AJO The American Journal of Orthopedics

Fragility Fractures: Diagnosis and Treatment

Publish date: December 19, 2018
Authors:
Lauren Bledsoe, RPAC Kaitlyn Alessi Jose B. Toro, MD Brian Giordano, MD Bryan T. Hanypsiak, MD
Author Affiliation | **Disclosures**

Ms. Bledsoe is a physician assistant, Naples Neprhology, Naples, Florida. Ms. Alessi is a biomedical engineering student, University of Florida, Gainesville. Dr, Toro is Orthopaedic Traumatologist, Orange Regional Medical Center, Middletown New York. Dr. Giordano is Associate Professor, Department of Orthopaedics and Rehabilitation, Division of Sports Medicine, University of Rochester, New York. Dr. Hanypsiak is Orthopedic Surgeon, Department of Orthopedic Surgery, Physician's Regional Medical Center, Naples, Florida, and Course Director, Innovative Techniques: Knee, Hip, and Shoulder Course, Las Vegas, Nevada.

Authors' Disclosure Statement: The authors report no actual or potential conflict of interest in relation to this article.

Address correspondence to: Bryan T. Hanypsiak, MD, Physicians Regional Hospital, 6101 Pine Ridge Road, Desk 23A, Naples FL 34119 (tel, 239-348-4221; email, bryan.hanypsiak@physiciansregional.com).

Am J Orthop. 2018;47(12).

Take-Home Points

- 3 million people sustain fragility fractures annually, and nearly 30% die within a year of the fracture.
- The incidence of fragility fractures increases in patients with comorbidities such as thyroid disease, diabetes, hypertension, and heart disease.
- The World Health Organization has developed a set of T-core criteria to diagnose osteoporosis in postmenopausal women: a score >–1 is normal; <–1 but >–2.5 signifies osteopenia; <–2.5 denotes osteoporosis; and <–2.5 with fragility fracture indicates severe osteoporosis.
- The Z score, not the T score, should be used to assess osteoporosis in premenopausal women, men <50 years, and children. The Z score is calculated by comparing the patient's BMD with the mean BMD of their peers of a similar age, race, and gender. Z scores <–2.0 indicate low BMD for chronological age. A Z score > –2.0 is considered within the expected range for age.
- After an initial fragility fracture, the risk for additional ones increases significantly, making treatment of osteoporosis essential. The National Osteoporosis Foundation recommends treating osteoporosis with pharmacotherapy in patients with a high risk for fracture (T score <–2.5) or history of fragility fracture.[26]

Fragility fractures are caused by falls from standing height or repetitive physiological loads.[1] With the growing aging population in the United States, it is estimated that 3 million people will be affected by fragility fractures yearly.[2] In the setting of osseous insufficiency, fractures that are typically associated with high-energy trauma are encountered in patients who simply trip over a parking lot curb or fall off their bike. After surgery, the severe disruption of patients' lives continues with a prolonged rehabilitation period.

Fragility fractures are not only traumatizing for patients; they are also associated with significantly increased mortality. A study by Gosch and colleagues found that 70.6% of patients died during the normal follow-up period, and 29.4% of patients died within the first year of suffering a fracture.[3] Also, the mean life expectancy post-fragility fracture was only 527 days.[3] Diagnosis and treatment of osteoporosis is imperative to prevent fragility fractures before they occur.

Risk Factors and Causes

The incidence of fragility fractures increases in patients with comorbidities such as thyroid disease, diabetes, hypertension, and heart disease.[4] Hyperthyroidism and treated hypothyroidism cause an imbalance between osteoblast and osteoclast activity, resulting in osteoporosis.[5] A thyroid-stimulating hormone level < 0.1 increases the risk of vertebral and non-vertebral fractures by a factor of 4.5 and 3.2 mIU/L respectively.[4] Patients with diabetes also have an increased risk of fragility fractures, which is due to impaired healing capabilities, especially that of bone healing. Approximately 2 million people are affected by type 1 diabetes in the United States, and 20% of those patients will develop osteoporosis.[6]

