Analysis of risk factors of secondary adjacent vertebral fracture after percutaneous kyphoplasty.

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Abstract

Objective: To explore the potential risk factors of secondary adjacent vertebral fracture after percutaneous kyphoplasty (PKP) for Osteoporotic Vertebral Compression Fractures (OVCFs).

Methods: Clinical data of 189 OVCFs patients undergoing PKP were retrospectively analyzed. Gender, age, bone density and other parameters were analyzed. Risk factors causing NSVF after PKP were investigated.

Results: Among 189 enrolled patients, 22 presented with secondary adjacent vertebral fracture after PKP. Univariate analysis revealed no statistical significance in age, gender, severity of compression fracture, bone cement volume and restoration rate of vertebral height between patients with and without secondary adjacent vertebral fracture (all P>0.05). Type of fracture, type of bone cement, leakage of bone cement, anesthesia approach, whether use of systemic anti-osteoporosis treatment or not, bone density, quantity of surgically augmented vertebra and postoperative incidence of vertebral fracture significantly differed between two groups (all P<0.05). Multivariate logistic regression analysis demonstrated that type of fracture, whether use of systemic anti-osteoporosis treatment or not, leakage of bone cement into intervertebral disc, anesthesia approach and bone density acted as risk factors causing secondary adjacent vertebral fracture following PKP (all P<0.05).

Conclusions: Fissure fracture, absence of systemic anti-osteoporosis therapy, leakage of bone cement into intervertebral disc, topical anesthesia and loss in bone density are high-risk factors of secondary adjacent vertebral fracture after PKP.

Introduction

A compression fracture is a collapse of a vertebra probably due to trauma or a weakening of the vertebra, which is frequently encountered in osteoporosis patients. In China, over 4 million osteoporosis patients suffer from vertebral compression fracture and 700 000 of them require clinical treatment due to untolerable pain [1]. For stable and mild injuries, conservative therapies are recommended, such as back brace for support, use of non-steroidal anti-inflammatory medications, etc.

Kyphoplasty and vertebroplasty are minimally invasive procedures in which bone cement is injected into the fractured vertebra. Percutaneous kyphoplasty (PKP) has been gradually applied to treat vertebral compression fracture in clinical practice. Nevertheless, it is likely to lead to postoperative complications, such as secondary adjacent vertebral fracture.

The underlying reasons remain elusive [2-6]. In this study, clinical data and follow-up outcomes of 189 osteoporotic vertebral compression fractures (OVCFs) patients undergoing PKP between January 2013 and October 2015 were retrospectively analyzed, aiming to identify the risk factors of secondary adjacent vertebral fracture after PKP.

Materials and Methods

Baseline data

Clinical data of 189 OVCF patients undergoing PKP in our hospital between January 2013 and October 2015 were retrospectively analyzed. There were 41 male and 148 female, aged from 54 to 90 years with a mean age of (67.78 ± 7.02) years. Prior to surgery, a total of 189 fresh fractured vertebra were obtained and subject to PKP including T6 in 1, T7 in 2, T8 in 4, T9 in 4, T10 in 2, T11 in 17, T12 in 55, L1 in 66, L2 in 37, L3 in 8, 10 in L4 and L5 in 4 vertebra.

Inclusion criteria

Those with lumbar pain as the main symptom and the vertebral fracture site was consistent with that of the lumbar pain verified by X-ray, CT scan or MRI; those with vertebral pathological changes manifested as vertebral edema with hyperintense signal; those with vertebral deformation, vacuum sign or pseudoarthrosis formation detected by X-ray of the lumbar spine; those with spontaneous osteoporosis demonstrated by X-ray for bone density testing.

Exclusion criteria

Those complicated with nerve injury symptom; those with a medical history of spinal surgery; those with a medical history of long-term use of hormone; those with potential secondary diseases to OVCF, such as metabolic diseases, bacterial infection or malignant tumors; those with intact imaging data; those with follow-up duration<1 year. Written informed consents were obtained from all participants. The study procedures were approved by the local ethics committee of our institution.

Methods

Among all patients, 101 underwent surgery under general anesthesia and 88 under topical anesthesia. According to the type of bone cement of polymethyl methacrylate, all patients were divided into the rapid (5 min, n=83) and slow coagulation groups (15 min, n=106). Prior to general anesthesia, endotracheal intubation was delivered. After induction of anesthesia, the patients lay in a prone position with a pillow at the pubic symphysis. The fractured spine was compressed for approximately 10 min and covered by a sterile drape. Before topical anesthesia, patients lay in a prone position with a pillow at the pubic symphysis. The fractured spine was compressed for approximately 10 min and covered by a sterile drape containing 1% lignocaine and physiological saline prepared for regional full-thickness anesthesia.

