表1 PVPの患者選択基準

適格条件	
1	悪性腫瘍の転移や原発性腫瘍による胸椎、腰椎の病変である。
2	1による疼痛が強く、日常生活の行動に制限を生じている。
3	主要臓器（骨髄、心、肝、肺、腎など）の機能が保たれている。
4	Performance status（ECOG）：3以下。
5	4週間以上の生存が見込める。
6	年齢20歳以上である。
除外条件	
1	補正しがたい出血傾向がある。
2	感染症を併発している。
3	薬物による維持療法が必要な心不全を伴っている。
4	骨病変に活動性炎症（結核性椎体炎、感染性椎体炎など）の疑いがある。
5	椎体後面の著しい破壊や脊髄の圧迫がみられる。（脊柱管の前後径が2/3以上に保たれ、脊髄と腫瘍の間に画像上脳脊髄液が介在する場合は可）
6	1回の治療で対象とする病変が4椎体以上存在する。
7	薬物、あるいは理学的処置を施しても治療体位が保持できない。
8	高度な薬物アレルギー歴を有する。
9	妊娠もしくは妊娠している可能性がある。

の生命予後が期待できず、手術適応がない患者を対象とすることが多い[9]。

有痛性骨腫瘍に対するPVPの治療適応

PVPの患者選択基準を表1に示す。胸椎および腰椎の椎体腫瘍で腫瘍による骨脆弱性に起因する強い疼痛を有し、外科手術の適応がないものが対象となる[10]。頸椎は技術的困難性・危険性などのため、一般的には対象としない。

多椎体に腫瘍がある症例に遭遇することは多いが、1回の治療で対象とする病巣が4椎体以上ある場合には慎重を期す必要がある。椎体の扁平化が進んでいる場合には技術的に困難となるが、それのみでは適応外とはしていない[10]。腫瘍により椎体後面が破壊されている症例では、脊柱管内へのセメント漏出あるいは腫瘍の突出による脊髄圧迫の発生が危惧される。しかし、これまでのわれわれの経験では、骨外へのセメント漏出は骨皮質破壊部からの直接漏出ではなく椎体静脈を介した漏出が多く[11]、椎体後面の軽度の破壊がただちに脊柱管内へのセメント漏出に結びつくとはいえない。脊柱管にセメントの漏れを許容できないような狭窄が存在するか否かという判断とともに、セメント漏出時の早期検出が重要である[10]。

PVPの治療手技－CT透視を用いた方法

ここでは、われわれが実践してきたCT透視を用いた方法について簡単に述べる[10]。材料・器具は市販のアクリル性骨セメント製剤、穿刺補助器具、骨セメントデリバリーシステムを用いている（図1）。

①輸液製剤で静脈路を確保しモニタを装着する。罹患骨周囲の皮膚上にマーカを貼付してCT撮影を行い（図2a）、この画像をもとに穿刺部位と経路を決定する（図2b）。穿刺経路は経椎弓根的アプローチを基本とする。

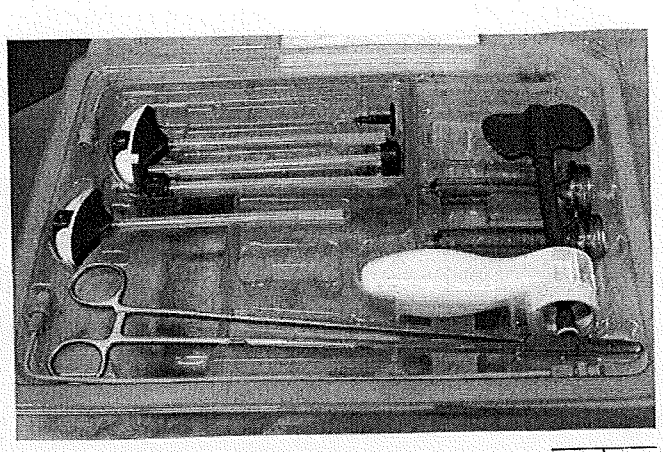

図1a | 図1b

図1　骨セメント器具・材料
a：骨セメント・穿刺補助器具・撹拌ボウル　　b：骨セメントデリバリーシステム

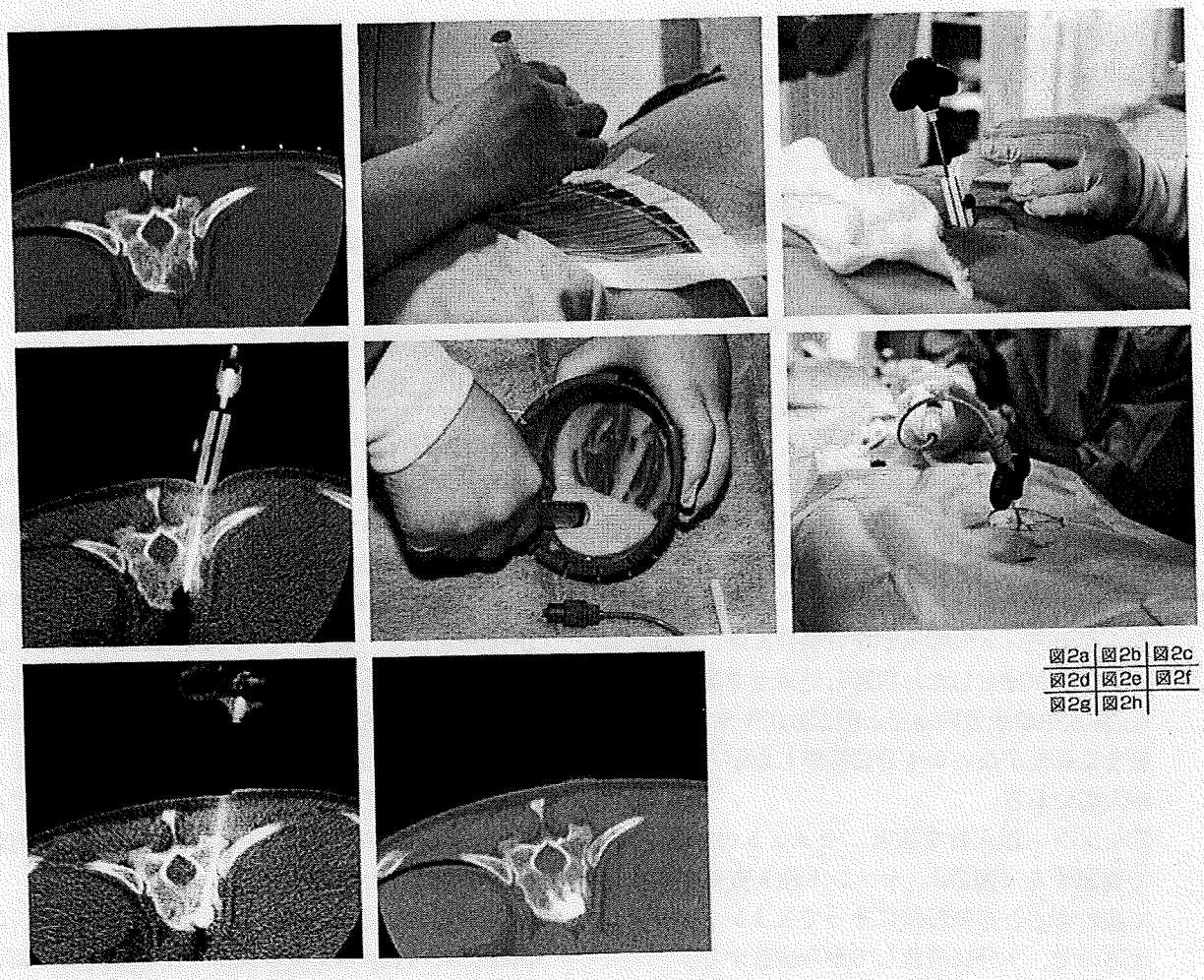

図2a | 図2b | 図2c
図2d | 図2e | 図2f
図2g | 図2h

図2　PVPの治療手技
a：位置決め用CT　　b：マーキング　　c：穿刺
d：穿刺時CT透視画像　　e：セメント調合　　f：セメント注入
g：セメント注入時CT透視画像　　h：セメント注入後CT画像

表2 JIVROSGによる多施設共同第Ⅰ/Ⅱ相臨床試験(JIVROSG-0202)

対象	33症例(有痛性悪性椎体腫瘍)
手技遂行率	100%
有害事象	治療に関連するGrade 3以上の有害事象なし
臨床的有効性 (day7のVAS値での評価)	著効61%、有効12%、無効27%
治療効果出現までの期間	平均2.4日、中央値1日

(2007年6月 結果発表)

②消毒、局所麻酔を行う。
③I-Iデバイスでセメント注入針を保持し、CT透視下に経路にそって用手的に進める(図2c、d)。針が椎弓根を過ぎた後は、基本的には椎体の前方1/3～1/4の部位まで注入針を進める。
④骨セメントの調製を行う(図2e)。CT透視を用いる場合は骨セメント製剤単独でも良好に視認できるため、バリウム製剤の添加は行っていない。骨セメントをインジェクタバレルに充填し、注入針、チューブ、インジェクタを接続すれば注入の準備が完了する。
⑤CT透視下に骨セメントを注入する(図2f、g)。CT断面を頭尾方向に動かしながら透視を行い、ゆっくり注入する。脊柱管内や骨外に骨セメントの漏出が確認された場合にはただちに注入を止める。骨セメントの漏出がない場合には、十分に骨セメントが分布したと判断した時点で注入を止める。注入中は患者の血圧と酸素分圧を常に監視し、患者に異変がないか十分に観察しながら行うことが大切である。
⑥セメント注入針を抜き、セメントの分布をCTで確認する(図2h)。セメント注入直後にあわてて抜針すると穿刺経路にそってセメントがリークするので、十分に硬化してから抜針する。
⑦患者は背臥位で術後2時間の安静とする。

CT透視を用いて行う利点は、針と椎体の関係を瞬時に明確に確認できること、骨セメントの視認性がよいためにセメント製剤にバリウム製剤を添加する必要がないこと、少量のセメント漏出を早期に検出できることが挙げられる。

手技中は患者のモニタリングを欠かさず、緊急時にはすぐに適切な処置が行える体制を整えておく必要がある。われわれは全例で麻酔科医による患者管理のもとに手技を行っている。

