Contemporary Concepts in Spine Care

Osteoporotic compression fractures of the spine; current options and considerations for treatment

David H. Kim, MD\textsuperscript{a,*}, Alexander R. Vaccaro, MD\textsuperscript{b}

\textsuperscript{a}The Boston Spine Group, Department of Orthopaedic Surgery, Tufts University Medical School, New England Baptist Hospital, 125 Parker Hill Avenue, Boston MA 02120, USA

\textsuperscript{b}Department of Orthopaedic Surgery, Thomas Jefferson University and The Rothman Institute, 925 Chestnut Street, Philadelphia, PA 19107, USA

Received 18 April 2006; accepted 18 April 2006

Abstract

BACKGROUND CONTEXT: Vertebral compression fractures affect at least one-fourth of all postmenopausal women. The most significant risk factor is osteoporosis, most commonly seen among Caucasian women a decade or so after menopause. Osteoporosis typically results from inadequate accumulation of bone mass during childhood and early adulthood followed by rapid resorption after menopause. Primary treatment of osteoporosis includes consideration of underlying metabolic abnormalities and provision of supplemental calcium/vitamin D in conjunction with bisphosphonates or calcitonin, or both. Routine hormone replacement therapy has fallen out of favor because of concerns regarding adverse effects identified in long-term follow-up studies. Acute osteoporotic vertebral compression fracture management includes bracing, analgesics, and functional restoration. Patients with chronic pain beyond 2 months may be appropriate candidates for vertebral body augmentation, ie, vertebroplasty or balloon tamp reduction. Open surgical management with decompression and stabilization should be reserved for the rare patient with neural compression and progressive deformity with neurologic deficits.

PURPOSE: To review current principles in the evaluation and treatment of osteoporotic compression fractures of the spine.

STUDY DESIGN/SETTING: A literature review on management of the osteoporotic spine.

METHODS: MEDLINE search of all English-language literature published between 1981 and 2005 on surgical and nonsurgical treatment of the osteoporotic spine. The references selected for listing at the conclusion of this review are those containing specific information cited within the text.

RESULTS: Over 200 separate scientific and clinical studies addressing the epidemiology, pathophysiology, diagnosis, and treatment of osteoporotic vertebral compression fractures were reviewed.

CONCLUSIONS: Osteoporotic vertebral compression fractures are a common presenting complaint to spinal care specialists. Thorough differential diagnosis should be considered before attributing fractures to osteoporosis. Appropriate evaluation and medical treatment of underlying osteoporosis should be recommended or instituted. Nonsurgical management of the spinal fracture should focus on pain control and maximizing functional outcome. The role of surgical treatment remains controversial and should be reserved for patients who fail initial nonsurgical management options. © 2006 Elsevier Inc. All rights reserved.

Keywords: Osteoporosis; Osteoporotic vertebral compression fracture (OVCF); Vertebroplasty; Balloon tamp reduction

Introduction

Osteoporosis is the most common metabolic disorder of bone, affecting approximately 100 million people worldwide. In the United States, it is estimated that at least 10 million people suffer from osteoporosis and an additional 18 million people are at significant risk for development of the disorder. Within this affected group, approximately 700,000 vertebral body compression fractures occur each year, twice the rate of hip fractures. Approximately 70,000 compression fractures result in hospitalization each year with an average hospital stay per patient of 8 days. It has been estimated that at least 25% of American women reaching menopause will experience at least one osteoporotic vertebral compression fracture (OVCF) in their lifetime [1]. Although considered a women’s health issue, osteoporosis also affects 33% of men by age 75 [2]. For any given patient, the diagnosis of a single OVCF increases the risk of subsequent fractures by a factor of five. Patient population studies suggest an increased mortality rate in patients with OVCFs that correlates with the number of involved vertebrae [3]. A benign natural history has long been assumed for OVCFs, but up to 30% of those who are symptomatic and seek treatment do not respond adequately to nonsurgical treatment [4,5].

Historic background

As recently as 1925, osteoporosis was relatively unrecognized and its metabolic causes were unknown. By the early 1980s, estrogen and calcium supplementation had become widely prescribed medical treatments for this condition. Over the past 20 years, additional medications including bisphosphonates, calcitonin, and parathormone have been introduced and successfully used in both treatment and prevention of osteoporosis [6,7].

Osteoporotic vertebral compression fractures have traditionally been considered benign injuries that heal rapidly without much in the way of complications. Investigators [8] in 1981 advocated horizontal bed rest for 7 to 10 days followed by judicious bracing and gradual mobilization. It has become increasingly clear through more rigorous follow-up of these patients that the favorable prognosis of OVCFs is hardly universal. Recognition of poor outcomes has motivated development of surgical solutions to chronic pain in this patient population.