After successful induction of anesthesia, a 0.5 cm incision was created and percutaneous puncture was performed at a extroversion angle of 10° to 15° into the pedicle of vertebral arch, and subsequently the puncture needle was inserted into the anterior 1/3 segment of the vertebra. The guide wire was inserted into the middle section of the compressed vertebra, then the guide wire was removed and a hole adjacent to the anterior margin of the vertebra was drilled and a sacculus was inserted through the working channel for expansion and compression. Then, the bone cement was mingled and prepared, slowly inserted into the vertebra via the working channel. The insertion speed was adjusted based upon the diffusion and filling status of bone cement. The insertion should be immediately terminated when the leakage of bone cement was observed. The working catheter was withdrawn after the bone cement was hardened. Intraoperatively, the variation in blood pressure was recorded. Postoperative, antibiotics medication was applied for 1 d. Patients were required to rest in bed for 12 h and then performed physical activities. During postoperative hospitalization, calcitonin was delivered via intramuscular injection once daily. After hospital discharge, compound alendronate sodium tablet was administered each week and Caltrate tablet was orally given once daily for anti-osteoporosis therapy. All patients were followed up at 1-, 3-, 6- and 12-month after discharge and subsequent follow-up once each year. During follow-up, anteriolateral X-ray of the thoracolumbar spine was performed in a standing position. For those with episode of lumbar pain, MRI examination was conducted to identify the incidence of fresh bone fracture.

Observation parameters

Clinical data of 189 OVCFs patients were retrospectively analyzed, including gender, age, bone density (X-ray test), severity of compression fracture (CT or MRI scan), type of fracture, quantity of surgically augmented vertebra (CT or MRI scan), type of bone cement, bone cement volume, leakage of bone cement, anesthesia approach, blood pressure variation before and after bone cement filling, restoration rate of vertebral height, whether adjacent to vertebral fracture or not, whether use of systemic anti-osteoporosis treatment or not, etc. The risk factors causing secondary adjacent vertebral fracture after PKP were analyzed. Severity of compression fracture was calculated according to the formula=preoperative vertebral height/estimated original vertebral height (average height between the two adjacent vertebra) × 100%. Restoration rate of vertebral height was equal=(postoperative vertebral height-preoperative vertebral height)/estimated original vertebral height-preoperative vertebral height) × 100%. The severity of compression fracture and restoration rate of vertebral height of the anterior and middle segments of the vertebra was calculated. The mean values of two computed data were defined as the compression ratio and recovery rate of each vertebra. The blood pressure variation was expressed as mean arterial pressure. Blood pressure variation=mean arterial pressure during surgery-daily mean arterial pressure. Mean arterial pressure was calculated as mean arterial pressure=(systolic blood pressure + 2 × diastolic blood pressure)/3.

Statistical analysis

SPSS 17.0 statistical software was utilized for data analysis (SPSS Inc., Chicago, USA). Enumeration data were statistically analyzed by χ^2 test. Measurement data were statistically analyzed by t-test or ANOVA. Univariate and multivariate logistic regression analysis was performed to identify the risk factors of secondary adjacent vertebral fracture after PKP. A P value of less than 0.05 was considered as statistical significance.

Results

Follow-up data

All patients successfully underwent the PKP. Postoperatively, no nerve injury was observed and most complications, such as

lung embolization were significantly mitigated. Lumbar and back pain was reported in few cases and alleviated after corresponding treatment. All patients received postoperative follow-up from 12 to 69 months with a mean duration of (36 ± 8) months.

Univariate analysis of risk factors

Univariate statistical analysis demonstrated that age, gender, vertebral compression rate, bone cement volume and restoration rate of vertebral height were not significantly correlated between the fracture and non-fracture groups (all P>0.05). However, type of fracture, type of bone cement, leakage of bone cement, anesthesia approach, whether use of systemic anti-osteoporosis treatment or not, bone density and quantity of surgically augmented vertebra were significantly correlated with postoperative incidence of vertebral fracture (all P<0.05), as illustrated in Table 1.

Table 1. Comparison of measurement parameters between the fracture and non-fracture groups.