有痛性骨腫瘍に対するPVPの治療成績と合併症

本療法の安全性と有効性についての前向きな臨床試験は世界的にも報告がなかったが、ようやく本邦で日本腫瘍IVRグループによる多施設共同第Ⅰ/Ⅱ相臨床試験(JIVROSG-0202)が椎体病巣をもつ33症例を対象に行われ、2007年6月に結果が発表された(表2)[12]。手技遂行率100%で治療に関連するGrade 3以上の有害事象はなく、術後1週間目のVAS値で評価された臨床的有効性は著効61%、有効12%、無効27%、治療効果出現までの期間が平均2.4日、中央値1日というものであった。この試験結果により、一定の安全性と有効性が示されたと考えられる。

これまでに報告された重篤な合併症としては、同時に7、11椎体を全身麻酔下で治療した2例の死亡例[13]、骨セメントによる肺動脈塞栓[14]、脊柱管内の骨セメント漏出による脊髄神経障害などがあるが、このような重篤な合併症はまれである。

PVPの今後の展望

現在、本治療法は保険診療として認可されていないが、高い疼痛緩和効果と即効性は明らかであり、早期の認可により疼痛に苦しむ進行癌患者の有効な治療オプションの1つとなることが望まれる。これまでPVPの長期成績は明らかでなく、中長期の予後が期待される患者には他の治療が優先されてきた。しかし最近、PVPの長期成績が予想以上によいという報告が散見され[15, 16]、長期成績について一定の結論が出されれば、PVPの位置づけがさらに変わる可能性もある。ただし、放射線治療の進歩やストロンチウム-89などの新しい治療法の登場もあり、現在、有痛性骨腫瘍に対する疼痛緩和療法はやや混沌としているのが実情である。

PVPは椎体以外の加重のかかる骨にも応用可能であり、臼蓋、坐骨、恥骨などで高い除痛効果と機能改善が報告されている[17, 18]。さらに胸骨[19]や肋骨などの加重のかからない骨でもその有効性が報告され、適応が拡大しつつある。今後、椎体以外の治療成績の集積とともに除痛機序や生体力学的な基礎的事項の解明も望まれる。

おわりに

PVPの本邦における現況を中心に概説した。正しく行えば安全で有効な治療法であるが、不用意に行えば重篤な合併症を引き起こす可能性もあるので、手技に十分習熟するとともに、救急医、麻酔科医、整形外科医とも連携を密にして施行することが必要である。

参考文献

1) Galibert P et al: Preliminary note on the treatment of vertebral angioma by percutaneous acrylic vertebroplasty. Neurochirurgie 33(2): 166-168, 1987
2) 小林健 ほか: 経皮的椎体形成術における治療効果に影響を与える因子の検討. 臨放 46: 1477-1482, 2001
3) Dean JR et al: The strengthening effect of percutaneous vertebroplasty. Clin Radiol 55(6): 471-476, 2000
4) Deramond H et al: Temperature elevation caused by bone cement polymerization during vertebroplasty. Bone 25(2 Suppl): 17S-21S, 1999
5) Weill A et al: Spinal metastases: indications for and results of percutaneous injection of acrylic surgical cement. Radiology 199(1): 241-247, 1996
6) Garmatis CJ : The effectiveness of radiation therapy in the treatment of bone metastases from breast cancer. Radiology 126(1): 235-237, 1978
7) Salazar OM et al: Single-dose half-body irradiation for palliation of multiple bone metastases from solid tumors. Final Radiation Therapy Oncology Group report. Cancer, 58(1): 29-36, 1986
8) Tomita K et al: Total en bloc spondylectomy for spinal tumors: improvement of the technique and its associated basic background. J Orthop Sci 11(1): 3-12, 2006
9) 山田圭輔 ほか: 転移性椎体腫瘍に対する手術療法と経皮的椎体形成術. ペインクリニック 27: 997-1003, 2006
10) 香田渉 ほか: 椎体腫瘍に対する経皮的椎体形成術. IVR会誌 23: 14-19, 2008
11) 小林健 ほか: CT透視を用いた経皮的椎体形成術の有用性. IVR会誌 14: 343-348, 1999
12) Arai Y et al: Phase I/II study of percutaneous vertebroplasty (PVP) for painful malignant vertebral compression fracture (PMVCF) JIVROSG-0202. Proc. ASCO 25(18S): 522s, 2007
13) Murphy KJ: Percutaneous vertebroplasty in benign and malignant disease. Neuroimaging Clin N Am 10(3): 535-545, 2000
14) Padovani B et al: Pulmonary embolism caused by acrylic cement: a rare complication of percutaneous vertebroplasty. AJNR Am J Neuroradiol 20(3): 375-377, 1999
15) Ramos L et al: Medium-term results of percutaneous vertebroplasty in multiple myeloma. Eur J Haematol 77(1): 7-13, 2006
16) Masala S et al: Percutaneous vertebroplasty in 1,253 levels: results and long-term effectiveness in a single centre. Eur Radiol 19(1): 165-171, 2009
17) Cotten A et al: Malignant acetabular osteolyses: percutaneous injection of acrylic bone cement. Radiology 197(1): 307-310, 1995
18) Kelekis A et al: Pelvic osteoplasty in osteolytic metastases: technical approach under fluoroscopic guidance and early clinical results. J Vasc Intervent Radiol 16(1): 81-88, 2005
19) Zhou B et al: Percutaneous osteoplasty for painful sternal lesion from multiple myeloma. Skeletal Radiol 38(3): 281-285, 2009

Prophylactic Vertebroplasty:

Cement Injection into Non-fractured Vertebral Bodies During Percutaneous Vertebroplasty[1]

Nobuo Kobayashi, MD, Yuji Numaguchi, MD, PhD, Sokun Fuwa, MD, Akihiro Uemura, MD, PhD
Masaki Matsusako, MD, PhD, Yuka Okajima, MD, Mitsutomi Ishiyama, MD, Osamu Takahashi, MD, MPH

Rationale and Objectives. We investigated the efficacy of prophylactic cement injection into the vertebral body adjacent to fractured vertebra to prevent new fractures after percutaneous vertebroplasty (PV).

Materials and Methods. Between February 2002 to August 2004, PV was performed for osteoporotic compression fractures in 89 consecutive patients. All patients underwent PV for only fractured vertebrae. Between September 2004 and October 2006, we performed prophylactic cement injection for 155 patients, with cement injected into the non-fractured vertebra adjacent to the fractured vertebra, immediately above the fractured vertebra in the same procedure. We evaluated the frequency of new vertebral fractures and the efficacy of prophylactic therapy.

Results. In the non-prophylactic group, 15 of 89 patients (16.8%) developed new fractures within 3 months and 20 of 89 patients (22.4%) developed new painful compression fractures within a year after the first PV. These fractures occurred mostly in adjacent vertebra, particularly in the vertebra immediately superior to the treated one and occurred in the lower thoracic and upper lumbar spine. In the prophylactic group, 7 of 155 patients (4.5%) developed new compression fractures within 3 months and 15 of 155 patients (9.7%) developed new compression fractures within 1 year. Statistical analysis showed that fewer new fractures developed in the prophylactic group than in the non-prophylactic group at both 3 months ($P = .0020$, Fisher's exact test) and 1 year ($P = .0079$).

Conclusions. Prophylactic cement injection into non-fractured vertebrae adjacent to fractured vertebrae may prevent new compression fractures after vertebroplasty for osteoporotic patients.

Key Words. Vertebroplasty; prophylactic; vertebral compression fracture.

© AUR, 2009

The first report of percutaneous vertebroplasty (PV) was published in 1987 by Galibert et al, who treated a patient with a giant angioma in the second cervical vertebra (1). Ever since, PV has been performed all over the world for tumor or osteoporosis-related compression fractures, particularly spinal compression fractures with difficult pain control. Many studies have demonstrated the therapeutic effects and complications of PV (2–8). In particular, PV is a highly effective and quick-responding measure against acute and subacute spinal compression fractures, and is effective in relieving pain from prolonged therapy due to pseudoarthrosis (9,10).

Even if PV improves the activities of daily living and sufficiently relieves pain, new spinal compression fractures often occur soon after therapy, causing back pain to recur. Because of recurrent painful compression fractures, second or multiple treatments are sometimes needed (11–14).

Acad Radiol 2009; 16:136–143

[1] From the Departments of Radiology (N.K., Y.N., S.F., A.U., M.M., Y.O., M.I.) and Internal Medicine (O.T.), St. Luke's International Hospital, Akashi-cho 9-1, Chuo-ku, Tokyo, 104-8560 Japan. Received March 31, 2008; accepted May 3, 2008. Presented in abstract form at the Annual Meeting of the Radiological Society of North America, November 27, 2006, Chicago, IL. **Address correspondence to:** N.K. e-mail: nobkob@luke.or.jp

© AUR, 2009
doi:10.1016/j.acra.2008.05.005

Although new spinal compression fractures are believed to be mostly caused by the natural course of osteoporosis (15), PV itself may be the cause (16). In particular, involvement of PV is suspected in new compression fractures occurring in the vertebrae adjacent to treated vertebrae soon after therapy (17). To lower the risk of new compression fractures, we performed PV for painful fractured vertebrae and then prophylactically injected bone cement into adjacent vertebrae at risk for fracture. Although accurate prediction is difficult, new vertebral fractures often occur on the adjacent vertebra in nearly half of the cases (18,19). We hypothesize that the risk of recurrence might be lowered by performing prophylactic PV to the adjacent vertebra.

The objective of the present study was to confirm whether non-fractured adjacent vertebrae should be treated as a prophylactic measure during the first PV treatment for painful osteoporotic compression fractures.

MATERIALS AND METHODS

Subjects

The present study was approved by the institutional review board. A written informed consent was obtained from all patients after thorough explanation of the procedure, including prophylactic treatment on non-fractured vertebral body adjacent to the fractured vertebra. Patients in Group 1, who were treated between February 2002 and August 2004, consisted of 89 patients with osteoporotic spinal compression fractures (78 women, 11 men; mean age 70.2 years; range 38–98 years). During this period, conventional PV was performed to treat pain-causing vertebrae based on magnetic resonance imaging (MRI) and computed tomography (CT). These 89 patients underwent 89 sessions to treat 143 spinal compression fractures. Mean number of treated vertebrae per surgery was 1.6 (range 1–5).

