AUTHOR QUERY FORM

ELSEVIER	**Journal:** JOCD **Article Number:** 819	**Please e-mail your responses and any corrections to:** **E-mail:** j.beckemeier@elsevier.com

Dear Author,

Please check your proof carefully and mark all corrections at the appropriate place in the proof (e.g., by using on-screen annotation in the PDF file) or compile them in a separate list. Note: if you opt to annotate the file with software other than Adobe Reader then please also highlight the appropriate place in the PDF file. To ensure fast publication of your paper please return your corrections within 48 hours.

For correction or revision of any artwork, please consult http://www.elsevier.com/artworkinstructions.

Any queries or remarks that have arisen during the processing of your manuscript are listed below and highlighted by flags in the proof.

Location in article	Query / Remark: Click on the Q link to find the query's location in text Please insert your reply or correction at the corresponding line in the proof
	If there are any drug dosages in your article, please verify them and indicate that you have done so by initialing this query
Q1	Please check whether the short title provided is correct. Kindly amend if necessary.
Q2	Please provide department name (if any) for the affiliation and location detail (e.g., city, state, and country) for the affiliation.
Q3	Please provide complete location details in the corresponding author field. Also kindly check whether the e-mail id retained is correct.
Q4	Please note that 3−5 keywords are required in the keywords section. Please provide two or more keywords.
Q5	Please check the edits to the sentence "Either symptomatic (painful) or …" and correct if necessary.
Q6	Please expand "PDC" in the sentence "The International Society for Clinical Densitometry …".
Q7	Please expand "PA" in the sentence "The underreporting of the presence of …".
Q8	Please note that the phrase "number of clinical that often" in the sentence "There are a number of clinical …" seems to be incomplete. Please check and make necessary changes.
Q9	Please check the edits to the sentence "Both the bisphosphonates …" and correct if necessary.
Q10	Please verify the page range in Ref. 26.
Q11	Refs. 13 and 35: Please provide accessed date.
Q12	Please confirm that given names and surnames have been identified correctly.

(*continued on next page*)

	Please check this box or indicate your approval if you have no corrections to make to the PDF file ☐

Thank you for your assistance.

Journal of Clinical Densitometry: Assessment & Management of Musculoskeletal Health, vol. ■, no. ■, 1–5, 2015

1094-6950/■:1–5/$36.00
http://dx.doi.org/10.1016/j.jocd.2015.08.006

Original Article

Clinical Management of Vertebral Compression Fractures

*Paul D. Miller**

Colorado Center for Bone Research and Medicine University of Colorado Health Sciences Center

Abstract

Vertebral compression fractures (VCF's) are the most common form of osteoporotic fractures. Whether symptomatic or asymptomatic, they both represent a high risk for not only vertebral but also nonvertebral fractures in untreated populations. This high risk of future fracture after a VCF is independent of the T-score because bone strength is a combination of bone mineral density and bone quality. VCFs are the single greatest risk for future fractures at all other skeletal sites in untreated populations, including hip fractures. They are often unrecognized despite their exceptionally high prevalence in all genders and most ethnic groups as age increases. This article highlights some of the key messages about VCF's, and how assessment for their presence and then management will reduce the risk of all osteoporotic fractures.

Key Words: Vertebral fractures.

Introduction

Vertebral compression fractures (VCFs) are the most common fragility fractures in all forms of osteoporosis, including postmenopausal, male, and glucocorticoid-induced osteoporosis *(1–3)*. The presence of a fragility vertebral fracture has several clinical and management implications.

Clinical Implications

Either symptomatic (painful) or asymptomatic (radiographically defined) VCFs have clinical implications if unrecognized and untreated. Either type of fractures reduces pulmonary vital capacity, leads to a greater risk of other fragility fractures at both vertebral bodies and other skeletal sites (hip, forearm, pelvis). It is as if vertebral fractures are conveying a signal of systemic skeletal fragility. Patients with either clinical (painful) or morphometric (X-ray diagnosed) have a greater risk for falling.

Management Implications

1. A VCF makes the diagnosis of osteoporosis independent of the bone mineral density (BMD) level or "T-score" *(4–9)*.
2. A VCF requires a work-up for secondary causes of osteoporosis *(10)*.
3. A VCF fulfills virtually all international clinical guidelines for pharmacological treatment in addition to adequate calcium, vitamin D, and fall prevention strategies *(11–14)*.