	Fracture group (n=22)	Non-fracture group (n=167)	F value	P value
Age	66.83 ± 6.92	67.35 ± 5.55	F=2.431	P>0.05
Bone density	-3.89 ± 0.72	-3.13 ± 0.59	F=64.219	P<0.05
Severity of compression fracture	0.57 ± 0.35	0.39 ± 0.60	F=9.465	P>0.05
Quantity of surgically augmented vertebra	1.71 ± 0.78	1.13 ± 0.39	F=53.837	P<0.05
Bone cement volume	4.53 ± 1.64	4.72 ± 1.28	F=3.560	P>0.05
Restoration rate of vertebral height	0.49 ± 0.86	0.46 ± 0.94	F=7.543	P>0.05
Gender (male/female)	5/17	36/131	χ^2=0.16	P>0.05
Vertebral fissure fracture (yes/no)	10/12	8/159	χ^2=37.304	P<0.05
Anesthesia approach (topical/general)	15/7	73/94	χ^2=4.678	P<0.05
Type of bone cement (fast/slow)	14/8	69/98	χ^2=3.932	P<0.05
Leakage of bone cement into intervertebral disc (yes/no)	6/16	4/163	χ^2=24.008	P<0.05
Leakage of bone cement into peripheral tissue (yes/no)	8/14	30/137	χ^2=4.097	P<0.05
Systemic anti-osteoporosis treatment (yes/no)	7/15	117/50	χ^2=12.599	P<0.05

Multivariate analysis of risk factors

To exclude the potential confounding effect of single factors, the correlated risk factors were further subject to multivariate logistic regression analysis. The statistical results revealed that type of bone fracture, whether use of systemic anti-osteoporosis treatment or not, leakage of bone cement into intervertebral disc, anesthesia approach and bone density were the risk factors leading to the incidence of secondary adjacent vertebral fracture following PKP (all P<0.05), as illustrated in Table 2.

Discussion

PKP is an efficacious approach for treating kyphosis deformity, which yields mild trauma, evident analgesic effect and rapidly improves the quality of life of patients. Previous investigators have designed a randomized controlled trail and found that PKP is a significantly safer and more efficacious therapy compared with conservative therapy [7,8]. Nevertheless, secondary adjacent vertebral fracture is a common complication after PKP, which severely affects the surgical efficacy and quality of life of the patients. Previous research revealed that the incidence rate of secondary adjacent vertebral fracture after PKP is estimated to be 12% to 52% [9]. The risk factors leading to vertebral fracture remain debated. Some scholars consider that it is a natural progress of osteoporosis, whereas most researchers propose that adjacent vertebral fracture is a complication secondary to PKP [9]. A variety of parameters are considered as the potential risk factors including gender, age, bone density, vertebral compression rate, fracture type, quantity of surgically augmented vertebra, bone cement volume, leakage of bone cement, restoration rate of vertebral height and whether use of systemic anti-osteoporosis treatment or not, etc. [10]. A case-control study with a large sample size and long-term follow-up is urgently required to identify the risk factors of bone fracture following PKP. Reduction in bone density has been proven to be a high-risk factor of postoperative fracture [10,11].

Cummings et al. [12] have demonstrated that the risk of bone fracture is decreased by 3% along with 1% increase in bone density. In addition, Uppin et al. [13] have proposed that the more severe degree of osteoporosis, the higher risk of vertebral fracture. The findings in current investigation are consistent with the conclusion mentioned above. Moreover, we also

demonstrated that standard anti-osteoporosis treatment can significantly decrease the incidence of postoperative fracture after PKP. Nevertheless, the price of anti-osteoporosis medication is high, the cycle of medication use is long, the medical effect is gradual and slow and patients lack of deep understanding of osteoporosis. Hence, a majority of osteoporosis patients fail to receive standard anti-osteoporosis therapy for a long term, which significantly elevates the risk of postoperative fracture after PKP. How to deepen the understanding of osteoporosis and long-term treatment is of top priority. The effect of different anesthesia approaches upon the incidence of bone fracture after PKP has been rarely reported.

