up was 8.0, which showed that the kyphotic angle had significantly improved from before surgery to the final follow-up ($P < 0.0001$). The average loss of correction angle was 5.1, which demonstrated that there was significant change from immediately after surgery to the final follow-up ($P = 0.0201$). The average AVBhr was significantly restored from 0.40 before surgery to 0.51 at the final follow-up ($P = 0.0025$). The average PVBhr was 0.36 before surgery and 0.34 at the final follow-up, which did not show significant improvement ($P > 0.05$). The average spinal canal occupation was significantly improved from 40.4% before surgery to 19.1% at the final follow-up ($P < 0.0001$) (Table 3).

No obvious surgery-related complications occurred except in one patient who developed acute myocardial infarction during the surgery and was treated conservatively. During the postoperative course, two cases showed some loosening of pedicle screws; however, bony fusion was observed without pullout of the screws at the final follow-up. Three patients developed subsequent vertebral compression fractures within the adjacent or nearby vertebrae above or below the fusion level (case 2, 9 and 10). All these patients suffered from one vertebral collapse and underwent short segment fixation (2 segments). Sagittal Cobb angle was 17, 5 and 7 immediately after surgery and 18, 6 and 10 at the final follow-up, respectively, which demonstrated that the sagittal profile was not significantly changed. They were conservatively treated without any additional surgery (Table 3).

Discussion

There have been several reports on one-stage posterior decompression and stabilization in the treatment of osteoporotic vertebral collapse [9, 11, 14, 15]. Shikata et al. [15] decompressed the spinal cord by performing laminectomies

Fig. 3 Lateral X-rays before surgery (a) and final follow-up (b) of a 74-year-old female patient (case 9). In this case, the short segment pedicle screw instrumentation was conducted at the adjacent vertebra immediately above and below the collapsed vertebra (T12), augmented with ultra-high molecular weight polyethylene cables. Vertebroplasty was also performed. New compression fracture was observed in the vertebra two levels below the stabilization, 10 months after surgery, which was managed conservatively

Fig. 4 Lateral X-ray before surgery (a) and final follow-up (b) of a 72-year-old female patient (case 4)

and removing the retropulsed bony fragments in the spinal canal through the posterior transpedicular route and stabilizing the posterior long 6–7 segments using Harrington sublaminar wiring systems. Kim et al. [9] performed the

same decompression procedure and reconstruction using pedicle screw fixation. However, there remains the issue of osteoporotic bone that makes it difficult for the spinal instrumentation to maintain the alignment of the fixed spine [21]. There is also a risk of loosening of the instrumentation when local kyphotic deformity remains [5, 7, 19, 21]. Patients with severe osteoporosis might have no other choice of obtaining long posterior fusion when a single posterior procedure is performed [5, 7, 21]. To enable both neural decompression and correction of kyphotic deformity, posterior closing wedge osteotomy including spinal shortening has been performed for treating delayed paraplegia after osteoporotic vertebral collapse [2, 14, 17]. However, laminectomy, total facetectomy and removal of other remaining posterolateral structures, including the pedicle, might bring greater instability in the remaining spinal components and limit the available space for bone grafting. Besides, this procedure is relatively invasive for elderly patients in terms of operating time and blood loss.