Patients in Group 2, between September 2004 and October 2006, consisted of 155 patients who had treatment of the pain-causing vertebrae and also of the adjacent vertebrae at risk for secondary fractures. In Group 2, the majority of prophylactically treated vertebrae were located between Th10 and L2. These 155 patients underwent 155 sessions to treat 264 spinal compression fractures and to perform prophylactic cement injection into 198 non-fractured vertebrae. Mean number of treated fractured vertebrae per surgery was 1.7 (range 1–5), and mean number of treated vertebrae including prophylactic non-fractured vertebrae was 2.9 (range 2–6). All subjects in the present study had osteoporosis, including steroid-induced osteoporosis. The group without prophylactic therapy (Group 1) and the group with prophylactic therapy (Group 2) were compared to ascertain whether prophylactic therapy could reduce the risk of new fractures based on statistical analysis of recurrence rates.

PV Procedures

PV was performed under local anesthesia in all patients. As a general rule, 11-gauge Osteosite bone biopsy needles (Cook, Bloomington, IN) were used. When treating vertebrae with small pedicles or crushed and flattened vertebrae, 13-gauge needles were used in some cases. As to stylets (internal needles), diamond-cut stylets were predominantly used, and when minor adjustments were needed in the direction of puncture, bevel-cut stylets were used. As a general rule, PV was performed using two needles placed through the left and right pedicles to achieve adequate cement filling in the vertebra. As bone cement, a liquid monomer (17 mL) and 30 g of polymethylmethacrylate powder (Cranioplastic kit; Codman, Raynham, MA) were mixed. To ensure radiopacity, sterile powdered barium (9 g, Neobargin HD; Kyosei Pharmaceutical, Otaru, Japan) was included (30% weight/volume). The cement was manually injected with 1-mL syringes under biplane fluoroscopic observation. However, the Osteoject bone cement delivery system (Integra, Plainsboro, NJ) was also used in some patients.

Cement injection was performed under the guidance of biplane x-ray fluoroscopy, and blood pressure, electrocardiogram, and arterial oxygen saturation were monitored during surgery.

Analysis

Preoperative imaging assessment consisted of plain thoracolumbar imaging, MRI (T1-weighted, fat-suppressed, T2-weighted and gadolinium-enhanced imaging) and 16-slice CT (3-mm reconstructed axial, sagittal, and coronal scans). Immediately after surgery, plain radiography was performed to confirm cement distribution. Subsequent observations mostly comprised clinical symptom analysis and plain radiography during periodic outpatient visits.

When clinical symptoms suggested a new fracture, MRI and CT were performed in all cases. Follow-up visits were made at 1 day and at 1, 3, 6, and 12 months after surgery. Thereafter, patients were followed by telephone interviews. If recurrence was suspected, plain radi-

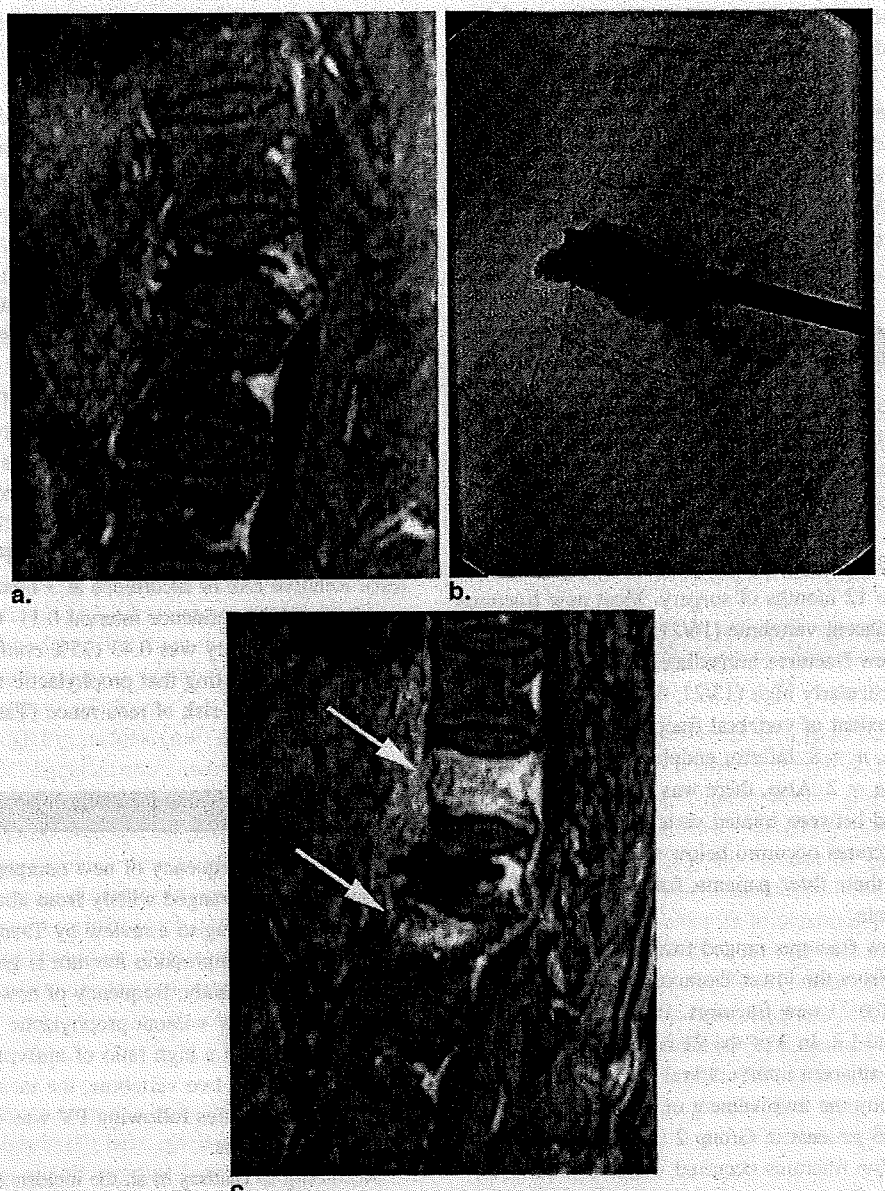

Figure 1. **(a)** Fat-suppressed contrast-enhanced T1-weighted sagittal image of a 78-year-old woman with osteoporotic compression fracture of the Th12 vertebra. **(b)** Cement was injected into the fractured vertebra. Fat-suppressed contrast-enhanced T1-weighted sagittal image. **(c)** Three weeks after percutaneous vertebroplasty, a new fracture developed in Th11 and L1 vertebrae. Bone marrow edema is seen throughout the entire vertebral body of Th11 and in the superior endplate of L1 (*white arrows*).

ography, MRI, and CT were performed. When disagreement existed in imaging assessment between surgeons and radiologists interpreting scans, a conference was held to reevaluate the scans.

Between groups with and without prophylactic therapy, the frequency of new postoperative compression fracture was compared, and statistical analysis (Fisher's exact test) was performed by a third party. As for a primary end

point, the presence or absence of new fracture was determined at 3 and 12 months after surgery. As for a secondary end point, the location of new fractures in relation to treated vertebrae, presence or absence of bone cement leakage into adjacent intervertebral disc spaces, and the frequency of compression fracture in adjacent vertebrae among patients with bone cement leakage into the intervertebral disc space were assessed.

RESULTS

In both Groups 1 and 2, no intraoperative complications were seen besides bone cement leakage into the intervertebral disc space in a limited number of patients.

Of the 89 patients in Group 1, new compression fractures occurred within 3 months of surgery in 15 patients (16.8%), and 21 new fractures occurred in 20 patients (22.4%) within 12 months of surgery. Most new fractures occurred in adjacent vertebrae (16/21, 76.1%) (Fig 1). The ratio of new fractures immediately above treated vertebrae was particularly high (13/21, 61.9%). For the 13 fractures, the extent of vertebral fracture was as follows: entire vertebra, $n = 8$; inferior endplate, $n = 3$; and superior endplate, $n = 2$. Also, there was one new vertebral fracture located between treated vertebrae. In three patients, new fractures occurred below treated vertebrae; and in two of these three patients, fracture occurred in the superior endplate.

Level of new fractures ranged from Th7 to L4, mostly concentrating from the lower thoracic to upper lumbar vertebrae. Of the 21 new fractures, 16 fractures were between Th11 and L2. In 2 of the 21 new fractures, cement leakage to the adjacent intervertebral disc space was noted, suggesting the involvement of cement leakage. Among the 155 patients in Group 2 (as seen in Fig 2), new compression fractures occurred within 3 months of surgery in seven patients (4.5%). Within 12 months of surgery, 17 new fractures occurred in 15 patients (9.7%). New fractures occurred between Th8 and L5.

Of the 17 fractures in the 15 patients, six fractures (35.3%) occurred immediately above treated vertebrae, and six more fractures occurred immediately below the treated vertebrae. Among the six vertebral fractures above the treated vertebrae, the extent of fracture was as follows: entire vertebra, $n = 2$; inferior endplate, $n = 2$; and superior endplate, $n = 2$. Among the six vertebral fractures below the treated vertebrae, the extent of fracture was as follows: entire vertebra, $n = 4$; and superior endplate, $n = 2$.

Including one vertebra sandwiched by treated vertebrae, 13 fractures occurred in adjacent vertebrae, but two fractures occurred in non-adjacent vertebrae (both Th8). Also, among non-fractured vertebrae treated by prophylactic cement injection, new painful fractures occurred in the superior endplate just above the injected cement within the same vertebra in two cases. In 2 of the 17 new fractures, cement leakage into the adjacent intervertebral disc space was seen, suggesting the involvement of cement leakage.

Statistical analysis showed that risk of recurrence was significantly lower for the group with prophylactic therapy than for the group without prophylactic therapy. A significant difference in rate of recurrence was seen at 3 months after surgery ($P = .0020$, Fisher's exact test) and at 12 months after surgery ($P = .0079$, Fisher's exact test). Relative risk of recurrence at 3 months after surgery was 0.27 (95% confidence interval 0.11–0.63) and at 12 months after surgery was 0.43 (95% confidence interval 0.23–0.80), suggesting that prophylactic therapy significantly lowered the risk of recurrence (Table 1).

