

Case Report

Percutaneous Vertebroplasty-Induced Adjacent Vertebral Compression Fracture

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Background: The risks associated with percutaneous vertebroplasty (PV) are low. Patients show marked improvement and are able to rapidly resume normal activities after PV. The sudden development of postoperative vertebral compression fracture (VCF) is a common complication, and additional PV is frequently performed in these cases. However, there have been no studies reporting acute compression fractures of an adjacent vertebra immediately after PV.

Objective: This case report presents a rare case in which the patient had to undergo a second PV because of PV-induced adjacent VCF. Further, we review previous studies and discuss the possible pathogenesis of this rare complication.

Study Design: Case report.

Setting: Pain management clinic.

Methods: A 62-year-old woman presented with a severe pain in the lower back, which started after she slipped. A radiograph showed severe vertebral collapse with a vertebral vacuum cleft in the T12 vertebral body. T1-weighted magnetic resonance imaging showed low signal intensity in T12, suggesting acute VCF, but the signals from the other vertebrae were normal.

Results: The patient underwent PV at T12. When the cannula was inserted into the fracture line of the vertebral body, reduction of the collapsed T12 was developed. Although the postoperative course was uneventful, the patient's pain did not resolve. Postoperative radiographic image obtained 4 hours after the PV showed reduction of T12 and adjacent acute VCF in T11. We performed a second PV at T11. However, 2 weeks later, adjacent acute VCF in L1 was developed and PV was performed.

Limitations: This report describes a single case.

Conclusion: To the best of our knowledge, this is the first case report of adjacent VCF that developed almost immediately after PV. Although the exact mechanism underlying this rare complication remains unclear, we assume that the VCF was induced by PV, although this was not proven. However, we suggest that the insertion of the cannula into the fracture line induced the iatrogenic dynamic mobility of the fractured vertebra. Reduction was caused by the cannula and positional gravity. The upward reduction may have had an effect on the upper and adjacent vertebrae.

Key words: Compression fractures, vertebroplasty, cannula, complication.

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Percutaneous vertebroplasty (PV) is a therapeutic, interventional radiological procedure that involves injecting bone cement into a vertebral body lesion to relieve pain and strengthen the bone. PV is considered a very safe and effective procedure for treating painful vertebral compression fractures (VCFs),

with nearly 90% of patients experiencing some pain relief (1). Symptomatic complications of PV are rare and have been reported to occur in less than 3% of the cases of osteoporotic VCFs (2). Sudden development of postoperative VCF is a common complication, and additional PV is frequently performed in these cases

(3-5). However, there have been no studies reporting acute compression fractures of an adjacent vertebra immediately after PV. We present a rare case in which the patient had to undergo a second PV because of PV-induced adjacent VCF. Further, we review previous studies and discuss the possible pathogenesis of this rare complication.

CASE REPORT

A 62-year-old woman presented with a 2-week history of severe pain in the lower back, which started after a slip. Her T-score for bone mineral density (BMD) was -1.5, which suggested osteopenia, and her visual analog scale (VAS) score was 8.1 {suggest clarifying that this was an 11 point scale or whatever the scale was}. Ten years before the present admission, she had undergone coronary artery bypass graft surgery for stable angina; mitral annuloplasty for mitral valve prolapse and mitral regurgitation; and patch closure for atrial septal defect. She had also been taking medications for chronic kidney disease, Parkinson disease, hypertension, diabetes mellitus, and gout for the past several years.

Lumbar magnetic resonance imaging showed low signal intensity on the T1-weighted image and high signal intensity on the short time inversion recovery image, suggesting acute VCF in L1, but the signals from

the other vertebrae were normal (Fig. 1). Radiographic analysis revealed severe vertebral collapse with intra-vertebral vacuum cleft in the T12 vertebra. The anterior vertebral height of T11 was 26 mm, T12 was 8 mm, and L1 was 30 mm (Fig. 2A). She underwent PV at T12 through a bilateral transpedicular approach. Under C-arm fluoroscopic guidance, we inserted a cannula into the fractured vertebral body. When the cannula was inserted into the fracture line of the vertebral body, a 19 mm reduction of the collapsed T12 was developed (Fig. 2B). Therefore, we could inject a larger amount of cement than expected without cement leakage.