Hypertension and osteoporosis are 2 diseases that occur often in the elderly. Common etiological factors believed to cause both hypertension and osteoporosis are low calcium intake, high consumption of salt, and vitamin D and vitamin K deficiency. Also, hypertension treated with loop diuretics has been found to cause negative effects on bone and increase the risk of osteoporosis.[7] The only antihypertensive medications that preserve bone mineral density (BMD) and reduce fracture risk are thiazide diuretics.[7] Lastly, an association between coronary artery disease and osteoporosis has been hypothesized. The link is not completely understood, but it is believed that oxidative stress and inflammation are the culprits in both diseases.[8] In contrast to previous hypotheses, Sosa and colleagues found an independent association between beta blockers and fragility fractures.[9] The idea that beta blockers and fragility fractures are linked is still controversial and needs more study. Unlike beta blockers, statins provide a protective effect on bone. They increase BMD and reduce fracture risk by inhibiting osteoclastogenesis.[10]

In addition to loop diuretics and beta blockers, inhaled glucocorticoids, oral glucocorticoids, proton pump inhibitors (PPIs), H_2 receptor antagonists, and anticonvulsants decrease bone density and increase the incidence of fragility fractures.[11] Chronic glucocorticoid therapy is the most common cause of secondary osteoporosis. Osteoblasts and osteocytes undergo apoptosis in the presence of glucocorticoids.[12] Patients on glucocorticoid therapy have an increased risk of fracture, even with higher BMD values.[13] Bone changes that occur while a patient is taking glucocorticoids may not be detected during BMD testing. Therefore, a high level of suspicion of osteoporosis in patients on long-term glucocorticoids is imperative.

Proton pump inhibitors are among the most prescribed medications in the world; they reduce bone resorption, increasing the risk of fracture.[14] Proton pump inhibitors and H_2 receptor antagonists are hypothesized to cause malabsorption of calcium and indirectly cause osteoporosis. The risk of osteoporosis increases with the length of PPI treatment.[15] However, exposure lasting <7 years does not increase the risk of fracture.[16] It is recommended that patients on long-term PPIs be referred for BMD testing.

An association between anticonvulsants and osteoporosis has been found in observational studies. The mechanism of this association is not yet fully understood, but it is believed that exacerbation of vitamin D deficiency leads to increased bone metabolism.[17] Gastrointestinal (GI) calcium absorption also decreases with anticonvulsant use. Prolonged antiepileptic therapy and high-dose therapy rapidly decrease BMD. Primidone, carbamazepine, phenobarbital, and phenytoin are the drugs most often associated with decreased BMD. Osteoporosis and fragility

fracture in these patients can be prevented with calcium, vitamin D, and the bisphosphonate risedronate. These medications have been shown to improve BMD by 69%.[18]

Diagnosis

Osteoporosis is diagnosed by the presence of a fragility fracture or by dual-energy x-ray absorptiometry (DXA) in the absence of a fragility fracture.[19] Measurements of the femoral neck by DXA are used to diagnose osteoporosis, although DXA can also be used to measure the bone density of the spine and peripheral skeleton.[20]

The World Health Organization developed a set of T score criteria to diagnose osteoporosis in postmenopausal women (**Table 1**). A T score >-1 is normal, <-1 but >-2.5 signifies osteopenia, <-2.5 is osteoporosis, and <-2.5 with fragility fracture is severe osteoporosis.[19] The Z score, not the T score, should be used to assess osteoporosis in premenopausal women, men <50 years, and children (**Table 2**). The Z score is calculated by comparing the patient's BMD with the mean BMD of their peers of a similar age, race, and gender.[19] Z scores <-2.0 indicate low BMD for chronological age. A Z score > -2.0 is considered within the expected range for age.[20] Bone mineral density testing is the rate- limiting step to starting osteoporosis treatment.[21] Without testing, treatment of osteoporosis is very unlikely.

