CLINICAL UPDATE

Acute Vertebral Fracture

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Vertebral fractures resulting from osteoporosis are a significant cause of morbidity and mortality in the aging population and are commonly seen in the hospital setting. Appropriately assessing and treating these conditions continues to be a challenge. This article is an evidence-based clinical update on the evaluation and management options of acute vertebral fractures, ranging from conservative treatment to surgical intervention. Hospitalists can play an influential role in the management of osteoporosis. *Journal of Hospital Medicine* 2009;4:E20–E24. © 2009 Society of Hospital Medicine.

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An 89-year-old female presents to the Emergency Department with lower back pain for the past 5 days. The patient has a past medical history of polymyalgia rheumatica and hypothyroidism. Her medications include prednisone 10 mg daily and levothyroxine 50 μ g daily. Aside from tenderness over the third lumbar vertebra, her physical exam is unremarkable. An x-ray shows a fracture of the third lumbar vertebra. Basic laboratory studies, including calcium and creatinine are normal.

The Clinical Problem and Impact

Vertebral fractures (often termed vertebral compression fractures) affect approximately 25% of all postmenopausal women.^{1,2} Only one-third of vertebral fractures are brought to medical attention.^{3,4} In the remaining two-thirds, patients are either asymptomatic or do not seek medical attention. The lifetime risk of a clinical vertebral fracture is approximately 16% and 5% in white women and men, respectively.¹ The risk of vertebral fracture increases with age, lower bone mineral density (BMD), and prior vertebral fracture.^{1,5} Women with a preexisting vertebral fracture have a 5-fold increased risk for a new vertebral fracture relative to those without a history of vertebral fracture.^{5,6} Approximately 20% of women who sustain a vertebral fracture will have a new vertebral fracture in the subsequent year.^{5,6} Vertebral fractures are frequently overlooked on chest x-rays and hence there is a need for increased awareness and improved recognition of radiographically-demonstrated fractures.⁷ Hospitalists must appreciate that a vertebral fracture is often the first clue to underlying osteoporosis. At least 90% of vertebral and hip fractures are attributable to osteoporosis.⁵

Typically, the pain related to an acute vertebral fracture improves over 4 to 6 weeks.^{8,9} However, pain can persist, resulting in functional impairment, and a decline in the quality of life.^{10–13} Vertebral fractures may lead to kyphosis and reduced lung function. This, in turn, may increase the

risk for pneumonia, the most common cause of death in patients with osteoporosis.⁴ Both clinical and subclinical vertebral fractures are, in fact, independently associated with increased mortality,^{4,14,15} particularly in the period immediately following the event.¹⁶ The economic impact of osteoporosis and its related fractures is substantial.^{17–19} In 1995, the annual direct medical cost for the inpatient care of vertebral fractures was estimated to be \$575 million.¹⁹

Evidence-Based Approach to the Hospitalized Patient Evaluation

Vertebral fractures most commonly occur between T7 and L4. Acute vertebral fracture pain is typically sudden in onset and located in the mid to lower back. The pain may occur while performing an ordinary task such as lifting an object or bending over, although in many cases there is no preceding trauma. Physical activity exacerbates the pain and patients' movements may be limited due to pain. Spinal tenderness is usually present. A history of weight loss, prior malignancy, or fever should serve as red flags to the hospitalist and prompt evaluation for underlying malignancy or infection.

For a suspected fracture, frontal and lateral radiographs of the thoracolumbar spine are the initial imaging of choice. Magnetic resonance imaging (MRI) is useful when infection, malignancy, or spinal cord compression is suspected. The presence of neurological deficits should always prompt imaging with MRI or computed tomography (CT).

Patients with vertebral fractures should be evaluated with bone density testing, as osteoporosis is usually the underlying etiology. Because a number of medical conditions commonly contribute to bone loss (Table 1), laboratory testing is indicated for most patients. Although debate exists as to the optimal testing strategy,²⁰ the National Osteoporosis Foundation guidelines recommend the evaluation of blood count, chemistry, and thyroid-stimulating hormone (TSH)

TABLE 1. Secondary CauseDensity	s of Low Bone Mineral
Endocrine disease or	Hypogonadism
metabolic causes	Cushing's syndrome
	Hyperthyroidism
	Anorexia nervosa
	Hyperparathyroidism
Nutritional conditions	Vitamin D deficiency
	Calcium deficiency
	Malabsorption
Drugs	Glucocorticoids