DISCUSSION

The reported frequency of new compression fractures following PV has ranged widely from about 10% to 50% (7,12), but according to a review by Trout et al, the frequency of new compression fracture is generally 20% to 25% (16). In our study, frequency of new fracture within 12 months after PV without prophylactic therapy was 22.4%, and despite a high ratio of multiple fractures involving more than two vertebrae, the incidence of new compression fractures following PV was comparable to the previous studies.

According to Lindsay et al, the incidence of another new compression fracture within 12 months of the initial fracture was 19.2% among patients with a history of spinal compression fracture and 24% among patients with a history involving more than two vertebrae (15). Syed et al. deduced that new compression fracture following PV is part of the natural course of osteoporosis (12). In a review of 432 PV cases, Trout et al (17) reported that new compression fractures occurred in adjacent vertebrae after PV in 41.4% of cases. They suggested that a new compression fracture occurring soon after surgery in the vertebra adjacent to the treated one is likely due to PV itself (17).

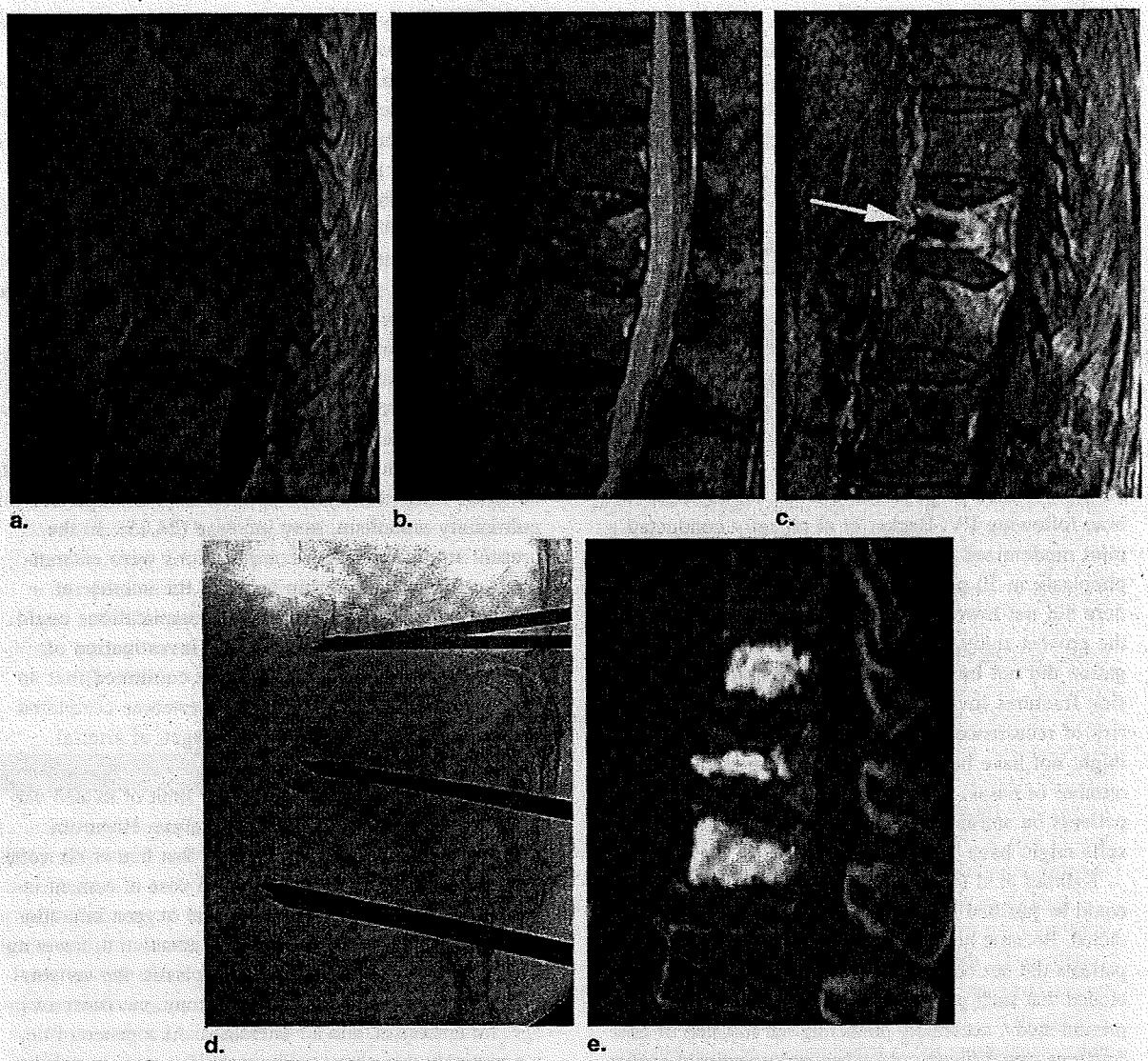

Figure 2. **(a)** T1-weighted, **(b)** fat-suppressed T2-weighted, and **(c)** fat-suppressed contrast-enhanced T1-weighted sagittal images of a 79-year-old man with osteoporotic compression fractures of L1 and L2 vertebrae. There is an unenhanced portion in L1 vertebral body indicating a cleft (*white arrow*). **(d)** Lateral fluoroscopic image showing 11-gauge needles inserted into the fractured vertebrae (L1, L2) and into a non-fractured vertebra (Th12) as prophylactic treatment. **(e)** Sagittal reconstructed computed tomography showing cement in the fractured vertebrae (L1, L2) and in the upper adjacent non-fractured vertebra (Th12).

In the present study, the group of patients without prophylactic treatment had new compression fractures occur in adjacent vertebrae in 16 of 21 (76.1%) fractures and 15 of the 20 patients (75.0%) with new fractures had the new fractures occur within three months of surgery. Because these fractures frequently occurred adjacent to the treated vertebrae, involvement of PV is suggested. Vertebral compression fractures tend to occur in the superior endplate. According to Baroud et al and other authors, analysis of new compression fractures often shows fractures above the treated vertebrae, and the incidence of fractures in the inferior endplate of the upper vertebrae is relatively high (18,19). In the present study, new compression fractures after PV often occurred in the upper adjacent vertebrae and, in such cases, fractures most often affected the entire vertebra or mostly the inferior end-

Table 1
Statistical Analysis (Fisher's exact test)

	New Fracture Within 3 Months (n = 244)	New Fracture Within a Year (n = 244)
Non-prophylactic group	15/89 (16.8%)	20/89 (22.4%)
Prophylactic group	7/155 (4.5%)	15/155 (9.7%)
P value	.0020	.0079
Relative risk	0.27 (0.11–0.63)	0.43 (0.23–0.80)

plate. Conversely, new fractures in the lower adjacent vertebrae often affected the superior endplate. This agrees with normal fracture patterns, and the effects of PV are unclear.

To the best of our knowledge, no previous authors have performed prophylactic therapy on adjacent vertebrae following PV. Becker et al recently conducted a pilot randomized controlled trial for prophylactic kyphoplasty in 50 patients and concluded that the procedure did not lower the risk of recurrence (20). Unlike the present study, because Becker et al's (20) investigation did not include patients with multiple compression fractures involving more than two vertebrae, the risk of recurrence was low and significant differences might not have been identified because of the low number of cases. If patients with multiple fractures or patients on steroid therapy had been included, the results might have been different.

Kallmes et al (4) mentioned that prophylactic therapy could be justified if the vertebrae at risk could be predicted. Because half of the secondary fractures in their patients did not occur in adjacent vertebrae, they concluded that such prediction was difficult (4). In the present study, accurately predicting the location of new fractures was difficult even when retrospectively analyzing preoperative MRI and CT. However, because the intervertebral disc adjacent to the superior endplate of a fractured vertebra is often degenerated from a decreased buffer action of the cartilaginous component, the risk of new fracture in an upper adjacent vertebra will be increased (21–23). For this reason, the prophylactic cement injection into the vertebra immediately above the fractured vertebra may be justified.

As shown in the Results section, the prophylactic therapy on the upper adjacent vertebrae of fractured vertebrae significantly lowered the frequency of subsequent compression fractures in our patients. The incidence of new fractures in lower adjacent vertebrae was relatively low; therefore, prophylactic therapy for these vertebrae may be unnecessary. However, prophylactic therapy for lower adjacent vertebra may be justified if the disc space immediately below the fractured vertebra is narrowed and the two vertebrae are closely related.

In Japan, PV is not covered by insurance and, as a general rule, patients pay 100% of the medical costs for the treatment. PV is thus an expensive treatment, and additional treatments incur substantial financial burdens for aged patients. If prophylactic therapy lowers the incidence of additional therapy, and if PV becomes covered by insurance in the future, the burden on the insurance system may also be lowered.

Adverse effects are the major concern when we perform multiple-level PV including prophylactic treatment. If the number of treated vertebrae increases, risk of latent complications, such as cement leakage and pulmonary embolism, may increase (24,25). In the present study, no severe complications were encountered, and the relationship between the number of treated vertebrae and the risk for complications could not be clarified; however, our past investigation of changes in alveolar partial pressure confirmed that an increase in the number of treated vertebrae correlated to decreased partial pressure of oxygen in arterial blood (26).

At present, we have set the upper limit of treated vertebrae per single session at four vertebrae. However, based on our experience, we believe that five or six vertebrae can be safely treated if the total dose of cement injection is less than 30 mL and arterial oxygen saturation is carefully monitored. We also pay attention to lowering such risks as minor cement leakage outside the vertebral body that cannot be seen by fluoroscopy, monomer seepage, fat embolism, and air embolism. As a general rule, we routinely use a two-needle technique even in prophylactic therapy for a non-fractured vertebra. While injecting cement from one side, the contralateral needle stylet is removed to lessen excessive increases in vertebral pressure during cement injection, thus preventing intravenous seepage of cement and vertebral fat tissue or cement leakage into the intervertebral disc space. We also try to reduce the risk of cement leakage by performing preoperative three-dimensional CT multiplanar reconstruction to thoroughly examine ruptured cortical bone and vertebral morphology.