The World Health Organization also developed a tool to predict fracture risk. The Fracture Risk Assessment Tool uses fracture history in addition to other risk factors to predict a patient's 10-year risk of major fracture.[22] Risk factors used to assess fracture risk include age, sex, weight, height, previous fracture, parental hip fracture history, current smoker, glucocorticoid use, rheumatoid arthritis, secondary osteoporosis, excessive alcohol use, and femoral neck BMD.

In 2011, the United States Preventive Services Task Force (USPSTF) recommended that all women ≥65 years should be screened for osteoporosis by DXA. Women <65 years with a 10-year fracture risk =/> than that of a 65-year-old white woman should also be screened for osteoporosis. These recommendations are different for men. It was concluded that the evidence was insufficient to support osteoporosis screening in men.[23] As of April 2017, Centers for Medicare and Medicaid Services current reimbursement rates for DXA scans are, on average, $123.10 in the hospital setting and $41.63 in the office setting. The axial DXA CPT code is 77080.

Treatment

Nonpharmacologic

Patients with mild osteoporosis may be treated first non-pharmacologically. Lifestyle changes such as calcium and vitamin D supplementation, exercise, and smoking cessation are non-pharmacologic treatment options. Calcium carbonate and calcium citrate are common supplements. Calcium carbonate is 40% elemental calcium, whereas calcium citrate supplements are only 21% elemental calcium. Calcium supplements are best absorbed when taken with food.[24] The recommended daily total calcium intake is 1200 mg.[25] Only 500 to 600 milligrams of calcium can be absorbed by the GI tract at a time. Therefore, calcium supplements should be taken at least 4 to 5 hours apart.[24] Patients should also be counseled that calcium supplements may cause GI side effects such as bloating and constipation. To reduce side effects, patients can slowly increase the dose of calcium to a therapeutic level.

Vitamin D supplementation works best in conjunction with calcium supplementation. Vitamin D functions to regulate calcium absorption in the intestine and stimulate bone resorption and maintain the serum calcium

concentration. The National Osteoporosis Foundation recommends 800 to 1000 international units of vitamin D daily.[24] Lifestyle changes may be sufficient to stop the progression of osteoporosis in its early stages. Once osteoporosis becomes severe enough, pharmacotherapy is needed to stop further bone destruction and improve BMD.

Pharmacologic

After an initial fragility fracture, the risk of additional ones increases significantly, making treatment of osteoporosis essential. The National Osteoporosis Foundation recommends treating osteoporosis with pharmacotherapy in patients with a high risk of fracture (T score <-2.5) or history of fragility fracture.[26] Bisphosphonates inhibit bone resorption and are considered the first-line therapy for postmenopausal women with osteoporosis. A common side effect of oral bisphosphonates is GI toxicity. Patients are advised to avoid lying down for at least 30 minutes after medication administration to avoid esophageal irritation. Oral bisphosphonates should also be taken in the morning on an empty stomach with at least 8 ounces of water. Recurrent bisphosphonate use should be avoided in patients with chronic kidney disease. Oral alendronate and risedronate are typically discontinued after 5 years of use.[27] Long-term bisphosphonate use may cause an increased risk of fragility fracture due to oversuppression of bone turnover. To avoid this risk, bisphosphonate “drug holidays” are an option. Bisphosphonates accumulate over time, creating reservoirs. Even after therapy is stopped, patients continue to have therapeutic effects for 2 to 5 years.[28]

Bisphosphonates are available in both oral and intravenous forms. Alendronate is available in doses of 10 mg and 70 mg for daily and weekly administration, respectively. Both are available in tablet form, but the 70 mg weekly dose is also available in a dissolvable formulation. Alendronate is available in a reduced dose for osteoporosis prevention. Alendronate dosing for osteoporosis prevention is 5 mg daily or 35 mg weekly. Risedronate is dosed as 5 mg daily, 35 mg weekly, or 150 mg monthly. Intravenous bisphosphonates are indicated when oral bisphosphonates are not tolerated, only after vitamin D has been assessed and is within the normal range. Zoledronic acid is administered as a 15-minute infusion once a year.