Anatomy/Pathophysiology

Osteoporosis is characterized by decreased bone mass or increased porosity and results in diminished structural support of the osseous spinal column. Primary osteoporosis is considered to have two etiologic variants. Type I affects postmenopausal women and is related to rapid loss of bone after menopause, whereas Type II affects individuals over 70 years old and involves age-related loss of bone or senile osteoporosis [9]. Secondary osteoporosis is loss of bone caused by an agent or disease process such as corticosteroids, endocrine disorders, or an inflammatory process [10,11].

Conceptually, primary osteoporosis may also be subdivided into high and low turnover states. High turnover osteoporosis implies enhanced osteoclastic bone resorption with the inability of osteoblast activity to maintain an equilibrium mineralization state. This is thought to be the primary etiology of Type I postmenopausal osteoporosis. Low turnover osteoporosis, commonly seen in the elderly, represents a primary deficiency of osteoblast activity to form bone with typically normal or even slightly subnormal osteoclastic activity.

With greater understanding of the pathophysiology and subtype variations of osteoporosis, pharmacologic treatment has become more specific and effective. High turnover states can be identified by high levels of collagen crosslink degradation products (N-telopeptide and pyridinoline peptide) and can be treated with a host of antiresorptive agents. These medications most commonly include the hormone replacement drugs (estrogen or raloxifene), the bisphosphonates, and calcitonin. Adverse effects of long-term hormone replacement therapy have significantly reduced the popularity of this form of treatment, and it is no longer routinely recommended for preventive treatment of chronic disease. Bisphosphonates have a significant incidence of gastrointestinal side effects that limit their usefulness in many patients but have been shown to significantly reduce the incidence of new vertebral fractures by almost 50% [12]. Likewise, calcitonin has recently been shown to reduce new OVCF risk by approximately one-third in women [13], with apparent specific efficacy in increasing bone density in the spine [14]. Low-turnover osteoporosis identified by decreased alkaline phosphatase and low collagen crosslink degradation products can be addressed with pharmaceutical agents such as the newly approved parathormone analog. Calcium and vitamin D supplementation remains a mainstay in the treatment and prevention of nearly all patients with osteoporosis.

Risk factors

In general, the risk of osteoporotic related fracture increases significantly with age for both men and women. The lifetime risk of all types of skeletal fractures for Caucasian women older than 50 years of age approaches 75%. The lifetime risk of clinically significant OVCF is 16% in this group. In women, the risk of OVCF increases sixfold from menopause to age 85 [10]. Women with a history of one OVCF have a fivefold risk of developing subsequent compression fractures [15].

The World Health Organization has developed a definition of osteoporosis using dual energy X-ray absorptiometry as a means of defining bone mass. If measurement reveals bone mass between 1 and 2.5 standard deviations below average
peak bone mass, the patient is considered to have osteopenia or mild to moderate bone deficiency. However, individuals with measurements greater than 2.5 standard deviations below average peak bone mass are considered to be osteoporotic with marked bone deficiency. Individuals already diagnosed with osteoporotic fragility fractures are by definition considered to have severe osteoporosis. A number of other associated risk factors have also been identified as predictive of the likelihood of incurring osteoporotic fragility fractures, including low body weight, recent weight loss, history of such fractures in the family, and a history of smoking. Age itself has been found to be an independent risk factor for fracture exclusive of bone density [16].

The identification of diminished bone mineral density with any of the aforementioned risk factors predicts a significantly greater risk of insufficiency fracture. The American Academy of Orthopaedic Surgery recommends bone mineral density scanning to rule out osteoporosis in all Caucasian women >65 years old and all postmenopausal women with one or more risk factors or who have sustained a fracture. Likewise, it is recommended that medical management of presumed osteoporosis be instituted in all postmenopausal women diagnosed with a hip or vertebral fracture [17].

Clinical research

Clinical presentation

Most patients with identified OVCF are asymptomatic at the time of initial diagnosis, and the age of the fracture may be impossible to determine. Patients presenting with back pain typically report the sudden or acute onset of pain in temporal relationship with relatively atraumatic activities such as bending forward, standing from a seated position, or even with vigorous coughing or sneezing. Pain may not be localized to the site of the fracture, as thoracolumbar fractures often present with low back or lumbosacral pain. Upon physical examination, the patient’s general appearance may reveal focal kyphosis or loss of lumbar lordosis. Multiple osteoporotic vertebral compression fractures can lead to noticeable loss of height. It has been suggested that if the patient’s fingertips extend to the lower thigh or knee during standing, then pathologic spinal shortening should be suspected [18]. In the acute stages, localized tenderness over the involved level is often present and is a useful means of identifying an acute fracture [19]. However, localized tenderness does not necessarily indicate significant injury to the posterior elements and does not distinguish between anterior and posterior column involvement.