In the event of bone cement leakage into the intervertebral disc space, the intervertebral disc may be in a pathologic state, and this may be one of the rationales for

prophylactic therapy of adjacent vertebrae. Prophylactic therapy may also be justified if preoperative diagnostic imaging shows ruptured cortical bone, leading to a high risk of bone cement leakage into the intervertebral disc space (27). Because the number of patients with intervertebral disc leakage was low in our study, we could not investigate the relationships between intervertebral leakage and secondary fracture.

One study reported that PV using a small amount of cement did not induce secondary fracture (28,29), but in some of our patients, use of a small amount of cement caused painful fracture above the site of cement injection within the same vertebra or new fracture between uneven left and right cement masses. Therefore, as mentioned previously, we use two needles to treat a fractured vertebra and perform prophylactic therapy while paying attention to evenly distributing cement inside the vertebra.

As for study limitations, the present study was not a prospective randomized trial. The group without prophylactic therapy was studied first, followed by the group with prophylactic therapy. Given this historical cohort design, improved proficiency and skills could have influenced assessments. Also, the number of treated vertebrae ranged greatly from one to six vertebrae. Furthermore, because risk of recurrence is particularly high for patients on long-term steroid therapy (30–32), exclusion of such patients might have made the patient groups more homogeneous. In the future, randomized control trials investigating the usefulness of PV and the efficacy of prophylactic therapy are needed.

CONCLUSION

Prophylactic cement injection into non-fractured vertebrae adjacent to fractured vertebrae may prevent new compression fractures after vertebroplasty of osteoporotic patients.

ACKNOWLEDGMENTS

We are grateful to Dr. Henry Wang for editing this manuscript.

REFERENCES

1. Galibert P, Deramond H, Rosat P, et al. Preliminary note on the treatment of vertebral angioma by percutaneous acrylic vertebroplasty [in French]. Neurochirurgie 1987; 33:166–168.
2. Lapras C, Dusquenel J. [Percutaneous injection of methylmethacrylate in osteoporosis and severe vertebral osteolysis (Galibert's technique)]. Ann Chir 1989; 43:371–376.
3. Heini PF, Walchli B, Berlemann U. Percutaneous transpedicular vertebroplasty with PMMA: operative technique and early results. A prospective study for the treatment of osteoporotic compression fractures. Eur Spine J 2000; 9:445–450.
4. Kallmes DF, Jensen ME. Percutaneous vertebroplasty. Radiology 2003; 229:27–36.
5. Evans AJ, Jensen ME, Kip KE, et al. Vertebral compression fractures: pain reduction and improvement in functional mobility after percutaneous polymethylmethacrylate vertebroplasty—retrospective report of 245 case. Radiology 2003; 226:366–372.
6. Mckiernan F, Faciszewski T, Jensen R. Quality of life following vertebroplasty. J Bone Joint Surg Am 2004; 86:2600–2606.
7. Grados F, Depriester C, Cayrolle G, et al. Long-term observations of vertebral osteoporotic fractures treated by percutaneous vertebroplasty. Rheumatology (Oxford) 2000; 39:1410–1414.
8. Layton KF, Thielen KR, Koch CA, et al. Vertebroplasty, first 1000 levels of a single center: evaluation of the outcomes and complications. AJNR Am J Neuroradiol 2007; 28:683–689.
9. Brown DB, Gilula LA, Sehgal M, et al. Treatment of chronic symptomatic vertebral compression fractures with percutaneous vertebroplasty. AJR Am J Roentgenol 2004; 182:319–322.
10. Irani FG, Morales JP, Sabharwal T, et al. Successful treatment of a chronic post-traumatic 5-year-old osteoporotic vertebral compression fracture by percutaneous vertebroplasty. Br J Radiol 2005; 78: 261–264.
11. Uppin AA, Hirsch JA, Centenera LV, et al. Occurrence of new vertebral body fracture after percutaneous vertebroplasty in patients with osteoporosis. Radiology 2003; 226:119–124.
12. Syed MI, Patel NA, Jan S, et al. New symptomatic vertebral compression fractures within a year following vertebroplasty in osteoporotic women. AJNR Am J Neuroradiol 2005; 26:1601–1604.
13. Tanigawa N, Komemushi A, Kariya S, et al. Radiological follow-up of new compression fractures following percutaneous vertebroplasty. Cardiovasc Intervent Radiol 2006; 29:92–96.
14. Lin CC, Chen IH, Yu TC, et al. New symptomatic compression fracture after percutaneous vertebroplasty at the thoracolumbar junction. AJNR Am J Neuroradiol 2007; 28:1042–1045.
15. Lindsay R, Silverman SL, Cooper C, et al. Risk of new vertebral fracture in the year following a fracture. JAMA 2001; 285:320–323.
16. Trout AT, Kallmes AF. Does vertebroplasty cause incident vertebral fractures? A review of available data. AJNR Am J Neuroradiol 2006; 27:1397–1403.
17. Trout AT, Kallmes DF, Kaufmann TJ. New fractures after vertebroplasty: adjacent fractures occur significantly sooner. AJNR Am J Neuroradiol 2006; 27:217–223.
18. Baroud G, Heini P, Nemes J, et al. Biomechanical explanation of adjacent fractures following vertebroplasty. Radiology 2003; 229:606–607.
19. Trout AT, Kallmes DF, Layton KF, et al. Vertebral endplate fractures: an indicator of the abnormal forces generated in the spine after vertebroplasty. J Bone Miner Res 2006; 21:1797–1802.
20. Becker S, Garoscio M, Meissner J, et al. Is there an indication for prophylactic balloon kyphoplasty? A pilot study. Clin Orthop Relat Res 2007; 458:83–89.
21. Baroud G, Nemes J, Heini P, et al. Load shift of the intervertebral disc after a vertebroplasty: a finite-element study. Eur Spine J 2003; 12:421–426.
22. Lunt M, O'Neill TW, Felsenberg D, et al. Characteristics of a prevalent vertebral deformity predict subsequent vertebral fracture: results from the European Prospective Osteoporosis Study (EPOS). Bone 2003; 33: 505–513.
23. Kim SH, Kang HS, Choi JA, et al. Risk factors of new compression fractures in adjacent vertebrae after percutaneous vertebroplasty. Acta Radiol 2004; 45:440–445.
24. Stricker K, Orler R, Yen K, et al. Severe hypercapnia due to pulmonary embolism of polymethylmethacrylate during vertebroplasty. Anesth Analg 2004; 98:1184–1186.

25. Syed MI, Jan S, Patel NA, et al. Fatal fat embolism after vertebroplasty: identification of the high-risk patient. AJNR Am J Neuroradiol 2006; 27:343–345.
26. Uemura A, Numaguchi Y, Matsusako M, et al. Effect on partial pressure of oxygen in arterial blood in percutaneous vertebroplasty. AJNR Am J Neuroradiol 2007; 28:567–569.
27. Lin EP, Ekholm S, Hiwatashi A, et al. Vertebroplasty: cement leakage into the disc increases the risk of new fracture of adjacent vertebral body. AJNR Am J Neuroradiol 2004; 25:175–180.
28. Liebschner MA, Rosenberg WS, Keaveny TM. Effects of bone cement volume and distribution on vertebral stiffness after vertebroplasty. Spine 2001; 26:1547–1554.
29. Hodler J, Peck D, Gilula LA. Midterm outcome after vertebroplasty: predictive value of technical and patient-related factors. Radiology 2003; 227:662–668.
30. Mathis JM, Petri M, Naff N. Percutaneous vertebroplasty treatment of steroid-induced osteoporotic compression fractures. Arthritis Rheum 1998; 41:171–175.
31. Koch CA, Layton KF, Kallmes DF. Outcome of patients receiving long-term corticosteroid therapy who undergo percutaneous vertebroplasty. AJNR Am J Neuroradiol 2007; 28:563–566.
32. Hiwatashi A, Westesson PL. Patients with osteoporosis on steroid medication tend to sustain subsequent fractures. AJNR Am J Neuroradiol 2007; 28:1055–1057.

ORIGINAL RESEARCH

Pedicle Involvement on MR Imaging Is Common in Osteoporotic Compression Fractures

M. Ishiyama
S. Fuwa
Y. Numaguchi
N. Kobayashi
Y. Saida

BACKGROUND AND PURPOSE: Pedicle involvement on MR imaging has been considered specific for malignancy. However, we also noted the findings in many patients with osteoporosis and hypothesized that it is not specific for malignant lesions. The aim of this study was to evaluate the prevalence of pedicle involvement in painful osteoporotic compression fractures and to determine whether the sign is specific for malignancy.

MATERIALS AND METHODS: We retrospectively reviewed MR images and CT scans of 152 patients who underwent PV for painful compression fractures. There were 140 patients (225 vertebrae) with osteoporotic fractures and 12 patients (19 vertebrae) with malignant fractures. Three radiologists evaluated the degree and extent of signal-intensity changes of the pedicle on MR imaging by consensus. The CT findings were also evaluated. The χ^2 test was used for statistical analyses.

RESULTS: Of the 225 vertebrae of osteoporotic fractures and 19 vertebrae of malignant fractures, pedicle involvement on MR imaging was seen in 144 (64%) and 16 (84.2%) vertebrae, respectively, and there was no statistically significant difference ($P = .065$). Positive pedicle involvement in osteoporotic fractures was seen in 84 (77%) of 109 vertebrae with early-phase fractures (≤3 months) and 60 (51.7%) of 116 vertebrae with chronic-phase fractures (>3 months), and this was statistically significant ($P < .001$). Among 144 osteoporotic vertebrae that showed positive pedicle involvement on MR imaging, 45 (31%) showed pedicle fractures and 55 (38.2%) showed sclerotic change on CT.

CONCLUSIONS: Pedicle involvement was seen frequently in patients with osteoporotic compression fractures and was not specific for malignancy in our study group.

ABBREVIATIONS: Gd-T1WI = gadolinium-enhanced T1WI; PV = percutaneous vertebroplasty; STIR = short-tau inversion recovery; T1WI = T1-weighted imaging; T2WI = T2-weighted imaging

Differentiation of benign osteoporotic compression fractures and malignant pathologic fractures is clinically important, particularly in the elderly, but is sometimes difficult. MR imaging and CT have been shown to be helpful in differentiating both conditions. Compression fractures due to malignant tumors have a convex posterior cortex of the vertebral bodies, epidural or paravertebral masses, or infiltration of the posterior elements.[1-5] Of these, pedicle involvement has been described as specific for malignant lesions.[3] Osteoporotic compression fractures usually show retropulsion of a posterior bone fragment or intravertebral fluid.[2,3,5,6]

In our experience of PV for painful compression fractures, abnormal signal-intensity change in the pedicle on MR imaging, which will be defined as "pedicle involvement," has commonly been noted not only in patients with malignant compression fractures but also in those with benign osteoporotic compression fractures, and differentiation by using only this sign is sometimes difficult.