Teriparatide (Forteo; PTH-1-34) is available for glucocorticoid-induced osteoporosis, postmenopausal women, and men with severe osteoporosis. It is indicated for patients in whom bisphosphonate treatment has failed or those who do not tolerate bisphosphonates. Teriparatide is a synthetic parathyroid hormone (PTH) that acts as an anabolic agent, stimulating bone formation, maturation, and remodeling.[29] In addition to its application as a bone-building hormone, teriparatide has gained popularity for various off-label uses. These include accelerated osteosynthesis, stress fracture healing, and in the nonoperative treatment of osteoarthritis.[29] Parathyroid hormone has been shown to stimulate the maturation, proliferation, and maintenance of osteoblast progenitor cells. More recently, PTH has been shown to regulate chondrocyte signaling, as well as differentiation and maturation. Further study on the chondroregenerative potential of PTH has demonstrated its efficacy as a novel disease-modifying agent in the treatment of osteoarthritis.[29] Teriparatide is administered as a daily subcutaneous injection. The United States dosing is 600 mcg/2.4 mL. Adverse effects such as orthostatic hypotension and osteosarcoma may occur. BMD testing should be performed 1 to 2 years after initiation of teriparatide and every 2 years thereafter.[26]

Abaloparatide (Tymlos), a human parathyroid hormone, is another treatment option for postmenopausal women at risk of osteoporotic fracture. In a study comparing the efficacy of abaloparatide and teriparatide, treatment with abaloparatide was found to induce higher BMD levels in a time frame of 12 months. The BMD differences could be attributed to many factors, such as an enhanced net anabolic effect or a reduced osteoblast expression. Furthermore, the risk of developing new vertebral and nonvertebral fractures decreased in the abaloparatide

group compared with the placebo group over a period of 18 months.[30]

The recommended daily dose for abaloparatide is 80 mcg via subcutaneous injection with calcium and vitamin D supplements.[31] Adverse reactions were consistent between abaloparatide and teriparatide, and included hypercalcemia, hypercalciuria, and orthostatic hypotension.[30] The use of parathyroid analogs for >2 years is not recommended due to the risk of osteosarcoma.

Denosumab (Prolia) is a monoclonal antibody that stops osteoclastogenesis by blocking the binding of RANKL to RANK.[31] It is indicated for patients intolerant to bisphosphonates or with impaired kidney function. Prolia is administered subcutaneously in 60 mg doses every 6 months in men and postmenopausal women with osteoporosis. Prolia is contraindicated in patients with hypersensitivity to any component of the medication, pregnancy, and hypocalcemia.

Selective estrogen receptor modulators (SERMs), such as raloxifene and tamoxifen, can treat osteoporosis effectively in postmenopausal women. Raloxifene is considered the SERM of choice due to the availability of more robust safety and efficacy data. Raloxifene increases BMD while decreasing bone resorption and bone turnover.[32] It is also used to reduce breast cancer risk; however, it increases the risk of thromboembolic events and hot flashes. Tamoxifen is not typically used to treat osteoporosis, but women treated for breast cancer with tamoxifen receive some bone protection.

Lastly, calcitonin and strontium ranelate are also options to treat osteoporosis. However, both calcitonin and strontium ranelate have weak effects on BMD. Calcitonin only transiently inhibits osteoclast activity.[33] Therefore, medications like bisphosphonates, teriparatide, denosumab, and SERMs are preferred.

A summary of medications used to treat osteoporosis can be found in **Table 3.**

Conclusion

With a growing aging population, the prevalence of osteoporosis is expected to increase. By 2025, experts estimate that there will be 2 million fractures yearly, costing the United States upwards of $25 billion.[34,35] This estimate does not include the cost of lost productivity or disability, which will likely cost billions more.[34,35] Understanding risk factors and eliminating medications known to cause decreased BMD are vital. Obtaining a BMD measurement is the rate-limiting step for treatment initiation. Without an appropriate diagnosis, treatment is unlikely. As providers, it us our responsibility to maintain a high level of suspicion of osteoporosis in the elderly and promptly diagnose and treat them.