Radiography

Radiographic evaluation of vertebral compression fractures may demonstrate the classic “wedge” fracture, which shows loss of anterior vertebral body height with relative preservation of posterior vertebral body height. The most frequent site of involvement is the thoracolumbar junction, with the second most frequent region being the midthoracic spine. Sagittal alignment typically demonstrates increased segmental kyphosis. However, in the absence of obvious vertebral deformity, acute vertebral pain may indicate a nondisplaced stress fracture, and bone scans or magnetic resonance imaging (MRI) may be required to make a definitive diagnosis [9].

Distinguishing acute from chronic fractures on plain radiographs can be difficult. Features indicating an acute injury include well-demarcated fracture lines or distinct discontinuity of a thin cortical margin. A more chronic injury is suggested by sclerosis of the fracture lines, a dense cortical margin, and osteophytes bordering the fracture site.

Because these fractures most frequently occur in the elderly, multilevel degenerative spondylosis is commonly present. Degenerative disc space narrowing may result in relative shortening and laxity of the functional posterior tension band structures predisposing to increased anterior column compression stress and a two-column pseudo-instability pattern. This clearly places the weakened osteoporotic vertebral body at risk for fracture anteriorly and may well be one of the structural factors predisposing to not only the original injury but also development of chronic debilitating symptoms.

Multiple classification systems have been proposed without general acceptance of a single system. Sugita et al. recently reviewed 135 cases of OVCF and developed a classification system based on lateral radiographic views comprising five distinct types: (a) “swelled-front” in which >50% of the anterior cortex was convex anteriorly; (b) the “bow-shaped” in which the anterior cortex was pinched inward and the superior end plate was collapsed; (c) “projecting,” in which <50% of the anterior cortex was convex anteriorly; (d) “concave,” in which the superior end plate was collapsed but the anterior cortex was intact; and (e) “dented,” in which the anterior cortex demonstrated a fracture stepoff. Both concave and dented types demonstrated good prognosis with relatively short duration of pain, rapid healing, and low rates of collapse [20]. The remaining types were associated with poorer prognosis and high rates of vacuum cleft formation as identified by gas-like radiolucency in the collapsed vertebra during extension stress views in the lateral decubitus position. Vacuum clefts were only observed in fractures of the thoracolumbar junction and required an average of 1.5 years to achieve bone union.

It should be noted that previous classification systems do not account for the important phenomenon of “dynamic fracture mobility” in which significant height restoration and sagittal plane deformity correction can be observed radiographically with supine or lateral decubitus extension positioning alone. This property directly affects the natural history of fractures and response to treatment. The presence of intravertebral clefts is associated with dynamic fracture mobility and should be looked for during patient evaluation. Clefts characteristically occur in the
with predictable pain improvement over 6 to 8 weeks, some patients experience persistent pain and disability. A study of patients with multiple osteoporotic vertebral compression fractures found significant decreases in trunk extension torque, spinal motion, functional reach, mobility skills and walking distance compared with the normal age-matched population [26]. Of potentially greater concern are studies suggesting increased mortality rates in patients with OVCF with a 23% to 34% increase in mortality over an 8-year period in one study [27]. In general, these mortality rates are not directly related to fracture. Rather, OVCF is a marker for medical frailty. In uncommon cases, death has been attributed to pulmonary dysfunction resulting from reduced intra-abdominal capacity with resultant limitation of diaphragmatic excursion [28].