The reality is that most VCF are missed by clinicians. The reasons behind this under diagnosis and under treatment of VCF include the following:

1. A lack of awareness that most VCFs are asymptomatic. Clinicians are looking for pain as the clue to the possible presence of a VCF *(15–20)*.
2. The underappreciation that even morphometric (radiological detected) VCFs convey a high risk, for not only more VCFs but also other fractures at other skeletal sites *(19–26)*.
3. That VCFs exist although the T-score is normal *(4,5)*.
4. That simple height measurements are often not even done in physician offices; or rather, if done, are often done on inaccurate scales (e.g., the "metal rod") rather than the wall mounted and inexpensive stadiometer.
5. Height loss should be the alerting signal that a VCF may be present. The International Society for Clinical Densitometry has established specific prevalent or interval height loss values that have a high probability of detecting either a prevalent or incident VCF (International Society for Clinical Densitometry PDC 2013

Received 11/19/14; Accepted 08/12/15.
*Address correspondence to: Paul D. Miller, MD. E-mail: illerjcd@aol.com

6. The underreporting of the presence of VCF by radiologists examining routine PA and lateral chest X-rays.

The "care gap": a gap exists between measurements of a patient's height, the recognition that a specific amount of height loss should trigger a vertebral radiograph or vertebral fracture assessment and the communication between the office staff concerning the result of the height measurement and the order for the radiological examination of the vertebrae. In part, this "gap" is regulated by reimbursement or legal systems that may not allow a non-licensed medical provider the ability to order an imaging examination.

This article provides some insights into pragmatic ways this author, who sees daily a large volume of referred patients potentially having osteoporosis and other metabolic bone diseases, tries to address these challenging issues of identification and management of VCF.

Symptomatic VCF

Patients who experience the acute onset of mid back pain, or, at times, referred pain to the sternal area are often first seen in the emergency department because they go there of their own motivation or are sent there by their managing physician. It is probably the right process as more immediate life-threatening conditions such as a myocardial infarction, dissecting aortic aneurysm, pulmonary embolus, or even esophageal rupture can often present with the same symptomatology.

Once the differential diagnosis includes a VCF, the "dating" of that VCF is important, and this can best be done by a good history with regard to the acuteness of the symptoms and magnetic resonance imaging (MRI) of that anatomical area. The MRI estimation of the duration of the event, is often implied by the degree of edema surrounding the MRI lesion, and this acute MRI pattern usually persists for several months after the fracture *(27,28)*. As important as the dating of the VCF is, the ability of MRI to exclude a pathological fracture, one especially due to metastatic cancer or multiple myeloma is equally important. MRIs of the vertebral fracture is specific for a pathological fracture; and, although an MRI might suggest Paget's disease, Paget's disease of bone is a very specific X-ray diagnosis.

Once the diagnosis of an acute osteoporotic VCF is made, then management focuses on 2 areas:

1. Pain management
2. Management of the skeletal fragility

"Pain management" includes an array of choices in which the physician should use multifactorial approaches: analgesic anti-inflammatory medications; ice packs; rest; and, in time, specific physical therapy *(29–32)*. The severity and duration of the pain from an acute VCF has a wide range of clinical manifestations. The most recalcitrant ones can become life threatening because the patients are often elderly, and their nutrition may become jeopardized quickly. Weight loss and loss of muscle mass (sarcopenia) can occur surprisingly fast if they remain immobilized and have concomitant loss of appetite. When medical management is ineffective, then consideration of vertebroplasty or kyphoplasty is important because both may have an acute analgesic effect and allow for patient recovery *(33–35)*. How long to wait before medical management is considered unsuccessful, and perform either vertebroplasty or kyphoplasty is a clinical judgment. It seems that vertebroplasty or kyphoplasty may be less effective once one has delayed their performance beyond 3–4 mo. In my experience, in the frail elderly, it becomes clear that medical management is unsuccessful within a week or 2 of the acute fracture.