Antiepileptic drugs Excessive thyroid medication

Multiple myeloma Rheumatoid arthritis Organ transplantation

Long-term heparin or low molecular weight heparin therapy (eg, >1 month)



in patients with osteoporosis.²¹ Because vitamin D deficiency is common in patients who sustain osteoporotic fractures, 25-hydroxyvitamin D levels should be checked in most patients.^{22–25} Depending on the clinical scenario, additional testing may include the following: serum testosterone, serum intact parathyroid hormone (PTH), 24-hour calcium excretion, serum protein electrophoresis, urine protein electrophoresis, erythrocyte sedimentation rate, and celiac sprue antibodies.²⁶

Management

Short Term

Other

Short-term management goals include the relief of pain and recovery of mobility. Nonsteroidal antiinflammatory drugs (NSAIDs) and low-dose opioid medications should be used first in an effort to relieve pain. Beyond its potential effect on bone density, calcitonin (Miacalcin, Fortical) has long been used in acute vertebral fractures for analgesia. A systematic review of 5 randomized controlled trials concluded that calcitonin significantly reduced the pain from acute vertebral fractures.²⁷ Calcitonin improved pain as early as 1 week into treatment and the benefit was persistent at 4 weeks. The analgesic mechanism of action for calcitonin is not entirely clear. Proposed mechanisms include increased β-endorphin release, an antiinflammatory effect, and a direct effect on specific receptors in the central nervous system.27,28

Pamidronate (Aredia) also has shown efficacy at reducing acute fracture pain. In a double-blinded trial, Armigeat et al.²⁹ evaluated pamidronate 30 mg daily for 3 days as compared to placebo in 32 patients. Pain scores were improved at 7 and 30 days with pamidronate. The mechanism of analgesia is unclear. Bisphosphonates are known to inhibit osteoclast activity, but may also work by blocking the effect of inflammatory cytokines.²⁹

FIGURE 1. Kyphoplasty. (A) In kyphoplasty, a cannula is placed into the collapsed vertebra, through which an inflatable bone tamp is inserted into the vertebral body. (B) The bone tamp is inflated, and (C) the cavity is filled with polymethylmethacrylate cement. (D) The hardened cement forms an internal cast. [Adapted from Mazanec et al.³⁶ with permission]

Early pain relief is critical in order to encourage physical activity. Bed rest should be avoided, as immobility may increase the risk for pressure ulcers, venous thromboembolism, and pneumonia.^{30–33} Although bracing is frequently used in acute vertebral fracture, the modality has not been formally studied. Although also not well studied in the acute setting, physical therapy has been shown to reduce pain and improve functioning for patients with chronic pain from vertebral fracture,³⁴ and is generally recommended.³⁵

Percutaneous vertebral augmentation procedures include vertebroplasty and kyphoplasty. In vertebroplasty, polymethylmethacrylate cement is injected through a needle under fluoroscopic guidance into the collapsed vertebral body. With kyphoplasty, balloon tamps are used to elevate vertebral endplates prior to injection of cement (Figure 1).³⁶ The proposed mechanism of action for both procedures is stabilization of the fracture by the hardened polymethylmethacrylate cement. These procedures are commonly performed by interventional radiologists without the need for general anesthesia; however, depending on the institution, they may be done by orthopedic surgeons, neurosurgeons, or anesthesiologists. The procedure can be performed as an outpatient, if indicated. Although the volume of these procedures has grown dramatically in recent years,^{37,38} the quality of

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evidence supporting their use is relatively weak.³⁹⁻⁴¹ Only 1 randomized controlled trial has been published evaluating the potential benefit of vertebroplasty over conservative management.⁴² Voormolen et al.⁴² evaluated patients with vertebral fractures and pain refractive to 6 weeks of optimal medical therapy. Patients were treated with vertebroplasty or continuation of medical therapy. Vertebroplasty significantly improved pain initially, but not after 2 weeks. Like the study by Voormolen et al.,42 most studies evaluating percutaneous vertebral augmentation procedures have been conducted on patients with long-term pain refractory to medical management. One notable exception is a nonrandomized trial published by Diamond et al.⁶ In that study, 55 patients were treated with vertebroplasty while 24 were treated conservatively. Pain at 24 hours was significantly improved in patients treated with vertebroplasty. At 6 weeks, however, there was no difference among the 2 groups.