Recently, there have been several reports on managing fresh burst fracture in short posterior instrumentation with CPC [10, 12, 20, 21]. There has been only one report for the treatment of osteoporotic vertebral fracture managed with vertebroplasty plus short posterior instrumentation. Matsuyama et al. [11] preliminarily reported that vertebral reconstruction using CPC combined with pedicle screw instrumentation was a safe and useful surgical treatment in their five cases. However, there still remains a risk of loosening the instrumentation even if it is augmented with CPC. Although the pedicle screw fixation method is capable of achieving strong spinal fixation [21] and CPC improves the stiffness of the instrumented construct and reduces screw failure [12], the severity of osteoporosis plays a major role in the pullout force of the screws [16]. Thus, we introduced the UHMWP cable system to enhance the stability of the instrumented construct and to prevent the pullout of the pedicle screws. The present study showed that the correction angle was significantly decreased at the final follow-up and two cases experienced loosening of pedicle screws during the follow-up period. It was thought that the loss of correction occurred at the interface between bone and screws. With the introduction of the cable system, however, most of the present cases did not have loosening of screws and complete loss of screws, and osseous fusion was achieved at the final follow-up. Recently, the cable system has been developed as a new fixation or anchorage device for the spine [13, 18]. Compared with the conventional metal wire, the fundamental differences in the cable system are that it is a soft and flexible material with a flat cross-sectional configuration (5-mm width and less than 0.5-mm thickness), which are factors that might be important to avoid neural damage and to reduce focal distribution of the stress to the lamina [13, 18]. The flat

Table 2 Neurological data of 21 patients with osteoporotic vertebral collapse with neurological deficits

No	ASIA			JOA			Activity of daily life			Associated comorbidities
	Preop	Postop	Final	Preop	Postop	Final	Preop	Postop	Final	
1	C	C	D	1	3	6	Difficulty in sitting	Wheelchair	Assisted walk	Parkinson's disease, chronic thyroiditis
2	D	D	D	3	4	5	Difficulty in sitting	Wheelchair	Assisted walk	Rheumatoid arthritis, interstitial pneumonitis
3	C	D	E	5.5	6	9	Gait instability	Assisted walk	Walk	Uterus removal for cancer (anti cancer drugs)
4	D	D	E	3	5	5	Gait instability	Assisted walk	Walk	Anticoagulant drugs for brain infarction
5	B	C	D	0.5	2	6	Difficulty in rolling over	Wheelchair	Walk	Aneurysm of the thoracic aorta, atrial fibrillation
6	B	D	E	0.5	6	9	Difficulty in rolling over	Assisted walk	Walk	Systemic lupus erythematosus, hypertrophic cardiomyopathy
7	C	D	D	2.5	4.5	5.5	Gait instability	Assisted walk	Walk	Arrhythmia
8	D	D	E	2.5	4	5	Difficulty in sitting	Wheelchair	Assisted walk	Parkinson's disease
9	C	D	E	2	4	8	Difficulty in sitting	Assisted walk	Walk	Mitral and tricuspid regurgitation
10	D	E	E	8	17	17	Gait difficulty	Walk	Walk	Rheumatoid arthritis
11	D	E	E	11	23	23	Gait difficulty	Walk	Walk	Hypertension
12	B	C	C	0.5	3	3	Difficulty in rolling over	Wheelchair	Assisted walk	Liver transplantation for liver cirrhosis, dialysis for chronic renal failure
13	B	C	C	1	13	16	Difficulty in rolling over	Wheelchair	Walk	Parkinson's disease
14	C	D	D	2.5	4	4	Gait instability	Assisted walk	Assisted walk	Rheumatoid arthritis
15	C	D	D	1	4	5	Difficulty in rolling over	Wheelchair	Assisted walk	Undernutrition
16	D	D	E	11	15	20	Gait instability	Walk	Walk	Asthma, liver malfunction
17	B	C	D	0	2	4	Difficulty in rolling over	Assisted wheelchair	Assisted walk	Systemic lupus erythematosus
18	C	D	D	4	4.5	7	Gait instability	Assisted walk	Walk	Rheumatoid arthritis, malignant lymphoma
19	C	D	D	2	6	7	Difficulty in sitting	Assisted walk	Walk	Diabetes mellitus
20	C	D	D	2	4	7	Difficulty in sitting	Assisted walk	Walk	Atrial fibrillation
21	C	D	E	1	4.5	5.5	Difficulty in sitting	Assisted walk	Walk	Hypertension

Assisted walk = walk with a cane, walker or care assistant

configuration has benefits for osteoporotic patients. The lamina of these patients is so fragile that focal distribution of a load on the narrow contact surface between bone and metal wire may cause fracture. Conversely, the flat configuration of the cable system distributes the load over a large contact area of lamina [18].