Skeletal fragility: equally important is the initiation of pharmacological therapy to increase bone strength to mitigate further VCF in the immediate time period (i.e., the "cascade" phenomenon) but also to treat the systemic disease of osteoporosis. Not only is the risk of a 2nd VCF very high within the first 12 mo after an acute VCF, but also is the risk of all clinical fractures as well. After the secondary work-up for other causes of osteoporosis has ruled out causes other than estrogen deficiency (e.g., postmenopausal osteoporosis), then the physician has a menu of effective and registered therapies used for the purpose of increasing bone strength and decreasing the risk for subsequent fractures. All the registered treatment for postmenopausal osteoporosis gained registration based on proven efficacy to reduce vertebral fractures as compared with placebo *(36–43)*. Hence, the oral and intravenous bisphosphonates, denosumab, and teriparatide are choices that the clinician can make in a shared decision-making process with these very sick patients.

There are 2 pharmacological agents that have some evidence for an analgesic effect—calcitonin and teriparatide.

The most suggestive acute analgesic effect comes from the injectable calcitonin data, the parenteral formulation [Calcimar; *(44)*]. Most of the analgesic efficacy of calcitonin comes from small studies with little control data. The analgesic effect seems to be dose related. Although the registered parenteral (subcutaneous) dose is 0.5 cc subcutaneous daily, the higher the dose (even 4 times a day) may have a greater analgesic effect. However, the higher the dose of calcitonin, the greater the side effects, especially nausea and vomiting. In addition, from the registration trials of calcitonin, especially the nasal formulation (Miacalcin), the fracture efficacy was only observed for vertebral fractures, and no analgesic effect was observed *(45)*. In Europe, calcitonin has been withdrawn from availability due to the concern about post-marketing reports of certain gastrointestinal cancers *(46)*.

Teriparatide (Forteo), the only currently available anabolic agent registered for osteoporosis management, also has data from clinical trials suggesting a reduction of pain in patients with VCF *(47)*. Because teriparatide is indicated for high-risk patients and patients with acute VCF are clearly at high risk, teriparatide becomes a strong first-line consideration. Because many health-care systems will only approve teriparatide in patients who have "failed" less expensive first-line therapies, the first-line use of teriparatide may be problematic in some health-care systems.

Asymptomatic Vertebral Compression Fractures

Morphometric VCFs are often asymptomatic yet have substantial prognostic implications *(11–14,21–26)*. Although neither the patient nor the physician may be able to date the VCF, they are associated with a high risk for all clinical fractures in the untreated patient; and, are associated with a greater risk for falls and reduction in lung function (vital capacity). Thus, they are serious.

The clinical management starts with identification and then conveying to the asymptomatic patient, whose "T-score" may be normal, that they have osteoporosis and need a work-up for secondary etiologies and treatment. These latter steps are often not without challenges because both patients and referring busy primary care physicians often have little information why these "silent" compressed vertebrae represent a "fracture" in the parlance of metabolic bone experts, or why they represent severe osteoporosis. Payers in this era of restricted reimbursement may deny payment for treatments if their clients' T-scores are not −2.5 or lower as they too are ill informed about the epidemiology implying the importance of morphometric VCF. A fracture is 1 element of bone strength with the other element of bone strength being bone quality, which until the recent introduction of trabecular bone score was not measurable in clinical practice. There are a number of clinical that often have normal T-scores and yet also have VCF where the bone strength is impaired by poor bone quality, especially diabetes mellitus, chronic kidney disease, and the syndrome of lower extremity fractures in healthy premenopausal women. Once trust is created, then the dialogue between patient and physician enters into discussions concerning differential diagnosis and management.

In addition to often overlooked basic interventions, for example, adequate calcium, vitamin D, and fall reduction programs, pharmacological therapy is indicated in these high-risk patients, as in all the registration clinical trials that led to the approval of treatments for postmenopausal osteoporosis, a greater reduction in incident VCF was seen in the treated as opposed to the placebo group, the latter given adequate calcium and vitamin D *(38–43)*.

Both the bisphosphonates (oral and intravenous), the human monoclonal antibody to soluble RANK-Ligand (denosumab) and the anabolic agent, teriparatide, have strong evidence for reduction in incident VCF. Once treatment with one of these agents is begun, then the important issue of defining a therapeutic effect becomes paramount. In addition to encouraging compliance, monitoring also includes serial BMD determinations (to be certain, the BMD does not go down); measurement of bone turnover markers (C-telopeptide in the case of antiresorptive agents and propeptide type I collagen for anabolic agents) to be sure, they change in the expected direction; and changes in height *(48–51)*. Height should remain stable over time. An additional reduction in height of 3/4″ or more justifies a repeat spine radiograph or vertebral fracture assessment to assess whether a new VCF has developed or a preexisting VCF has become worse *(52)*. No drug abolishes risk, but new VCF on therapy should be followed by a reevaluation for previously undiscovered secondary causes of fragility and consideration of changing treatment to therapies with a different mechanism of action.