Although the postoperative course was uneventful, the patient's pain did not resolve even at 4 hours after the operation; her VAS score increased to 9.2. A postoperative radiographic image obtained 4 hours after the PV showed reduction of the T12 vertebral body. However, the anterior vertebral height of T11 had further reduced to 15 mm, which suggested adjacent acute VCF in T11 (Fig. 2C). She received conservative treatment, including facet joint and medial branch blocks and medication, and was advised bed rest.

Despite receiving this treatment for 2 weeks, the pain did not subside; therefore, we performed a second PV at T11. The patient showed relief from pain and was discharged one week after the operation. However,

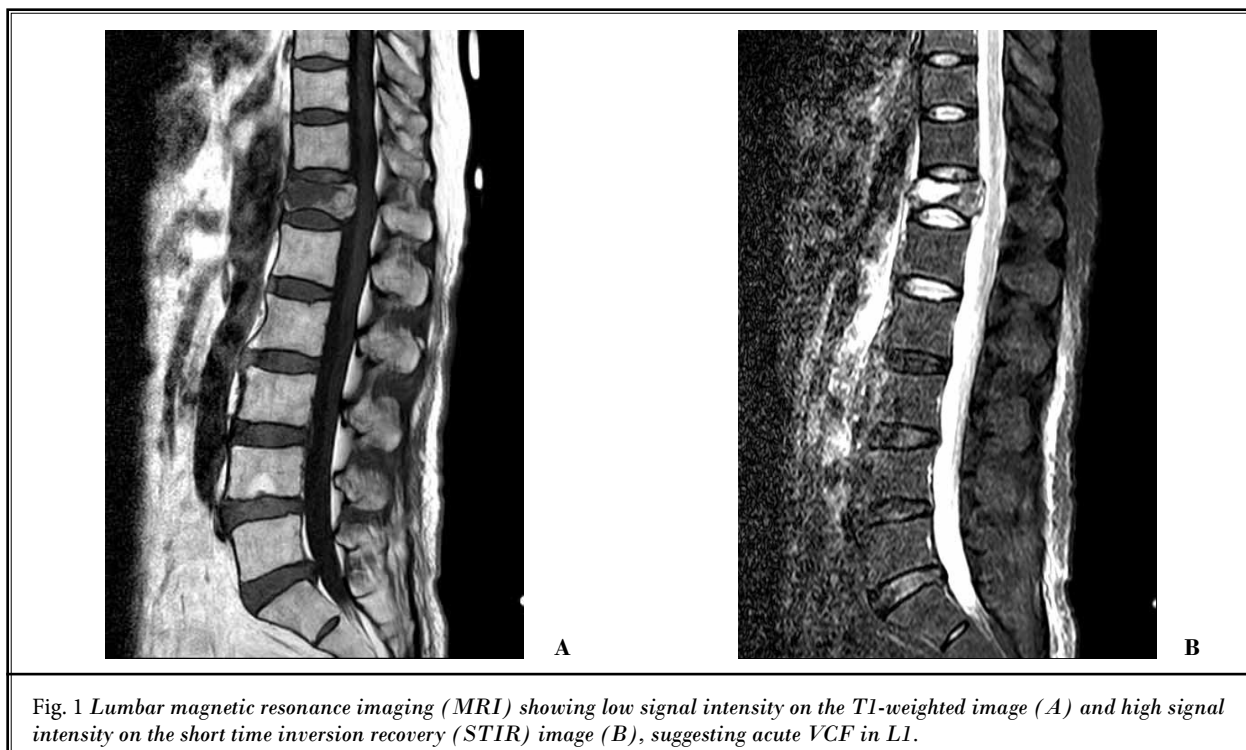


Fig. 1 Lumbar magnetic resonance imaging (MRI) showing low signal intensity on the T1-weighted image (A) and high signal intensity on the short time inversion recovery (STIR) image (B), suggesting acute VCF in L1.