Table 2. *Logistic regression analysis between each correlation factor and postoperative incidence of secondary adjacent vertebral fracture.*

	Regression coefficient B	Regression coefficient standard error (S.E)	Wald χ^2 value	P value	Odds ratio
Fracture type	-1.999	0.878	5.180	0.023	0.135
Bone density	4.044	1.264	10.232	0.001	57.079
Type of bone cement	1.145	0.721	2.519	0.112	3.142
Bone cement leakage into intervertebral disc	2.194	0.877	6.265	0.012	8.975
Bone cement leakage into peripheral tissue	0.290	0.867	0.112	0.738	1.336
Quantity of surgically augmented vertebra	0.256	0.689	0.147	0.704	1.291
Systemic anti-osteoporosis treatment	2.270	1.166	4.368	0.024	3.793
Anesthesia approach	2.392	0.960	6.209	0.013	10.939

PKP under topical anesthesia is much safer compared with general anesthesia because the vertebral canal leakage can be identified and processed as early as possible [13]. Previous investigators have reported that the incidence of bone fracture after PKP under topical anesthesia ranges from 13.4% to 42.6% [14-17]. In this study, 88 patients underwent PKP under topical anesthesia and 15 cases presented with postoperative fracture with an incidence rate of 17.04%, which is consistent with previous findings [14-17]. Interestingly, the incidence rate is 6.93% in those undergoing PKP under general anesthesia, significantly lower compared with that in the topical anesthesia group. The underlying causes remain to be elucidated. During the early stage of sacculus expansion and bone cement filling, evident elevation in blood pressure was observed in patients undergoing PKP under topical anesthesia. However, during the late stage, blood pressure was significantly declined, which significantly differed from the blood pressure variation in the general anesthesia group. The obvious blood pressure rise probably results from acute pain during sacculus expansion and bone cement filling in patients under topical anesthesia. Vertebral venous pressure is elevated. During early stage of bone cement filling, bone cement is likely to diffuse into the lower margin of vertebral lamina with relative small pressure. During the late stage of bone cement filling, patients presented with allergic shock and then the blood pressure rapidly declines and the vertebral venous pressure decreases. However, bone cement is in coagulation status and lack of diffusivity. Consequently, non-uniform diffusion of bone cement is observed under topical anesthesia. Under general anesthesia, the blood pressure variation is smaller and the bone cement is more evenly distributed. Uneven distribution of bone cement may cause different stress pressure upon each segment, which probably increases the risk of secondary adjacent vertebral fracture.

Previous investigations have demonstrated that the anterior 1/3 segment of the vertebra plays a vital role in blood supply of the vertebra [17]. The fracture of this segment is likely to destroy vertebral arteriole, thereby leading to the incidence of ischemic osteonecrosis. Vertebral fissure-like lesion is a manifestation of vertebral ischemic necrosis, which is intimately linked to the incidence of OVCF after nonunion. This phenomenon is commonly encountered in clinical practice [18]. Normal bone structure is absent whereas bone necrosis is present in fissure-like lesions. Thus, bone cement is difficult to permeate into the fissure-like lesions. Additionally, the elastic modulus disparity between bone cement and vertebral fissure-like lesions may induce load shift and elevate the risk of bone fracture. Wiggins et al. [19] demonstrated that bone cement is distributed in masses within the fissure rather penetrate through the peripheral osteosclerosis band. Due to significant increase in axial load within the vertebral fissure, the incidence of vertebral fracture is considerably increased under long-term stress. In this investigation, postoperative relief of pain of patients with vertebral fissure-like lesions was worse and postoperative length of bed stay was longer compared with those of their counterparts without vertebral fissure-like lesions. Short-term administration of analgesics was required to relieve the pain. The incidence of secondary adjacent vertebral fracture was elevated after PKP with statistical significance (P<0.05). During early stage of vertebral fracture,

early treatment can decrease the risk of vertebral fissure-like lesions and improve postoperative clinical efficacy.

Table 3. Comparison of observation parameters between two anesthesia approaches.

	Quantity of postoperative fracture	Blood pressure variation during early stage of bone cement filling	Blood pressure variation during late stage of bone cement filling	Leakage of bone cement	Fracture of adjacent vertebra (yes/no)
Topical anesthesia (n=88)	15	37.7 ± 7.1	-21.6 ± 5.9	29	7/8
General anesthesia (n=101)	7	11.5 ± 4.6	7.9 ± 6.4	18	3/4
Statistical value	χ^2=4.678	F=57.673	F=64.257	χ^2=5.764	χ^2=0.28
	P<0.05	P<0.05	P<0.05	P<0.05	P>0.05