In conclusions, VCFs, both symptomatic and asymptomatic, acute and chronic, are symbolic of severe systemic skeletal fragility. Most asymptomatic VCFs are not diagnosed because the connection between height loss and the potential for a VCF is underappreciated. Even if seen, the connection between the finding of a "silent" VCF and severe osteoporosis is often not made; hence, therapy is not initiated. In the field of osteoporosis, the proportion of people in certain health plans who receive a pharmacological agent for osteoporosis after a hip fracture has declined since 2001. If such a disconcerting discovery is seen even after a very major symptomatic fracture (hip), one can only surmise that the proportion of patients with asymptomatic VCF not treated is even greater. Kanis et al *(53)* recently published an editorial on how the osteoporosis community has failed in its mission to reduce the burden of osteoporotic fractures. It is time that, as an international community, we reverse this perspective so those patients with VCF, both clinical (painful) and morphometric (asymptomatic), receive proper management.

Acknowledgments

Author's disclosures: (1) Scientific advisory boards: Alexion, Amgen, AgNovos, Lilly, Merck, Radius Pharma, Roche; (2) Research Grants: Amgen, Boehringer Ingelheim, Immunodiagnostics, Lilly, Merck, Merck Serrano, Novartis, Novo Nordisk, Radius Pharma, Roche Diagnostics, Takeda; (3) Equity: none; (4) Speakers bureaus: Alexion Pharmaceuticals and Radius Health.

References

1. Cauley JA, Hochberg MC, Lui LY, et al. 2007 Long-term risk of incident vertebral fractures. JAMA 298:2761–2767.
2. Melton LJ III, Lane AW, Cooper C, et al. 1993 Prevalence and incidence of vertebral deformities. Osteoporos Int 3: 113–119.
3. Cooper C, O'Neill T, Silman A. 1993 The epidemiology of vertebral fractures. European Vertebral Osteoporosis Study Group. Bone 4(1 Suppl):S89–S97.
4. Miller PD. 2006 Guidelines for the diagnosis of osteoporosis: T-scores vs fractures. Rev Endocr Metab Disord 7:75–89.
5. Siris ES, Adler R, Bilezikian J, et al. 2014 The clinical diagnosis of osteoporosis: a position statement from the National Bone Health Alliance Working Group. Osteoporos Int 25: 1439–1443.
6. Greenspan SL, von Stetten SE, Emond SK, et al. 2001 Instant vertebral assessment: a noninvasive dual X-ray absorptiometry technique to avoid misclassification and clinical mismanagement of osteoporosis. J Clin Densitom 4:373–380.
7. Maghraoui El A, Rezqi A, Mounach A, et al. 2013 Systematic vertebral fracture assessment in asymptomatic postmenopausal women. Bone 52:175–180.
8. Vokes T, Bachman D, Baim S, et al. 2006 Vertebral fracture assessment: the 2005 ISCD official positions. J Clin Densitom 9:37–46.