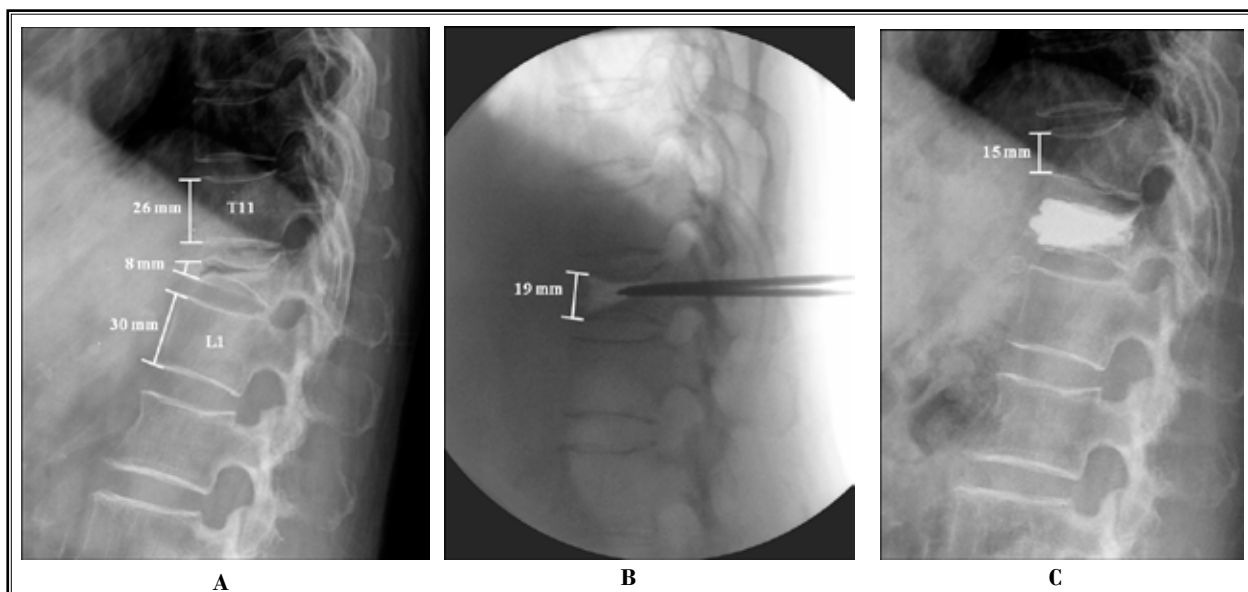


Fig. 2 (A) Radiographic image showing severe vertebral collapse with vacuum cleft in T12. (B) Intraoperative lateral image of C-arm fluoroscopy showing cannula induces reduction of collapsed T12 vertebral body. (C) Radiographic image obtained 4 hours after the percutaneous vertebroplasty showing reduction of the T12 vertebral body and cement within T12 and acute vertebral compression fracture in T11.