In the present study, the incidence of additional vertebral fracture at the adjacent segment during follow-up was 14%, which was less than that in previous reports [1, 3]. Ataka et al. [1] investigated 14 consecutive patients who had incomplete neurological deficits following vertebral collapse in the osteoporotic thoracolumbar spine and underwent posterior instrumented fusion without neural

decompression. They reported that 36% of patients showed vertebral fractures adjacent to the fused level (average fused segment was 6.2 segments). The average fusion length of the present study was 3.4 segments. Although the patients in this study received some anti-osteoporotic drugs after surgery, which might affect the rate of new fractures, the relatively short segments might explain the low incidence of vertebral fracture.

This study has some limitations. The present study was not designed to obtain any insight into the effects of postsurgical application of bracing and/or UHMWP cable system on the rate of screw loosening in osteoporotic bone, because this study had no control over comparing those

Table 3 Radiographic data of 21 patients with osteoporotic vertebral collapse with neurological deficits

No	Cobb angle			AVBHR		PVBHR		SCO (%)		Complications
	Preop	Postop	Final	Preop	Final	Preop	Final	Preop	Final	
1	15	5	5	0.25	0.28	0.35	0.33	52	32	Pedicle screw loosening (L3)
2	23	17	18	0.22	0.29	0.3	0.21	63	23	T7, 9 and L3 compression fracture (postop 53 months)
3	37	27	37	0.01	0.01	0.35	0.33	50	15	None
4	18	6	6	0.27	0.36	0.22	0.18	14	14	None
5	14	8	16	0.67	0.90	0.39	0.31	47	24	None
6	38	30	30	0.11	0.22	0.39	0.33	29	18	None
7	34	17	20	0.38	0.31	0.4	0.33	38	31	None
8	34	14	22	0.14	0.12	0.27	0.26	41	28	None
9	15	5	6	0.45	0.85	0.37	0.43	31	14	L3 compression fracture (postop 10 months)
10	11	7	10	0.73	0.75	0.33	0.39	35	15	L3 compression fracture (postop 10 months)
11	5	−5	−2	0.60	0.81	0.38	0.40	41	13	None
12	18	18	19	0.42	0.21	0.28	0.14	31	21	None
13	20	−13	29	0.43	0.38	0.28	0.27	56	25	Pedicle screw loosening (L2, 4)
14	38	30	30	0.37	0.43	0.34	0.33	50	0	None
15	40	30	30	0.52	0.67	0.35	0.31	36	0	None
16	6	−10	−8	0.59	0.83	0.44	0.37	53	38	None
17	7	3	3	0.69	0.79	0.42	0.43	8	0	None
18	13	−3	−1	0.56	0.68	0.36	0.39	29	21	None
19	25	−6	9	0.31	0.65	0.27	0.42	42	21	None
20	40	15	20	0.38	0.60	0.33	0.31	53	21	None
21	27	6	10	0.20	0.49	0.71	0.67	50	28	None

AVBHR anterior vertebral body height ratio, PVBHR posterior vertebral body height ratio, SCO spinal canal occupation

patients with instrumentation without the bracing and cables. Although surgical treatment of osteoporotic vertebral collapse is very difficult in any procedure, our goal of this treatment is to achieve bony fusion. Both the bracing and UHMWP cable are very important for osteoporotic patients to prevent screw loosening and we had to use every measure to enhance the fusion rate. Thus, the present study could not evaluate these effects on the rate of screw loosening separately. Also, this study could not explain the benefits of CPC instead of PMMA. Moreover, the present study did not have an objective measurable evidence of neurological damage that had improved after surgery, such as electrophysiological studies. It might be important to have some objective data about the motor function and pain-limiting ambulation in the future.

In conclusion, the current results indicated that one-stage posterior instrumentation surgery augmented with UHMWP cables could provide significant neurological improvement without additional surgery. Further long-term follow-up studies with an increased patient number within these clinical series are expected to draw definitive conclusions.

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