9. Lewiecki EM, Laster A. 2006 Clinical review: clinical applications of vertebral fracture assessment by dual-energy x-ray absorptiometry. J Clin Endocrinol Metab 91:4215−4222.
10. Miller PD. 2012 Unrecognized and underappreciated secondary causes of osteoporosis. Endocrinol Metab Clin North Am 4:613−628.
11. Papaioannou A, Morin S, Cheung AM, et al. 2010 2010 clinical practice guidelines for the diagnosis and management of osteoporosis in Canada: summary. CMAJ 182:1864−1873.
12. Cosman F, de Beur SJ, LeBoff MS, et al. 2014 A clinician's guide to the prevention and treatment of osteoporosis. Osteoporos Int 25:2359−2381.
13. The International Society For Clinical Densitometry. 2013 ISCD official positions—adult. Available at: http://www.iscd.org/official-positions/2013-iscd-official-positions-adult/. 2013. The International Society For Clinical Densitometry. [Ref Type: Online Source]. Accessed: ■ ■, ■.
14. Watts NB, Bilezikian JP, Camacho PM, et al. AACE Osteoporosis Task Force. 2010 American Association of Clinical Endocrinologists medical guidelines for clinical practice for the diagnosis and treatment of postmenopausal osteoporosis. Endocr Pract 16(3 Suppl):1−37.
15. Delmas PD, van de Langerijt L, Watts NB, et al. 2005 Underdiagnosis of vertebral fractures is a worldwide problem: the IMPACT study. J Bone Miner Res 20:557−563.
16. McCloskey EV, Spector TD, Eyres KS, et al. 1993 The assessment of vertebral deformity: a method for use in population studies and clinical trials. Osteoporos Int 3:138−147.
17. Jackson SA, Tenenhouse A, Robertson L. 2000 Vertebral fracture definition from population-based data: preliminary results from the Canadian Multicenter Osteoporosis Study (CaMos). Osteoporos Int 11:680−687.
18. Waterloo S, Ahmed LA, Center JR, et al. 2012 Prevalence of vertebral fractures in women and men in the population-based Tromso Study. BMC Musculoskelet Disord 13:3.
19. Majumdar SR, Kim N, Colman I, et al. 2005 Incidental vertebral fractures discovered with chest radiography in the emergency department: prevalence, recognition, and osteoporosis management in a cohort of elderly patients. Arch Intern Med 165:905−909.
20. Fink HA, Milavetz DL, Palermo L, et al. 2005 What proportion of incident radiographic vertebral deformities is clinically diagnosed and vice versa? J Bone Miner Res 20:1216−1222.
21. Black DM, Arden NK, Palermo L, et al. 1999 Prevalent vertebral deformities predict hip fractures and new vertebral deformities but not wrist fractures. Study of Osteoporotic Fractures Research Group. J Bone Miner Res 14:821−828.
22. Delmas PD, Genant HK, Crans GG, et al. 2003 Severity of prevalent vertebral fractures and the risk of subsequent vertebral and nonvertebral fractures: results from the MORE trial. Bone 33:522−532.
23. Schousboe JT, Fink HA, Lui LY, et al. 2006 2006 Association between prior non-spine non-hip fractures or prevalent radiographic vertebral deformities known to be at least 10 years old and incident hip fracture. J Bone Miner Res 21(10): 1557−1564.
24. Lindsay R, Silverman SL, Cooper C, et al. 2001 Risk of new vertebral fracture in the year following a fracture. JAMA 285:320−323.
25. van Geel TA, Huntjens KM, van den Bergh JP, et al. 2010 Timing of subsequent fractures after an initial fracture. Curr Osteoporos Rep 8:118−122.
26. Johnell O, Kanis JA, Oden A, et al. 2004 Fracture risk following an osteoporotic fracture. Osteoporos Int 15: 175−179.
27. Geith T, Schmidt G, Biffar A, et al. 2014 Quantitative evaluation of benign and malignant vertebral fractures with diffusion-weighted MRI: what is the optimum combination of b values for ADC-based lesion differentiation with the single-shot turbo spin-echo sequence? Am J Roentgenol 203:582−588.
28. Cicala D, Briganti F, Casale L, et al. 2013 Atraumatic vertebral compression fractures: differential diagnosis between benign osteoporotic and malignant fractures by MRI. Musculoskelet Surg 97(2 Suppl):S169−S179.
29. MacLean C, Newberry S, Maglione M, et al. 2008 Systematic review: comparative effectiveness of treatments to prevent fractures in men and women with low bone density or osteoporosis. Ann Intern Med 148:197−213.
30. Stadhouder A, Buskens E, Vergroesen DA, et al. 2009 Nonoperative treatment of thoracic and lumbar spine fractures: a prospective randomized study of different treatment options. J Orthop Trauma 23:588−594.
31. Pfeifer M, Begerow B, Minne HW. 2004 Effects of a new spinal orthosis on posture, trunk strength, and quality of life in women with postmenopausal osteoporosis: a randomized trial. Am J Phys Med Rehabil 83:177−186.
32. Triantafyllopoulos IK, Lambropoulou-Adamidou K, Nacopoulos CC, et al. 2014 EMAS position statement: the management of postmenopausal women with vertebral osteoporotic fracture. Maturitas 78:131−137.
33. Blasco J, Martinez-Ferrer A, Macho J, et al. 2012 Effect of vertebroplasty on pain relief, quality of life, and the incidence of new vertebral fractures: a 12-month randomized follow-up, controlled trial. J Bone Miner Res 27:1159−1166.
34. Mudano AS, Bian J, Cope JU, et al. 2009 Vertebroplasty and kyphoplasty are associated with an increased risk of secondary vertebral compression fractures: a population-based cohort study. Osteoporos Int 20:819−826.
35. NICE. Percutaneous vertebroplasty and percutaneous balloon kyphoplasty for treating osteoporotic vertebral compression fractures. 2013. Available at: guidance.nice.org.uk/ta279. [cited 2014 Sept. 4]. Accessed: ■ ■, ■.
36. McClung M, Harris ST, Miller PD, et al. 2013 Bisphosphonate therapy for osteoporosis: benefits, risks, and drug holiday. Am J Med 126:13−20.
37. Khosla S, Bilezikian JP, Dempster DW, et al. 2012 Benefits and risks of bisphosphonate therapy for osteoporosis. J Clin Endocrinol Metab 97:2272−2282.
38. Harris ST, Watts NB, Genant HK, et al. 1999 Effects of risedronate treatment on vertebral and nonvertebral fractures in women with postmenopausal osteoporosis: a randomized controlled trial. Vertebral Efficacy With Risedronate Therapy (VERT) Study Group. JAMA 282:1344−1352.
39. Reginster J, Minne HW, Sorensen OH, et al. 2000 Randomized trial of the effects of risedronate on vertebral fractures in women with established postmenopausal osteoporosis. Vertebral Efficacy with Risedronate Therapy (VERT) Study Group. Osteoporos Int 11:83−91.
40. Black DM, Cummings SR, Karpf DB, et al. 1996 Randomised trial of effect of alendronate on risk of fracture in women with existing vertebral fractures. Fracture Intervention Trial Research Group. Lancet 348(9041):1535−1541.
41. Neer RM, Arnaud CD, Zanchetta JR, et al. 2001 Effect of parathyroid hormone (1-34) on fractures and bone mineral density in postmenopausal women with osteoporosis. N Engl J Med 344:1434−1441.
42. Cummings SR, San Martin J, McClung MR, et al. FREEDOM Trial. 2009 Denosumab for prevention of fractures in postmenopausal women with osteoporosis. N Engl J Med 361: 756−765.