**Nonsurgical treatment**

The most important treatment principle in osteoporosis is prevention. Two goals of prevention are to ensure attainment of peak premenopausal bone mass and prevent postmenopausal resorption. However, once osteoporosis is clearly recognized, active treatment needs to be instituted to reduce the risk of pathological fracture. Postmenopausal women with osteoporosis should be treated with 1,500 mg of supplemental calcium and 400 IU of vitamin D daily. All men with compression fractures should be considered for serum testosterone testing because of the high incidence of hypogonadism in this population [18]. Any patient with an osteoporotic fracture and an elevated alkaline phosphatase should be suspected of having osteomalacia [29]. Cigarette smoking should be discouraged and alcohol consumption allowed only in moderation. A practical weight-bearing daily exercise regimen should be recommended to improve generalized trunk and lower extremity strength and to improve balance with the goal of preventing falls. Estrogen therapy has been shown to increase bone mass by approximately 2% per year [30], but when estrogen is terminated, there is a rapid bone loss with complete loss of all benefit after 7 years. More importantly, a large prospective randomized trial, the Women’s Health Initiative, has demonstrated that hormone replacement therapy is associated with increased rates of coronary artery disease, breast cancer, stroke, venous thromboembolism, and cholecystitis; and routine prescription of this treatment is no longer recommended for chronic disease prevention, including osteoporosis [31]. Newer medicines include bisphosphonates such as alendronate which has been shown to significantly reduce the incidence of new vertebral fractures by almost 50% [32]. At least 40 prospective randomized trials have been reported involving 15 different pharmacologic agents with specific reference to an effect on vertebral fracture risk [33]. Both alendronate and risedronate have demonstrated efficacy in multiple studies. Other agents with clinical evidence supporting their efficacy include raloxifene, parathormone, and calcitonin. Inhaled calcitonin has excellent patient tolerance and has been shown...
to increase spinal bone density and decrease new vertebral fracture risk by approximately 37% [34] (Table 1).

Treatment of acute fractures may necessitate a short period of bed rest for no longer than a few days, followed by gradual mobilization. Continuous hyperextension bracing with a spinal orthosis may be beneficial for up to the first 6 to 8 weeks until the acute pain resolves. Unfortunately, bracing is often poorly tolerated in elderly patients, and its efficacy in preventing further vertebral collapse has not been established. Appropriate analgesic medications should be prescribed to reduce pain and allow daily living activities. First-line medications include acetaminophen, salicylates, or nonsteroidal anti-inflammatory medication. Concerns regarding the latter two categories of medications involve the potential for inhibition of bone healing. Narcotics should be reserved for patients failing to obtain adequate relief with the above medications and activity modification. A major concern with narcotics is a more significant side effect profile including high rates of gastrointestinal dysmotility and cognitive deficits such as loss of balance and the potential for increased falls, injury, and depression [9]. Although drug dependence and abuse are always concerns, treatment of acute pain may involve a relatively lower risk of developing narcotic addiction. When the pain has sufficiently subsided, the patient should begin a carefully designed rehabilitation and exercise program, including elements of fall prevention and education regarding spinal biomechanics and preservation. In this setting, back extension exercises have demonstrated superiority over abdominal flexion exercises in terms of achieving lower incidence of new fractures [35].

**Vertebral body augmentation**

Patients with chronic pain after vertebral compression fracture have traditionally had few treatment options. However, new vertebral body augmentation techniques offer these patients the attractive alternative of a minimally invasive