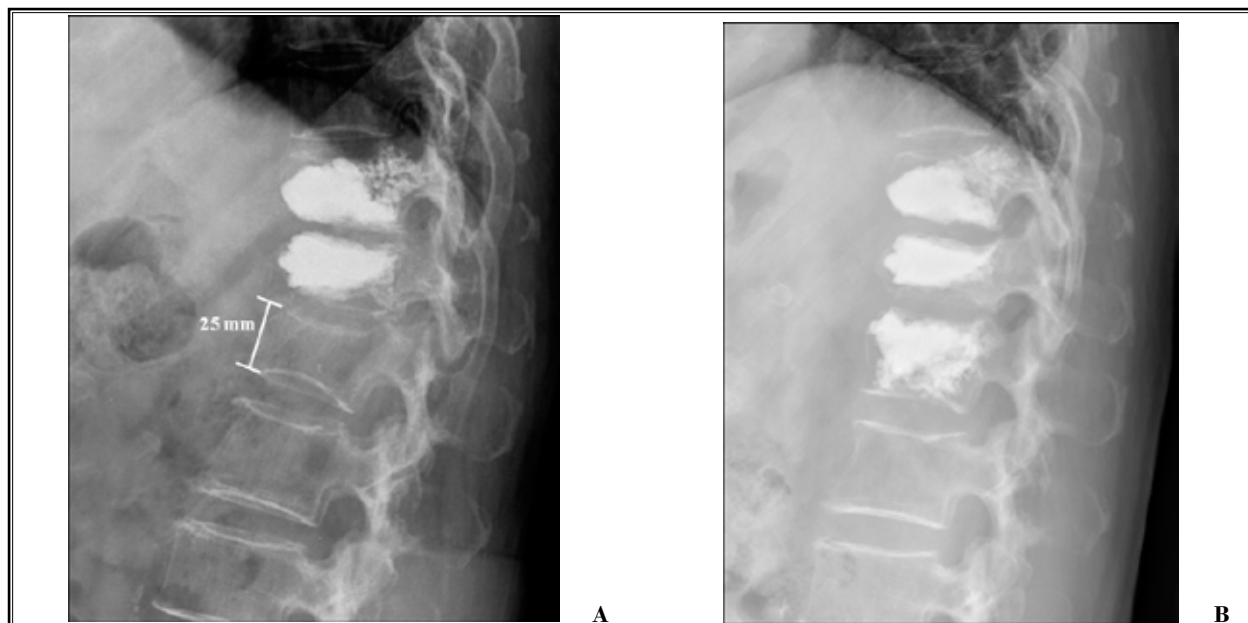


Fig. 3 (A) Radiographic image obtained 2 weeks after PV at T11 showing T11 and T12 without any cement leakage and acute vertebral compression fracture in L1. (B) Postoperative radiographic image showing cement within T11, T12, and L1.

one week later, she was readmitted with a 2-day history of severe back pain, similar to the pain she had experienced after the initial PV. Her trauma history was unremarkable. Images obtained from repeated radiography

revealed that the anterior vertebral height of L1 had reduced to 25 mm, suggesting acute VCF of L1 (Fig. 3A). The patient rejected conservative treatment; hence, we performed PV at L1 (Fig. 3B). She reported relief from

pain (VAS score, 3.1) and was discharged on the fourth postoperative day. Five months after the third PV, the patient showed good recovery without any PV-related complications.

Discussion

Currently, PV is performed in patients with osteoporotic VCFs who present with severe mechanical back pain, have restricted mobility, and require opioid analgesics. The risks associated with PV are low. Patients show marked improvement and are able to rapidly resume normal activities (2,6). Biomechanical tests have shown that PV generally restores or increases vertebral body strength and stiffness, in comparison to the corresponding prefracture values; the cement injected during PV may also form an internal splint that immobilizes the fracture site (7,8).

The overall complication rate for PV in the treatment of osteoporotic VCFs is reported to be 1–3% (2). The major complications of PV include epidural hematomas caused by a medial pedicle-wall breach, inaccurate cement filling because of needle malposition, leakage of bone cement into the epidural and neural foraminal areas, pulmonary embolization caused by polymethylmethacrylate, arterial injury, and death (9). However, these complications are very rare.

Studies have shown that the incidence of VCF after PV varies from 12% to 52% (4). It is uncertain whether PV itself is the cause of subsequent VCFs. Some authors have reported that a fracture after PV is indicative of the progression of an underlying disease, whereas others have suggested that cement augmentation and physical activity after PV may cause VCFs (4,10,11). Ahn et al (4) postulated that the mechanisms underlying the development of compression fractures after PV differ for adjacent and nonadjacent vertebra.

The development of adjacent fractures can be explained by the direct pillar effect. Cement augmentation because of intradiscal cement leakage may increase the strength gradient, leading to a fracture in the weaker adjacent vertebra. The development of a nonadjacent fracture can be explained by the dynamic hammer effect. If an adjacent vertebral segment is already rigid, the pillar effect is not prominently observed; however, a remote mobile segment may be affected by the strength gradient created by the use of additional cement. The difference in mobility between the rigid adjacent vertebral segment and the relatively remote mobile segment may cause a nonadjacent fracture. Patients usually experience rapid clinical im-

provement after undergoing PV, after which they may engage in activities that they were unable to perform previously. The resultant increased axial load on the vertebra may cause an increase in stress and compression of adjacent vertebra. Further, during normal activities, a patient may fall, resulting in the fracture of other vertebrae, or even fractures in the extremities and hip (12).