43. Ettinger B, Black DM, Mitlak BH, et al. 1999 Reduction of vertebral fracture risk in postmenopausal women with osteoporosis treated with raloxifene: results from a 3-year randomized clinical trial. Multiple Outcomes of Raloxifene Evaluation (MORE) Investigators. JAMA 282:637−645.
44. Lyritis GP, Trovas G. 2002 Analgesic effects of calcitonin. Rev Bone 30(5 Suppl):71S−74S.
45. Chesnut CH 3rd, Silverman S, Andriano K, et al. 2000 A randomized trial of nasal spray salmon calcitonin in postmenopausal women with established osteoporosis: the prevent recurrence of osteoporotic fractures study. PROOF Study Group. Am J Med 109:267−276.
46. Overman RA, Borse M, Gourlay ML. 2013 Salmon calcitonin use and associated cancer risk. Ann Pharmacother 47: 1675−1684.
47. Nevitt MC, Chen P, Dore RK, et al. 2006 Reduced risk of back pain following teriparatide treatment: a meta-analysis. Osteoporos Int 17:273−280.
48. Miller PD. 2005 Bone density and markers of bone turnover in predicting fracture risk and how changes in these measures predict fracture risk reduction. Curr Osteoporos Rep 3: 103−110.
49. Civitelli R, Armamento-Villareal R, Napoli N. 2009 Bone turnover markers: understanding their value in clinical trials and clinical practice. Osteoporos Int 20:843−851.
50. Krege J, Lane N, Eastell R, Miller PD. 2014 PINP as a biological marker during treatment for osteoporosis. Osteoporos Int 25:2159−2171.
51. Siminoski K, Warshawski RS, Jen H, Lee K. 2006 The accuracy of historical height loss for the detection of vertebral fractures in postmenopausal women. Osteoporos Int 17:290−296.
52. Siminoski K, Jiang G, Adachi JD, et al. 2005 Accuracy of height loss during prospective monitoring for detection of incident vertebral fractures. Osteoporos Int 16:403−410.
53. Kanis JA, Svedbom A, Harvey N, McCloskey EV. 2014 The osteoporosis treatment gap. J Bone Miner Res 29:1926−1928.