Leakage of bone cement is regarded as a common complication after PKP with a leakage rate up to 65% [20]. Bone cement may leak into the venous plexus, vertebral margin, vertebral canal and intervertebral disc. Vertebral canal leakage is likely to provoke paraplegia and nerve injury, whereas the incidence rate is relatively low. In this study, four patients developed vertebral canal leakage without nerve injury manifestations. Vertebral margin leakage yields no apparent clinical symptoms, which can be detected by postoperative radiography and CT scan. In our study, vertebral margin leakage and vertebral canal leakage were not significantly correlated with the incidence of postoperative fracture following PKP. However, leakage of bone cement into intervertebral disc was a high-risk factor causing bone fracture after PKP. In most cases, intervertebral disc leakage provokes no clinical symptoms, whereas it probably causes persistent high stress upon the intervertebral disc, accelerates endplate injury and fibrous ring degeneration, reduces the osmosis of intervertebral disc cells, affects absorption of nutrient content and metabolism of intervertebral disc, thereby leading to the degeneration of the entire intervertebral disc, reduction in spinal stability and high risk of postoperative fracture [21]. Preoperative imaging analysis was performed to observe the presence of endplate fracture. The puncture site should be distant from the endplate fracture. Bone cement filling at a constant and slow speed contributes to decreasing the risk of bone cement leakage. Previous investigators demonstrated that leakage of bone cement into intervertebral disc is likely to provoke 58% of adjacent vertebral fracture [22]. Therefore, when bone cement leakage into unilateral intervertebral disc is identified, PKP should be immediately performed for the adjacent vertebra, aiming to reduce the risk of postoperative fracture.

There are several limitations should be acknowledged. First, this retrospective single-center study has a small sample size. Second, it is a challenge to control the patients' conditions, especially pain, dysphoria and blood pressure fluctuation under topical anesthesia. Third, postoperative time and intensity of physical exercise are not considered in this study. The activity intensity of the patients is not explicitly evaluated by rehabilitation physicians, which is also a potential risk factor of postoperative fracture following PKP. Thus, a prospective study with a large sample size is urgently required to validate this conclusion.

Conclusion

Taken together, fissure fracture, no use of systemic anti-osteoporosis therapy, leakage of bone cement into intervertebral disc, topical anesthesia and reduction in bone density are high-risk factors of secondary adjacent vertebral fracture after PKP. Much attention should be diverted to the understanding of osteoporosis disease, early diagnosis and treatment of spinal fracture and enhancing surgical skills. PKP under general anesthesia is an effective approach to reduce the risk of secondary adjacent vertebral fracture after PKP.

References

1. Liu T, Zhou ZW, Zhou ZX, Xu SW. Percutaneous vertebroplasty versus percutaneous kyphoplasty for the treatment of old vertebral osteoporotic compression fracture. Chinese J Tissue Eng Res 2013; 17: 6920-6925.

2. Sun YC, Teng MM, Yuan WS, Luo CB, Chang FC, Lirng JF, Guo WY, Chang CY. Risk of post-vertebroplasty fracture in adjacent vertebral bodies appears correlated with the morphologic extent of bone cement. J Chin Med Assoc 2011; 74: 357-362.

3. Ahn Y, Lee JH, Lee HY, Lee SH, Keem SH. Predictive factors for subsequent vertebral fracture after percutaneous vertebroplasty. J Neurosurg Spine 2008; 9: 129-136.

4. Mudano AS, Bian J, Cope JU, Curtis JR, Gross TP, Allison JJ, Kim Y, Briggs D, Melton ME, Xi J, Saag KG. Vertebroplasly and kyphoplasty are associated with an increased risk of secondary vertebral compression fractures:a population-based cohortstudy. Osteoporos Int 2009; 20: 819-826.

5. Hadley C, Awan OA, Zoarski CH. Biomechanics of vertebral bone augmentation. Neuroimaging Clin N Am 2010; 20: 159-167.

6. Voormolen MH, Lohle PN, Juttmann JR, van der Graaf Y, Fransen H, Lampmann LE. The risk of new osteoporotic vertebral compression fractures in the year after percutaneous vertebroplasty. J Vasc Interv Radiol 2006; 17: 71-76.

7. Klazen CA, Lohle PN, de Vries J, Jansen FH, Tielbeek AV, Blonk MC, Venmans A, van Rooij WJ, Schoemaker MC, Juttmann JR, Lo TH, Verhaar HJ, van der Graaf Y, van Everdingen KJ, Muller AF, Elgersma OE, Halkema DR, Fransen H, Janssens X, Buskens E, Mali WP. Vertebroplasty versus conservative treatment in acute osteoporotic vertebral compression fractures (Vertos II): an open-label randomised trial. Lancet 2010; 376: 1085-1092.