The aim of this study was to evaluate the prevalence and features of pedicle-involvement change on MR imaging in painful osteoporotic compression fractures and to determine whether these findings are truly specific for malignancy.

Received August 4, 2009; accepted August 24.

From the Department of Radiology, St. Luke's International Hospital, Tokyo, Japan.

Please address correspondence to Mitsutomi Ishiyama, MD, Department of Radiology, St. Luke's International Hospital, 9-1 Akashi-Cho, Chuo-Ku, Tokyo, Japan; e-mail: mitishi@luke.or.jp

DOI 10.3174/ajnr.A1905

Materials and Methods

All protocols for this retrospective Health Insurance Portability and Accountability Act–compliant study were approved by the institutional review board. The need for informed consent was waived.

Subjects

We retrospectively reviewed medical records and MR imaging/CT data from 200 consecutive patients who had undergone PV for painful osteoporotic or malignant vertebral fractures. Patients who underwent full imaging studies (plain CT, T1WI, STIR imaging, and Gd-T1WI) were included. Exclusion criteria were the following: an insufficient imaging study, traumatic compression fracture without osteoporosis, PV for sacral fractures or Schmorl nodes, and repeat PV for vertebrae that had already undergone PV. The final study group comprised 140 patients with osteoporotic compression fractures (225 vertebrae; 35 women, 105 men; mean age, 77.8 years; range, 55–96 years) and 12 patients with malignant fractures (19 vertebrae; 7 women, 5 men; mean age, 61 years; range, 46–71 years). Diagnosis was primarily made on the basis of medical history, thorough physical examination, laboratory findings, and imaging studies, including plain radiography, CT, and MR imaging. When there were the characteristic findings indicating a benign process such as retropulsion of the posterior bone fragment or intravertebral cleft without bone destruction, the diagnosis was a benign compression fracture.

The final diagnosis of benign compression fracture was made by means of follow-up plain radiography and clinical history at 3 months and 1 year after PV. When the appearance of a follow-up radiographic study did not change significantly and no new malignancy was found, the fracture was considered to be caused by a benign process. Biopsy was performed to rule out malignant lesions for 3 patients with osteoporosis with a history of malignancy. When there was an intra- or

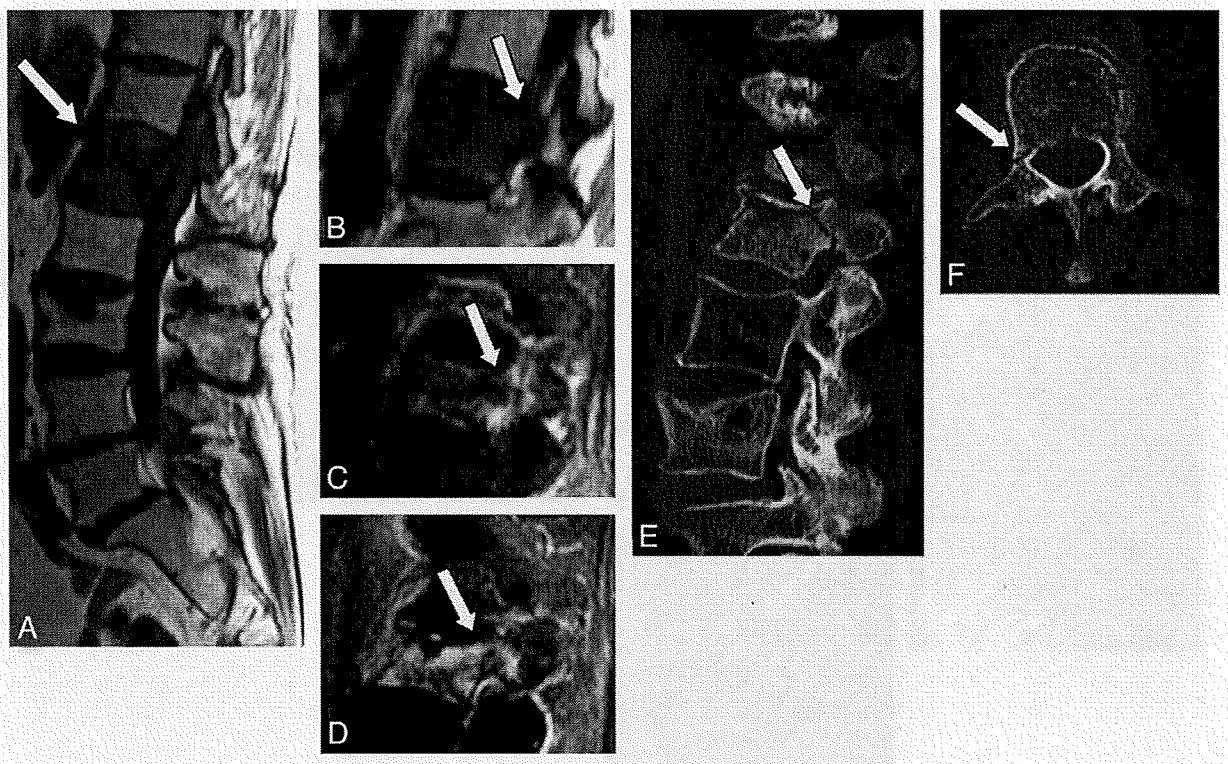

Fig 1. A 79-year-old woman with benign compression fracture which occurred one-and-a-half months earlier. *A* and *B*, T1WI shows diffuse hypointensity in the vertebral body and the right pedicle of L1 (*arrow*). *C*, STIR image shows heterogeneous hyperintensity in these areas (*arrow*). *D*, Contrast-enhanced T1WI shows marked enhancement (*arrow*). *E* and *F*, On sagittal (*E*) and axial (*F*) CT scans, fracture is seen in the right pedicle (*arrow*), which is a presumable reason for the abnormal signal intensity on MR imaging.

paravertebral mass or cortical bone destruction in the fractured vertebra, it was diagnosed as a malignant pathologic fracture. Among the 12 patients with malignancy, 11 patients had metastatic carcinoma (bronchogenic carcinoma, $n = 5$; prostate carcinoma, $n = 2$; breast carcinoma, $n = 1$; vaginal carcinoma, $n = 1$; pancreatic carcinoma, $n = 1$; and cervical carcinoma, $n = 1$), and 1 patient had multiple myeloma.

Imaging

MR imaging of the thoracolumbar spine was performed with 1 of two 1.5T scanners (Signa Excite, GE Healthcare, Milwaukee, Wisconsin; or Intera Achieva, Philips Medical Systems, Best, the Netherlands) by using a spine-array surface coil. The imaging protocol included sagittal spin-echo T1WI (TR/TE = 474–550/10.5–13 ms), STIR images (TR/TE/TI = 2500–6000/70–93/150 ms), and fat-saturated spin-echo Gd-T1WI (TR/TE = 460–600/6.2–12; flip angle = 80°–90°; 0.2 mmol/kg) with a 4-mm section thickness and 0.5-mm section spacing. The matrices were 512 × 306 and 512 × 384 with an FOV of 480 mm or 320 × 224 with an FOV of 280 mm (divided into 2 series). In some cases, axial images were obtained on Gd-T1WI, which were not evaluated in this study.

CT was also performed by using a 64-section scanner or a 16-section scanner. Reconstructed axial, transverse, and sagittal images were obtained with a 3-mm section thickness. The matrix was 512 × 512.

Image Analysis

Three radiologists evaluated the degree and extent of signal-intensity changes of the pedicle on MR imaging by consensus. Signal-intensity changes were classified as iso-, hyper-, or hypointense to the marrow of normal unaffected pedicles in the same patient on T1WI, iso- or hyperintense on STIR images and as showing mild or marked enhancement on Gd-T1WI. Pedicle involvement was defined as positive when at least 2 of 3 sequences showed abnormal signal-intensity change in the pedicle. The extent of the pedicle involvement was classified as partial or diffuse. CT findings of the pedicle were categorized into 3 groups: fracture (Fig 1), sclerosis (Fig 2), or no apparent abnormality (Fig 3).

Statistical Analysis

The χ^2 test was used for statistical analyses, with values of $P < .05$ considered significant.

Results

Pedicle involvement on MR imaging in benign osteoporotic compression fractures and malignant pathologic fractures is shown in Table 1. Of the 225 vertebrae of osteoporotic fractures and 19 vertebrae of malignant pathologic fractures, pedicle involvement was seen in 144 (64%) and 16 (84.2%) vertebrae, respectively. No significant difference ($P = .065$) was apparent. Sensitivity and specificity of pedicle involvement for malignant pathologic fractures were 84% and 36%, respectively. Among osteoporotic fractures ($n = 225$), 85 vertebrae (41.3%) showed signal-intensity changes in both T1WI and STIR images and diffuse contrast enhancement of the pedicle.