Key Info

Figures/Tables

Figures / Tables:

Table 1. T Score Criteria

T score	Diagnosis
> -1.0	Normal
-1.0 to -2.5	Osteopenia
< -2.5	Osteoporosis
< -2.5 with fragility fracture	Severe osteoporosis

Table 2. Z Score Criteria

Z score	Diagnosis
> -2.0	Normal BMD for age
< -2.0	Low BMD for age

Table 3. Overview of Common Medications Used in the Treatment and Prevention of Osteoporosis

Medication	**Indication**	**Dosing**
Calcium supplementation	Mild osteoporosis	1200 mg oral/d
Vitamin D supplementation	Mild osteoporosis	800 to 1000 IU oral/d

Alendronate	Postmenopausal osteoporosis Osteoporosis prevention	10 mg oral/d 70 mg oral/wk 5 mg/d 35 mg/wk
Risedronate	Postmenopausal osteoporosis	5 mg oral/d 35 mg oral/wk 150 mg oral/mo
Teriparatide (Forteo)	Glucocorticoid-inducted osteoporosis, postmenopausal osteoporosis, men with severe osteoporosis	600 mcg/2.4 mL subcutaneous/d
Abaloparatide (Tymlos)	Postmenopausal osteoporosis	80 mcg subcutaneous/d
Denosumab (Prolia)	Patients intolerant to bisphosphonates; patients with impaired kidney function.	60 mg subcutaneous every 6 mo
Raloxifene	Postmenopausal osteoporosis	60 mg oral/d
Tamoxifen	Postmenopausal osteoporosis	20 mg oral/d
Calcitonin	Postmenopausal osteoporosis	100 units intramuscular or subcutaneous/d 200 units (1 spray) intranasal/d
Strontium ranelate	Postmenopausal osteoporosis Severe osteoporosis in men	2 g/d dissolved in water, prior to bedtime Not recommended in CrCl <30 mL/min

Abbreviation: CrCl, creatinine clearance.

References

References

1. Dietz SO, Hofmann A, Rommens PM. Haemorrhage in fragility fractures of the pelvis. *Eur J Trauma Emerg Surg*. 2015;41:363-367. doi: 10.1007/s00068-014-0452-1
2. Burge R, Dawson-Hughes B, Solomon DH, Wong JB, King A, Tosteson A. Incidence and economic burden of osteoporosis-related fractures in the United States, 2005-2025. *J Bone Miner Res*. 2007;22(3):465-475. doi: 10.1359/jbmr.061113.
3. Gosch M, Hoffmann-Weltin Y, Roth T, Blauth M, Nicholas JA, Kammerlander C. Orthogeriatric co-management improves the outcome of long-term care residents with fragility fractures. *Arch Orthop Trauma Surg*. 2016; 136(10):1403-1409. doi: 10.1007/s00402-016-2543-4.
4. Maccagnano G, Notarnicola A, Pesce V, Mudoni S, Tafuri S, Moretti B. The prevalence of fragility fractures in a population of a region of southern Italy affected by thyroid disorders. *BioMed Res Int*. 2016. doi: 10.1155/2016/6017165.
5. Mosekilde L, Eriksen EF, Charles P. Effects of thyroid hormones on bone and mineral metabolism. *Endocrinol Metab Clin North Am*. 1990;19(1):35-63. doi: 10.1016/S0889-8529(18)30338-4.
6. Liporace FA, Breitbart EA, Yoon RS, Doyle E, Paglia DM, Lin S. The effect of locally delivered recombinant human bone morphogenic protein-2 with hydroxyapatite/tri-calcium phosphate on the