8. Rousing R, Hansen KL, Andersen MO, Jespersen SM, Thomsen K, Lauritsen JM. Twelve-months follow-up in forty-nine patients with acute/semiacute osteoporotic vertebral fractures treated conservatively or with percutaneous vertebroplasty: a clinical randomized study. Spine (Phila Pa 1976) 2010; 35: 478-482.

9. Li YA, Lin CL, Chang MC, Liu CL, Chen TH, Lai SC. Subsequent vertebral fracture after vertebroplasty incidence and analysis of risk factors. Spine (Phila Pa 1976) 2012; 37: 179-183.

10. Klazen CA, Venmans A, de Vries J, van Rooij WJ, Jansen FH, Blonk MC, Lohle PN, Juttmann JR, Buskens E, van Everdingen KJ, Muller A, Fransen H, Elgersma OE, Mali WP, Verhaar HJ. Percutaneous vertebroplasty is not a risk factor for new osteoporotic compression fractures:results from VERTOS II. AJNR Am J Neuroradiol 2010; 31: 1447-1450.

11. Lin WC, Cheng TT, Lee YC, Wang TN, Cheng YF, Lui CC, Yu CY. New vertebral osteoporotic compression fractures after percutaneous vertebroplasty:retrospective analysis of risk factors. J Vasc Interv Radiol 2008; 19: 225-231.

12. Cummings SR, Karpf DB, Harris F, Genant HK, Ensrud K, LaCroix AZ, Black DM. Improvement in spine bone density and reduction in risk of venebral fractures during treatment with antiresorptive drugs. Am J Med 2002; 112: 281-289.

13. Uppin AA, Hirsch JA, Centenera LV, Pfiefer BA, Pazianos AG, Choi IS. Occurrence of newvertebral body fracture after percutaneous vertebroplasty in patients with osteoporosis. Radiology 2003; 226: 119-124.

14. Ou YC, Chen ZM, Ma HS. Analysis on the associated factors of non-surgical Vertebral fracture secondary to percutaneous kyphoplasty for osteoporotic vertebral compression fracture. Chinese J Spine Spinal Cord 2013; 23: 222-227.

15. Yu P, Xia Q, Pi HL, Jin HB, Wu QH. Associated factors for subsequent adjacent vertebral body refracture after percutaneous vertebroplasty. Chin J Trauma 2013; 29: 1063-1067.

16. Jia P, Fei Q, Li JJ, Wang BQ, Tang H. Correlative factor analysis on recurrent vertebral fractures after percutaneous kyphoplasty in the treatment of osteoporotic vertebral compression fracture. Chinese J Bone Joint Surgery 2013; 6: 153-156.

17. Sun H, Zang XH, Gao LH. Refracture following percutaneous vertebroplasty through polymethyl methacrylate injection in the paste period for osteoporotic vertebral compression fractures. J Clin Rehab Tissue Eng Res 2010; 14: 4657-4660.

18. Libicher M, Appelt A, Berger I, Baier M, Meeder PJ, Grafe I, Dafonseca K, Nöldge G, Kasperk C. The intravertebral vacuum phenomen as specific sign of osteonecrosis in vertebral compression fractures: results from a radiological and histological study. Eur Radiol 2007; 17: 2248-2252.

19. Wiggins MC, Sehizadeh M, Pilgram TK, Gilula LA. Importance of intravertebral fracture clefts in vertebroplasty outcome. AJR Am J Roentgenol 2007; 188: 634-640.

20. Chen WJ, Kao YH, Yang SC, Yu SW, Tu YK, Chung KC. Impact of cement leakage into disks on the development of adiacent vertebral compression fractures. J Spinal Disord Tech 2010; 23: 35-39.

21. Hutton WC, Elmer WA, Boden SD, Hyon S, Toribatake Y, Tomita K, Hair GA. The effect of hydrostatic pressure on intervertebral disc metabolism. Spine (Phila Pa 1976) 1999; 24: 1507-1515.

22. Lin EP, Ekholm S, Hiwatashi A, Westesson PL. Vertebroplasty:cement leakage into the disc increases the risk of new fracture of adjacent vertebral body. AJNR Am J Neuroradiol 2004; 25: 175-180.

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