The relationship between pedicle involvement on MR imaging and the duration from the onset of pain in patients with osteoporotic compression fracture is given in Table 2. With osteoporotic fractures, pedicle involvement was seen in 84 (77%) of 109 vertebrae with early-phase fractures (≤3

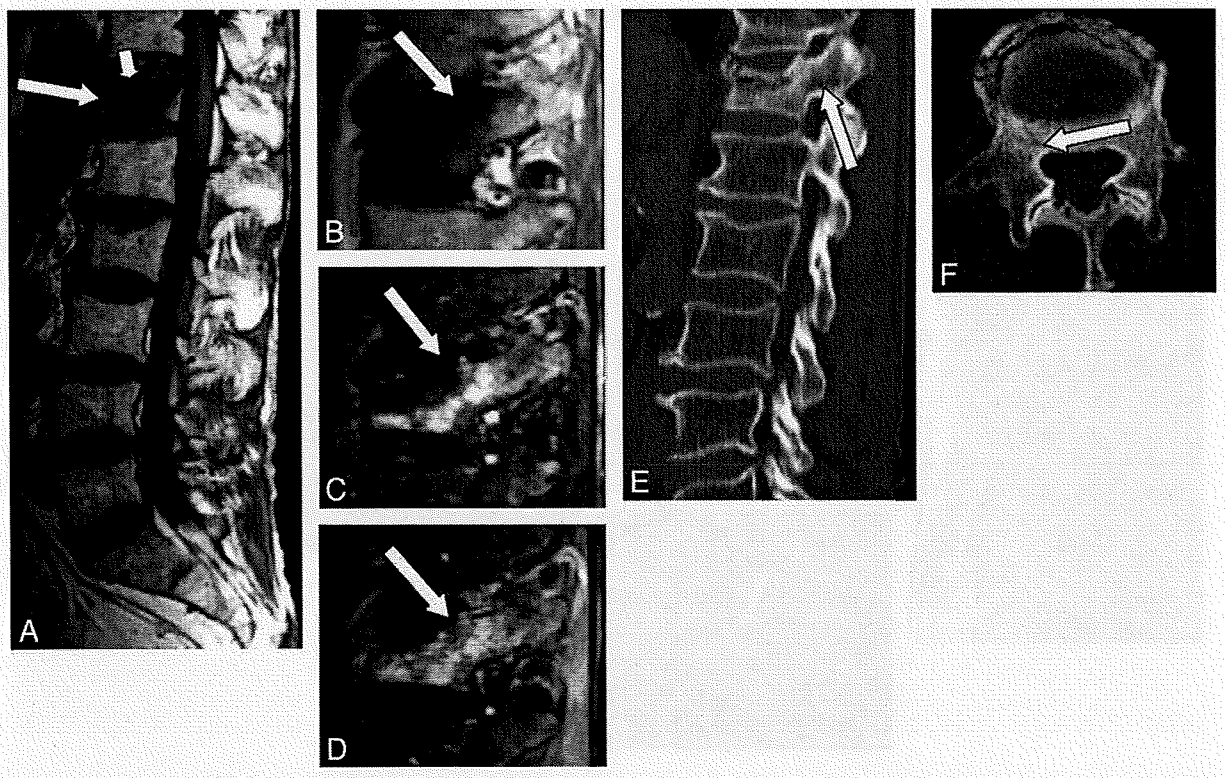

Fig 2. A 65-year-old man with benign compression fracture, which occurred 2 months earlier. *A* and *B*, T1WI shows diffuse hypointensity in the vertebral body and the right pedicle of T12 (*arrow*). There is a marked hypointensity band adjacent to the superior endplate, indicating a gas cleft (*small arrow*). *C*, STIR image shows heterogeneous hyperintensity in these areas (*arrow*). *D*, Contrast-enhanced T1WI shows moderate enhancement in the vertebral body and in the right pedicle (*arrow*). *E* and *F*, On sagittal (*E*) and axial (*F*) CT scans, sclerosis is seen around the gas cleft including the base of the right pedicle (*arrow*).

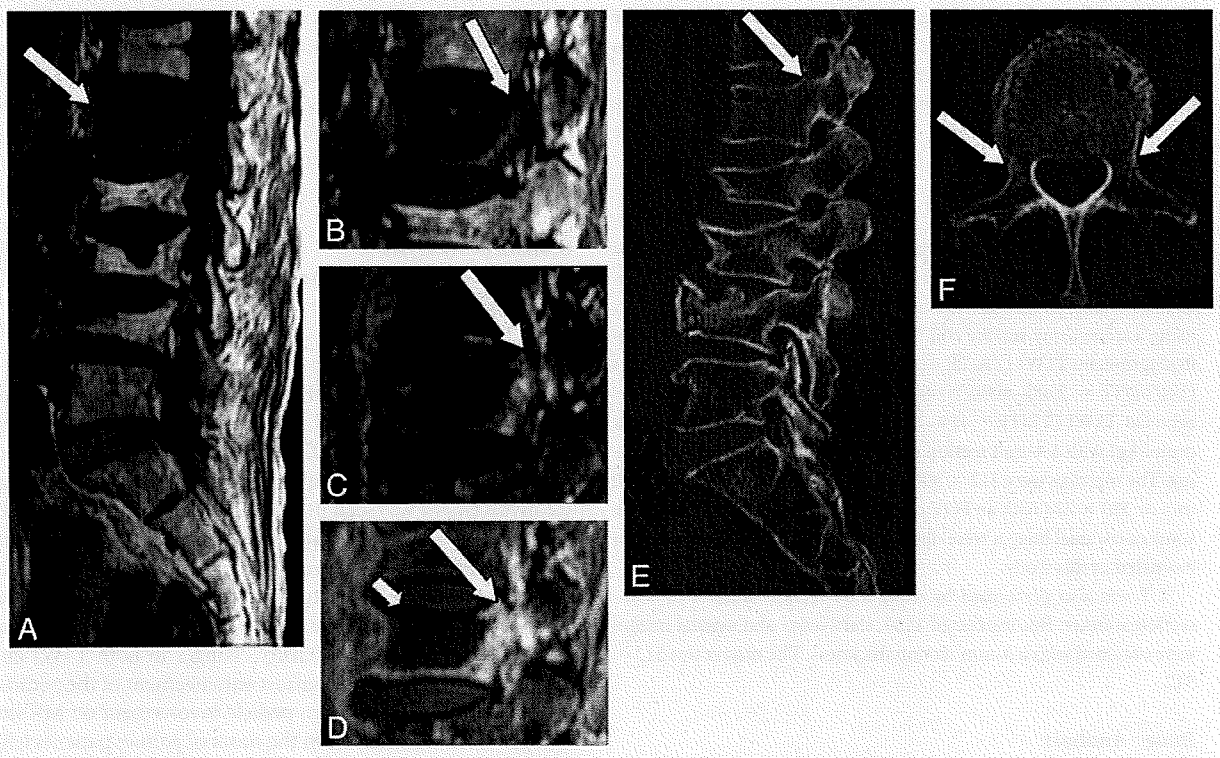

Fig 3. A 76-year-old woman with benign compression fracture, which occurred 1 month earlier. *A* and *B*, T1WI shows diffuse hypointensity in the vertebral body and in the left pedicle of L1 (*arrow*). *C*, STIR image shows heterogeneous hyperintensity in these areas (*arrow*). *D*, Contrast-enhanced T1WI shows marked enhancement (*arrow*). A large area of low signal intensity indicates necrosis or cleft (*small arrow*). *E* and F, On sagittal (*E*) and axial (*F*) CT scans, there is no apparent abnormality in the pedicle (*arrow*).

Table 1: Pedicle involvement in osteoporotic compression fractures (benign) and malignant pathologic fractures on MR imaging

	Pedicle Involvement on MR Imaging	
Fracture	Positive	Negative
Benign (n = 225)	144 (64%)[a]	81
Malignant (n = 19)	16 (84%)[a]	3

[a] P = .065.

Table 2: The relationship between pedicle involvement and duration from onset of pain in patients with osteoporotic compression fractures

	Pedicle Involvement on MR Imaging	
Duration from Onset	Positive (n = 144)	Negative (n = 81)
1–3 Months (n = 109)	84 (77%)[a]	25
>3 Months (n = 116)	60 (51%)[a]	56

[a] P < .001.

Table 3: The correlation between pedicle involvement in osteoporotic fractures and CT findings

	Pedicle Involvement on MR Imaging	
CT Findings	Positive (n = 144)	Negative (n = 81)
No findings (n = 111)	44	67
Sclerosis (n = 69)	55	14
Fracture (n = 45)	45	0

months) and 60 (51.3%) of 116 vertebrae with chronic-phase fractures (>3 months). Early-phase fractures showed a significantly larger number of patients with pedicle involvement than chronic-phase fractures did (P < .001).

The correlation between pedicle involvement on MR imaging for osteoporotic fractures and CT findings is given in Table 3. In 144 osteoporotic fractures with pedicle involvement on MR imaging, sclerosis and pedicle fractures were seen in 55 vertebrae (38.2%) and 45 vertebrae (31%) on CT, respectively. No abnormality in the pedicle was seen in 44 vertebrae (30.5%). MR imaging showed pedicle involvement in all patients with pedicle fracture on CT.

Discussion

Differentiating benign osteoporotic compression fractures from malignant pathologic fractures is clinically important because both occur frequently in elderly patients. For many years, various diagnostic signs on CT and MR imaging have been considered useful for such evaluation.[1-12]

Morphologic changes suggest malignancy when a convex posterior cortex of the vertebral body is seen due to a mass effect or epidural and/or paravertebral masses.[1-4,8] Among these signs, the presence of an epidural mass has been reported as both specific and sensitive. Pedicle involvement has also been described as specific for malignant lesions.[3] In addition, findings such as destruction of anterolateral or posterior cortical bone of the vertebral body or the pedicle on CT have been reported to suggest a malignant process.[2]

On the other hand, benign osteoporotic compression fractures show various signal-intensity patterns on MR imaging. Early-phase fractures typically show a bandlike low signal intensity on T1WI, which is localized adjacent to the collapsed endplate. In most cases, signal-intensity changes are focal, and normal marrow is preserved in at least 1 area of the vertebral body.[3] An intravertebral cleft including fluid or gas and a linear signal-intensity hyperintensity on STIR are significantly associated with osteoporotic fractures.[6] These are believed to reflect avascular necrosis or a nonunionized bony gap with pseudoarthrosis. In addition, retropulsion of a posterior bone fragment is considered both sensitive and specific for osteoporotic fractures.[2]

The usefulness of diffusion-weighted MR imaging or chemical shift MR imaging in differentiating benign and malignant vertebral lesions has been reported recently, but the results are controversial.[13,14]

Correct diagnosis can be made in most cases of osteoporotic compression fracture because the useful signs mentioned above usually coexist in the collapsed vertebrae. However, in our experience, imaging features mimic malignant compression fractures in acute osteoporotic compression fractures, when the height of the vertebral body is preserved and diffuse signal-intensity changes are seen on T1WI, STIR, and Gd-T1WI. In such cases, a differential diagnosis is not always easy.