biomechanical properties of bone in diabetes-related osteoporosis. *J Orthop Traumatol.* 2015;16(2):151-159. doi: 10.1007/s10195-014-0327-6.
7. Ilic K, Obradovic N, Vujasinovic-Stupar N. The relationship among hypertension, antihypertensive medications, and osteoporosis: a narrative review. *Calcif. Tissue Int*. 2013;92(3):217-227. doi: 10.1007/s00223-012-9671-9.
8. Yesil Y, Ulger, Z, Halil M, et al. Coexistence of osteoporosis (OP) and coronary artery disease (CAD) in the elderly: it is not just a by chance event. *Arch Gerontol Geriatr*. 2012;54(3):473-476. doi: 10.1016/j.archger.2011.06.007.
9. Sosa M, Saavedra P, de Tejada MJG, et al, GIUMO Cooperative Group. Beta-blocker use is associated with fragility fractures in postmenopausal women with coronary heart disease. *Aging Clin Exp Res.* 2011;23(3):112-117. doi: 10.3275/7041.
10. An T, Hao J, Li R, Yang M, Cheng G, Zou M. Efficacy of statins for osteoporosis: a systematic review and met-analysis. *Osteoporos Int*. 2017;28(1):47-57. doi: 10.1007/s00198-016-3844-8.
11. Munson JC, Bynum JP, Bell J, et al. Patterns of prescription drug use before and after fragility fracture. *JAMA Intern Med*. 2016;176(10):1531-1538. doi: 10.1001/jamainternmed.2016.4814.
12. Saag KG, Agnesdei D, Hans D, et al. Trabecular bone score in patients with chronic glucocorticoid therapy-induced osteoporosis treated with alendronate or teriparatide. *Arthritis Rheumatol*. 2016;68(9):2122-2128. doi: 10.1002/art.39726.
13. Chuang MH, Chuang TL, Koo M, Wang YF. Trabecular bone score reflects trabecular microarchitecture deterioration and fragility fracture in female adult patients receiving glucocorticoid therapy: A pre-post controlled study. *BioMed Res Int.* 2017. doi: 10.1155/2017/4210217.
14. Andersen BN, Johansen PB, Abrahamsen B. Proton pump inhibitors and osteoporosis. *Curr Opin Rheumatol*. 2016;28(4):420-425. doi: 10.1097/BOR.0000000000000291.
15. Jacob L, Hadji P, Kostev K. The use of proton pump inhibitors is positively associated with osteoporosis in postmenopausal women in Germany. *Climacteric*. 2016; 19(5):478-481. doi: 10.1080/13697137.2016.1200549.
16. Targownik LE, Lix LM, Metge CJ, Prior HJ, Leung S, Leslie WD. Use of proton pump inhibitors and risk of osteoporosis-related fracture. *Can Med Assoc J*. 2008;179:319-326. doi: 10.1503/cmaj.071330.
17. Lee RH, Lyles KH, Colon-Emeric C. A review of the effect of anticonvulsant medications on bone mineral density and fracture risk. *Am J Geriatr Pharmacother.* 2010;8(1):34-46. doi: 10.1016/j.amjopharm.2010.02.003.
18. Arora E, Singh H, Gupta YK. Impact of antiepileptic drugs on bone health: Need for monitoring, treatment, and prevention. *J Family Med Prim Care*. 2016;5(2):248-253. doi: 10.4103/2249-4863.192338.
19. Maghraoui AE, Roux C. DXA scanning in clinical practice. *Q J Med*. 2008;101(8):605-617. doi: 10.1093/qjmed/hcn022.
20. Watts NB, Lewiecki EM, Miller PD, Baim S. National osteoporosis foundation 2008 clinician's guide to prevention and treatment of osteoporosis and the world health organization fracture risk assessment tool (FRAX): What they mean to the bone densiometrist and bone technologist. *J Clin Densitom*. 2008;11(4):473-477. doi: 10.1016/j.jocd.2008.04.003.
21. MacLean C, Newberry S, Maglione M, et al. Systematic review: comparative effectiveness of treatments to prevent fractures in men and women with low bone density or osteoporosis. *Ann Intern Med*. 2007;148(3):197-213. doi: 10.7326/0003-4819-148-3-200802050-00198.
22. Beaton DE, Vidmar M, Pitzul KB, et al. Addition of a fracture risk assessment to a coordinator's role improved treatment rates within 6 months of screening in a fragility fracture screening program. *J Am Geriatr Soc*. 2017; 28(3):863-869. doi: 10.1007/s00198-016-3794-1.