The long-term risk of subsequent fractures is high after a VCF; new compression fractures occur repeatedly after PV (5). Tseng et al (5) reported that the risk of occurrence of new adjacent fractures is high after PV. These fractures tend to occur earlier than nonadjacent fractures (adjacent fractures, 71.9 ± 71.8 days after PV; nonadjacent fractures, 286.8 ± 232.8 days after PV). The risk factors for multiple VCFs (2 or more VCFs) are old age, low baseline BMD, and other pre-existing VCFs (5).

In the present case, although the anterior vertebral height of T11 was intraoperatively observed to be normal under C-arm fluoroscopy, radiographic images obtained 4 hours after PV showed acute VCF in T11. The authors think that this acute VCF of T11 was induced by PV. The reasons for this assumption are as follows: First, the patient was on bed rest without performing any weight-bearing activities, including standing or sitting, until she underwent postoperative radiography. Second, we observed certain characteristics during the operation that were different from the usual PV for patients with osteoporotic VCFs. We observed a reduction of the fractured vertebra when the cannula was inserted into the fracture line, and we assume that insertion of the cannula via a transpedicular approach may have induced the iatrogenic dynamic mobility of the fractured vertebra. McKiernan et al (13) defined dynamic mobility as "any measurable changes in vertebral body height between the standing and supine positions in radiographs." This phenomenon could be observed in 44% of VCF patients. In our case, the reduction is caused by the cannula and the effect of gravity when the patient is in the prone position. As a result, a lot of bone cement was injected into the fractured vertebra densely during operation. The upward reduction of T12 after PV may have had an effect on the upper and adjacent T11 (Fig 4).

We evaluated why this complication developed in our patient. Although her BMD level indicated osteopenia, we were able to inject more cement than is usually possible for other osteoporotic patients. Further, our patient had a history of many diseases and was

taking many medicines for many years. Therefore, despite having relatively sufficient bone density, she may have had weak strength and stiffness in the vertebral body, and weak adjacent vertebrae might be vulnerable to minor trauma, such as the upward reduction of T12 after PV.

CONCLUSION

To the best of our knowledge, this is the first case report of adjacent VCF that developed almost immediately after PV. Although the exact mechanism underlying this rare complication remains unclear, we assume that the VCF was induced by PV. PV is an effective treatment for painful osteoporotic VCFs. However, we suggest that patients who have had many previous diseases and who have been taking several medicines, should be informed about the significantly potentially high risk of subsequent adjacent and nonadjacent VCFs after PV. Further, biomechanical and clinical studies should be conducted to obtain more precise data.