The risk of short-term complications from vertebral augmentation procedures is difficult to assess in light of the small sample sizes and methodological limitations of existing studies. Cement leakage occurs in 40% to 41% of patients treated with vertebroplasty, as compared with 8% to 9% with kyphoplasty.39,41 Pulmonary emboli occur in 0.6% and 0.01% of patients treated with vertebroplasty and kyphoplasty, respectively, while neurologic complications occur in 0.6% and 0.03% of patients.⁴¹ Concern exists about whether percutaneous vertebral augmentation procedures might increase the risk for subsequent fractures,^{43,44} as the incidence of new fractures appears to be elevated in the period immediately following the procedure and approximately two-thirds of new fractures occur in vertebrae adjacent to the augmented vertebra.^{39,41,44} However, the 20% incidence of new vertebral fractures in the year following vertebral augmentation is similar to the fracture rate seen in patients not treated with osteoporosis therapy.44

Assessment of the impact of vertebral augmentation procedures on the cost of care is limited by the lack of highquality clinical studies.^{45,46} Randomized controlled trials evaluating the benefit and risk of these procedures compared to conservative management are underway.^{47–49} Pending further evidence, these procedures are best reserved for patients who fail to benefit from other measures to control pain and improve mobility.

Long Term

A comprehensive discussion of the long-term management of osteoporosis is beyond the scope of this work. However, the inpatient setting presents an opportune time to initiate long-term medical therapy. Studies show that the majority of patients who sustain osteoporotic fractures do not receive pharmacologic treatment for osteoporosis.^{50–53} Hospitalists have the opportunity to start medications that can reduce the risk for subsequent fracture by nearly 50%.^{54–58} A total calcium intake of 1200 to 1500 mg per day and vitamin D of 400 to 800 IU per day are recommended for all postmeno-

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pausal women. Patients who smoke should receive smoking cessation counseling and be considered for pharmacologic treatment for tobacco dependence. All patients should be assessed for fall risk, including a review of medications and assessment of alcohol intake.

Before considering pharmacologic treatment for osteoporosis, secondary causes of low bone mass must be excluded. Bisphosphonates are generally considered first-line pharmacologic therapy for osteoporosis. Alendronate (Fosamax), risedronate (Actonel), and ibandronate (Boniva) have been shown in randomized trials to increase bone density and reduce the risk of osteoporotic fractures.^{55,56,59} Daily, weekly, and monthly preparations of bisphosphonates now exist. Pill-induced esophagitis is a potential adverse effect of bisphosphonate therapy, but is extremely rare if proper precautions are taken. Patients should take oral bisphosphonates on an empty stomach, with a full glass of water, sitting upright, and have nothing to eat or drink for at least one half hour. If compliance with oral bisphosphonates is not possible, or esophageal abnormalities preclude oral bisphosphonate use, one may consider the use of intravenous ibandronate or zoledronic acid (Reclast). A 3-year randomized controlled trial of yearly zoledronic acid improved bone density and reduced the incidence of osteoporotic fractures.⁶⁰ Bisphosphonates are generally not recommended when creatinine clearance is less than 30 mL/ minute. Other pharmacologic options for the treatment of osteoporosis include selective estrogen receptor modulators and anabolic agents. The reader is referred to an excellent review by Rosen⁶¹ for additional discussion of these therapies. The American College of Rheumatology clinical guidelines for the management of glucocorticoid induced osteoporosis are also worthy of review.62

Hospitalists are naturally suited to improve the quality of care for patients hospitalized with vertebral fractures. Most patients who currently sustain osteoporotic fractures do not receive appropriate evaluation and treatment. One study used an interdisciplinary team to identify, assess, and begin treatment for appropriate patients hospitalized with osteoporotic fractures.⁶³ The intervention resulted in significantly more patients taking osteoporosis treatment medications 6 months after the incident fracture.

Conclusions

Acute vertebral fracture is a common clinical problem associated with significant morbidity and increased risk of mortality. Treatment of vertebral fracture should include analgesics and physical therapy. Percutaneous augmentation procedures may be considered in patients who fail optimal medical therapy. Because most vertebral fractures are due to osteoporosis and the healthcare system currently fails to appropriately assess and treat most patients who have sustained osteoporotic fractures, hospitalists are in an optimal position to initiate long-term preventative treatment for these patients.

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