23. U.S. Preventative Services Task Force. Screening for osteoporosis. *Ann Intern Med.* 2011;154(5):356-364. doi: 10.7326/0003-4819-154-5-201103010-00307.
24. Sunyecz JA. The use of calcium and vitamin D in the management of osteoporosis. *Ther Clin Risk Manag*. 2008;4(4):827-836.
25. Eastell, R. (1998). Treatment of postmenopausal osteoporosis. *N Engl J Med*. 1998;338:736-746. doi: 10.1056/NEJM199803123381107.
26. Cosman F, de Beur SJ, LeBoff MS, et al, National Osteoporosis Foundation. Clinician's guide to prevention and treatment of osteoporosis. *Osteoporos Int*. 2014;25(10):2359-2381. doi: 10.1007/s00198-014-2794-2.
27. Black DM, Schartz AV, Ensrud KE, et al, doi:10.1001/jama.296.24.2927.
28. Schmidt GA, Horner KE, McDanel DL, Ross MB, Moores KG. Risks and benefits of long-term bisphosphonate therapy. *Am J Health Syst Pharm*. 2010;67(12):994-1001. doi: 10.2146/ajhp090506.
29. Kraenzlin, ME, Meier C. Parathyroid hormone analogues in the treatment of osteoporosis. *Nat Rev Endocrinol*. 2011;7(11):647-656. doi: 10.1038/nrendo.2011.108.
30. Miller P, Hattersley G, Riis B, et al. Effect of abaloparatide vs placebo on new vertebral fractures in postmenopausal women with osteoporosis. *JAMA*. 2016;316(7):722-733. doi: 10.1001/jama.2016.11136.
31. TYMLOS™ [prescribing information]. Waltham, MA: Radius Health, Inc; 2017.
32. Tetsunaga T, Tetsunaga T, Nishida K, et al. Denosumab and alendronate treatment in patients with back pain due to fresh osteoporotic vertebral fractures. *J Orthop Sci*. 2017;22(2):230-236. doi: 10.1016/j.jos.2016.11.017.
33. Recker, RR, Mitlak BH, Ni X, Krege JH. Long-term raloxifene for postmenopausal osteoporosis. *Curr Med Res Opin*. 2011;27(9):1755-1761. doi: 10.1185/03007995.2011.606312.
34. Yildirim K, Gureser G, Karatay S, et al. Comparison of the effects of alendronate, risedronate and calcitonin treatment in postmenopausal osteoporosis. *J Back Musculoskelet Rehabil.* 2005;18(3/4):85-89. doi: 10.3233/BMR-2005-183-405.
35. Christensen L, Iqbal S, Macarios D, Badamgarav E, Harley C. Cost of fractures commonly associated with osteoporosis in a managed-care population. *J Med Econ.* 2010;13(2):302-313. doi: 10.3111/13696998.2010.488969.

Multimedia

Product Guide

Product Guide

- STRATAFIX™ Symmetric PDS™ Plus Knotless Tissue Control Device
- STRATAFIX™ Spiral Knotless Tissue Control Device
- BioComposite SwiveLock Anchor
- BioComposite SwiveLock C, with White/Black TigerTape™ Loop

×
×

Citation

Lauren Bledsoe, RPAC Kaitlyn Alessi Jose B. Toro, MD Brian Giordano, MD Bryan T. Hanypsiak, MD . Fragility Fractures: Diagnosis and Treatment . Am J Orthop.
Publish date: December 19, 2018

Lauren Bledsoe, RPAC