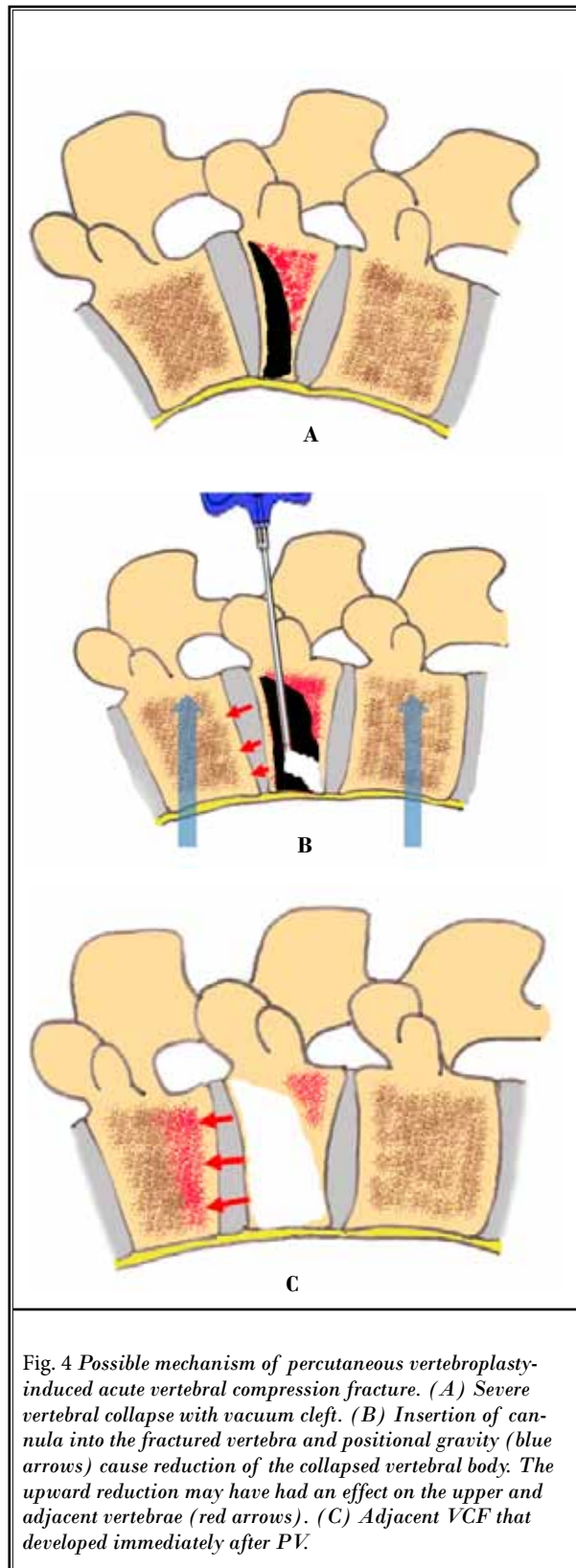


Fig. 4 Possible mechanism of percutaneous vertebroplasty-induced acute vertebral compression fracture. (A) Severe vertebral collapse with vacuum cleft. (B) Insertion of cannula into the fractured vertebra and positional gravity (blue arrows) cause reduction of the collapsed vertebral body. The upward reduction may have had an effect on the upper and adjacent vertebrae (red arrows). (C) Adjacent VCF that developed immediately after PV.

REFERENCES

1. Hulme PA, Krebs J, Ferguson SJ, Berlemann U. Vertebroplasty and kyphoplasty: A systematic review of 69 clinical studies. *Spine (Phila Pa 1976)* 2006; 31:1983–2001.
2. Uppin AA, Hirsch JA, Centenera LV, Pfiefer BA, Pazianos AG, Choi IS. Occurrence of new vertebral body fracture after percutaneous vertebroplasty in patients with osteoporosis. *Radiology* 2003; 226:119–124.
3. Deramond H, Depriester C, Galibert P, Le Gars D. Percutaneous vertebroplasty with polymethylmethacrylate. Technique, indications, and results. *Radiol Clin North Am* 1998; 36:533–556.
4. 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.
5. Tseng YY, Yang TC, Tu PH, Lo YL, Yang ST. Repeated and multiple new vertebral compression fractures after percutaneous transpedicular vertebroplasty. *Spine (Phila Pa 1976)* 2009; 34:1917–1922.
6. Tohmeh AG, Mathis JM, Fenton DC, Levine AM, Belkoff SM. Biomechanical efficacy of unipedicular versus bipedicular vertebroplasty for the management of osteoporotic compression fractures. *Spine (Phila Pa 1976)* 1999; 24:1772–1776.
7. Baroud G, Bohner M. Biomechanical impact of vertebroplasty. Postoperative biomechanics of vertebroplasty. *Joint Bone Spine* 2006; 73:144–150.
8. Dean JR, Ison KT, Gishen P. The strengthening effect of percutaneous vertebroplasty. *Clin Radiol* 2000; 55:471–476.
9. Cosar M, Sasani M, Oktenoglu T, Kaner T, Ercelen O, Kose KC, Ozer AF. The major complications of transpedicular vertebroplasty. *J Neurosurg Spine* 2009; 11:607–613.
10. Lindsay R, Burge RT, Strauss DM. One year outcomes and costs following a vertebral fracture. *Osteoporos Int* 2005; 16:78–85.
11. Kim SH, Kang HS, Choi JA, Ahn JM. Risk factors of new compression fractures in adjacent vertebrae after percutaneous vertebroplasty. *Acta Radiol* 2004; 45:440–445.
12. Jensen ME, Dion JE. Percutaneous vertebroplasty in the treatment of osteoporotic compression fractures. *Neuroimaging Clin N Am* 2000; 10:547–568.
13. McKiernan F, Jensen R, Faciszewski T. The dynamic mobility of vertebral compression fractures. *J Bone Miner Res* 2003; 18:24–29.