Because pedicle involvement is a common notion suggesting a malignant nature, we tend to diagnose malignant compression fracture when pedicle involvement is the only sign (Fig 1). We always evaluate the pedicle with CT and MR imaging before PV, because fracture or abnormal signal-intensity changes of the pedicle may be the source of pain and instability. In such cases, pediculoplasty may be beneficial to relieve the pain.[15-18] From our experience, abnormal signals of the pedicle are frequently seen on MR imaging with osteoporotic compression fractures, particularly in the early phase, and fracture of the pedicle is seen in approximately one-third of our patients.

As described in the "Results" section, abnormal signal intensity and contrast enhancement were seen in the pedicle in 64% of benign compression fractures and 84.2% of malignant pathologic fractures, with no significant difference between groups. This suggests that pedicle involvement is also common in benign osteoporotic compression fractures. These results differ from those of previous articles.

Yuh et al[1] compared MR imaging findings between 84 benign fractures and 25 malignant fractures on T1WI and T2WI. They reported pedicle involvement in 22 of the 25 patients with malignant compression fractures but in none of 52 nontraumatic compression fractures, contrasting starkly with our observations. They did not perform STIR or Gd-T1WI, which could have demonstrated subtle pathologic abnormalities in the pedicle. The results might, thus, have differed if these sequences had been used. Moreover, onset of fracture and duration of pain were not described. The prevalence of pedicle involvement varies depending on the age of the fracture. Tumor involvement usually remains for a long duration if untreated, but signal intensity abnormality of the pedicle may disappear after healing of a benign compression fracture.

Cuenod et al[3] compared MR imaging findings between 63 osteoporotic fractures and 30 malignant compression fractures in the acute phase (<2 months). The MR imaging protocol included T1WI, T2WI, T2*WI, and Gd-T1WI without

fat suppression. Pedicle involvement was seen in 24 malignant fractures and 4 benign fractures, and sensitivity and specificity for malignancy were 80% and 94%, respectively, suggesting pedicle involvement as a specific sign for malignancy. They did not use a fat-suppression technique, which might be the reason for the low detectability of pedicle involvement in their patients with osteoporosis. Generally, detection of pedicle involvement by tumor is not difficult due to the presence of homogeneous diffuse signal-intensity changes or a mass effect, but signal-intensity changes in osteoporotic fractures are sometimes difficult to recognize because they are often focal and inhomogeneous. Bone marrow is almost completely replaced with fatty tissue in elderly patients, so fat suppression is considered necessary for the evaluation of diseased vertebrae.

Shih et al[4] examined MR imaging findings for 37 patients with single-level vertebral compression fractures, including 21 patients with benign causes and 16 patients with malignancy. The imaging protocols included T1WI, T2WI with fat suppression, and Gd-T1WI with fat suppression, similar to those in the present study. They identified pedicle involvement in 6 vertebrae with benign fractures (28.6%) and 11 vertebrae with malignant fractures (68.8%), showing a clearly higher prevalence of pedicle involvement in benign compression fractures compared with those in the other studies mentioned above.[1,11] This higher rate of pedicle involvement in osteoporotic compression fractures compared with other studies is probably due to the use of fat-suppression techniques. However, a difference from our study is seen in patient selection. They selected patients with solitary vertebral collapse as the only inclusion criterion. Symptoms of patients were not mentioned in that investigation, while we analyzed patients with acute-to-chronic painful fractures. Signal-intensity changes are commonly seen in unhealed painful fractures and presumably contributed to the difference from our results.

To the best of our knowledge, no previous reports have identified correlations between the findings of pedicle involvement on MR imaging and CT. We investigated CT findings of the pedicle with or without pedicle involvement on MR imaging. Pedicle involvement in osteoporotic compression fractures exhibited 3 morphologic patterns on CT in this study: fracture, sclerotic change, and no abnormality. Fracture and sclerotic change of the pedicle were noted in 31% and 38.2% of vertebrae, respectively, in which MR imaging showed signal-intensity abnormality. Laredo et al[2] compared CT findings of 34 benign osteoporotic fractures with those of 32 malignant acute vertebral compression fractures. They found pedicle fracture in 3 vertebrae with osteoporotic fractures (8.8%). That prevalence was significantly lower than that found in the present study. The reason is unclear but may be attributable to differences in the CT imager and imaging protocol because they evaluated by using axial images alone. We analyzed images with reconstructed axial, sagittal, and coronal planes with 3-mm section thickness obtained by using 16- or 64-detector row CT, which seems likely to have contributed to the higher detectability of abnormal findings in the pedicle in our series compared with that of Laredo et al.[2]

Sclerosis of the pedicle appears to represent reactive change or the healing process of microfracture. Signal-intensity changes on MR imaging are considered to represent the inflammatory process, including exudation, inflammatory cell infiltration, granulation tissue, or fibrosis.

In our study, 30% of vertebrae in which abnormal signals were seen in the pedicle on MR imaging showed no obvious abnormality on CT. In those cases, the fractured vertebral body showed low signal intensity on T1WI and high signal intensity on STIR and Gd-T1WI, indicating edema and inflammatory changes. These changes presumably extended posterior to the pedicle. Our results show that abnormal signals can be seen more frequently in the pedicle with early-phase fractures than with chronic fractures. Such signal-intensity changes are likely to disappear when the healing process is completed, as with fractures in other locations; however, a long period may be required for complete healing.

The present study had some limitations. First, diagnosis of benign or malignant fracture was mainly made on the basis of patient history and clinical presentation, and biopsy was performed in only selected patients. Some patients diagnosed with benign fracture thus could possibly have had malignant compression fractures. However, we believe such cases were unlikely and would have had little influence on the study result because no new malignancies were found in any patients at 3-month and 1-year follow-ups after PV.

Second, our study had a selection bias. We included only patients who underwent PV. It is possible that patients included had a higher likelihood of pedicle involvement than the general population because we performed PV for patients who had some signal-intensity changes on MR imaging in fractured vertebrae. So, this result may not be applicable to the larger population.

Third, due to time constraints, the MR imaging protocol for PV at our institution included only sagittal images, and we could not assess axial images in any except a few patients. Axial imaging may thus yield some additional information.

Conclusions

Pedicle involvement, which is accepted as a common indicator of malignant processes, is also frequent in patients with osteoporotic compression fractures, particularly in the early phase, and was not specific for malignancy in our study group. Although differentiating benign osteoporotic compression fractures and malignant pathologic fractures is possible in most cases, a diagnosis of malignant pathologic fracture should not be assumed when pedicle involvement is the only sign.

References

1. Yuh WT, Zachar CK, Barloon TJ, et al. **Vertebral compression fractures: distinction between benign and malignant causes with MR imaging.** *Radiology* 1989;172:215–18
2. Laredo JD, Lakhdari K, Bellaiche L, et al. **Acute vertebral collapse: CT findings in benign and malignant nontraumatic cases.** *Radiology* 1995;194:41–48
3. Cuenod CA, Laredo JD, Chevret S, et al. **Acute vertebral collapse due to osteoporosis or malignancy: appearance on unenhanced and gadolinium-enhanced MR images.** *Radiology* 1996;199:541–49
4. Shih TT, Huang KM, Li YW. **Solitary vertebral collapse: distinction between benign and malignant causes using MR patterns.** *J Magn Reson Imaging* 1999;9:635–42
5. Baur A, Stabler A, Arbogast S, et al. **Acute osteoporotic and neoplastic vertebral compression fractures: fluid sign at MR imaging.** *Radiology* 2002;225:730–35
6. Oka M, Matsusako M, Kobayashi N, et al. **Intravertebral cleft sign on fat-suppressed contrast-enhanced MR: correlation with cement distribution pattern on percutaneous vertebroplasty.** *Acad Radiol* 2005;12:992–99
7. Sugimura K, Yamasaki K, Kitagaki H, et al. **Bone marrow diseases of the spine:**

differentiation with T1 and T2 relaxation times in MR imaging. *Radiology* 1987;165:541–44
8. Baker LL, Goodman SB, Perkash I, et al. **Benign versus pathologic compression fractures of vertebral bodies: assessment with conventional spin-echo, chemical-shift, and STIR MR imaging.** *Radiology* 1990;174:495–502
9. Algra PR, Heimans JJ, Valk J, et al. **Do metastases in vertebrae begin in the body or the pedicles? Imaging study in 45 patients.** *AJR Am J Roentgenol* 1992;158:1275–79
10. An HS, Andreshak TG, Nguyen C, et al. **Can we distinguish between benign versus malignant compression fractures of the spine by magnetic resonance imaging?** *Spine* 1995;20:1776–82
11. Mehta RC, Marks MP, Hinks RS, et al. **MR evaluation of vertebral metastases: T1-weighted, short-inversion-time inversion recovery, fast spin-echo, and inversion-recovery fast spin-echo sequences.** *AJNR Am J Neuroradiol* 1995;16:281–88
12. Zajick DC Jr, Morrison WB, Schweitzer ME, et al. **Benign and malignant processes: normal values and differentiation with chemical shift MR imaging in vertebral marrow.** *Radiology* 2005;237:590–96
13. Baur A, Stabler A, Bruning R, et al. **Diffusion-weighted MR imaging of bone marrow: differentiation of benign versus pathologic compression fractures.** *Radiology* 1998;207:349–56
14. Spuentrup E, Buecker A, Adam G, et al. **Diffusion-weighted MR imaging for differentiation of benign fracture edema and tumor infiltration of the vertebral body.** *AJR Am J Roentgenol* 2001;176:351–58
15. Fuwa S, Numaguchi Y, Kobayashi N, et al. **Percutaneous pediculoplasty for vertebral hemangioma involving the neural arch: a case report.** *Cardiovasc Intervent Radiol* 2008;31:189–92
16. Eyheremendy EP, De Luca SE, Sanabria E. **Percutaneous pediculoplasty in osteoporotic compression fractures.** *J Vasc Interv Radiol* 2004;15:869–74
17. Gailloud P, Beauchamp NJ, Martin JB, et al. **Percutaneous pediculoplasty: polymethylmethacrylate injection into lytic vertebral pedicle lesions.** *J Vasc Interv Radiol* 2002;13:517–21
18. Martin JB, Wetzel SG, Seium Y, et al. **Percutaneous vertebroplasty in metastatic disease: transpedicular access and treatment of lysed pedicles—initial experience.** *Radiology* 2003;229:593–97