---

Table 1

<table>
<thead>
<tr>
<th>Class</th>
<th>Medication</th>
<th>Mechanism of action</th>
<th>Trade name</th>
<th>Indications</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bisphosphonates</td>
<td>Alendronate</td>
<td>Antiresorptive; inhibits osteoclast activity by inducing osteoclast apoptosis</td>
<td>Fosamax</td>
<td>Prevention and treatment and postmenopausal osteoporosis, treatment of glucocorticoid-induced osteoporosis, treatment of osteoporosis in men</td>
<td>Prevention: 5 mg/d or 35 mg/wk Treatment: 10 mg/d or 70 mg/wk or 70 mg/wk+D vitamin 150 mg/month</td>
</tr>
<tr>
<td></td>
<td>Ibandronate</td>
<td></td>
<td>Bonvia</td>
<td>Prevention and treatment of postmenopausal osteoporosis</td>
<td>5 mg/d of 35 mg/wk or 35 mg/wk + calcium</td>
</tr>
<tr>
<td></td>
<td>Risedronate</td>
<td></td>
<td>Actonel</td>
<td>Prevention and treatment of postmenopausal osteoporosis</td>
<td></td>
</tr>
<tr>
<td>Calcitonin</td>
<td>Calcitonin</td>
<td>Parathyroid hormone decreases skeletal release of calcium, phosphorus, and hydroxyproline; antiresorptive; decreases bone loss, increases bone density; may reduce pain from insufficiency fracture; reduces risk of spine fracture</td>
<td>Miacalcin, Calcimar, Fortical</td>
<td>Prevention and/or treatment of postmenopausal osteoporosis</td>
<td>Injection 50–100 IU/d or nasal spray 200 IU/d</td>
</tr>
<tr>
<td>Estrogen</td>
<td>Estrogen</td>
<td>Antiresorptive; specific mechanism of action obscure; reduces bone loss; increases bone density; reduces risk of spine fracture</td>
<td>Multiple brand names available</td>
<td>Prevention of osteoporosis</td>
<td>Pill or skin patch, 0.3 mg/d (low dose) or 0.625 mg/d (standard dose)</td>
</tr>
<tr>
<td>Parathyroid Hormone</td>
<td>Teriparatide</td>
<td>Stimulates new bone formation; possibly by maintaining viability of osteocytes; increases intestinal calcium absorption; decreases urinary calcium excretion; increases production of 2-vitamin D-3; increases bone mineral density; reduces risk of spine fracture in women and men</td>
<td>Forteo</td>
<td>Treatment of postmenopausal osteoporosis; treatment of men at high risk for fracture</td>
<td>Daily injection for up to 24 months</td>
</tr>
<tr>
<td>Selective estrogen receptor modulators (SERMs)</td>
<td>Raloxifene</td>
<td>Antiresorptive; increases bone mass; reduces risk of spine fracture</td>
<td>Evista</td>
<td>Prevention of treatment or postmenopausal osteoporosis</td>
<td>60 mg/d</td>
</tr>
<tr>
<td>Mineral supplement</td>
<td>Calcium</td>
<td></td>
<td></td>
<td></td>
<td>1000–1500 mg/d</td>
</tr>
<tr>
<td>Vitamin supplement</td>
<td>Vitamin D</td>
<td></td>
<td></td>
<td></td>
<td>Up to 400 IU/d</td>
</tr>
</tbody>
</table>
procedure with relatively low risk and high—at least short-term—clinical success rates. Because fracture biomechanics are primarily determined by disruption of the trabecular microarchitecture [36,37] and ongoing symptoms may be related to ineffective structural repair and motion, acrylic cement support often yields predictable symptomatic relief.

Vertebroplasty

Originally developed in France in the late 1980s, vertebroplasty was introduced in the United States in 1994 [38,39]. This procedure is now being performed in an outpatient setting or associated with an overnight hospital admission. An attempt at postural reduction is performed by careful prone positioning on the operating room table. A 10- or 11-gauge needle is then introduced percutaneously into the involved vertebra using either a transcervical or posterolateral extrapedicular approach. Fluoroscopy is used to guide needle and sheath placement, and a mixture of polymethylmethacrylate (PMMA) and barium is injected under direct fluoroscopic visualization. Great care is taken to avoid cement extravasation posteriorly into the spinal canal or neuroforamen, the most commonly observed significant complication of this procedure. Cement migration anterolaterally into the paraspinal soft tissue is observed more commonly but has not been associated with significant clinical effects. If central venous filling is observed, the injection is temporarily halted to prevent pulmonary embolization of cement, a much rarer but potentially fatal complication. The mechanism of pain relief after vertebroplasty is most commonly through fracture stabilization; although it is possible that thermal and chemical ablation of nerve endings in the vertebral body may also contribute to pain relief.

The optimal volume of PMMA for vertebroplasty remains undetermined. Biomechanical studies have indicated that 2 mL is required for fractured osteoporotic vertebral to regain prefracture strength. Four to eight mL is required to restore prefracture stiffness. To date, no correlation has been established between injection volume and clinical outcome. However, some investigators have suggested that a balance exists between injecting enough PMMA to restore strength, stability, and relieve pain and injecting large amounts that may lead to excessive increases in stiffness with increased risks of adjacent level fracture [24].

The rate of PMMA leakage is controversial and appears somewhat technique-dependent, but is rarely associated with clinical sequelae [40]. On the high end, radiculopathy rates of 4% and cord compression rates of less than 0.5% have been reported [41]. Overall, when PMMA extravasation is included along with postoperative infection and worsened pain, complication rates for vertebroplasty are typically reported as less than 5%. Deaths have been reported in association with treatment of several levels in one procedure as a result of pulmonary embolization of PMMA. The effect of vertebral augmentation procedures on adjacent levels is controversial. It has been suggested that increased segmental stiffness as a result of vertebroplasty may be associated with an increase in the relative risk of adjacent level fracture [42]. However, this phenomenon may be related more to diminished adjacent level bone density rather than to increased biomechanical stresses imposed by prior vertebral augmentation [43]. In one small retrospective study of patients undergoing vertebroplasty, cement leakage into the intervening disc was associated with an increased risk of adjacent level fracture of 58% during 1-year follow-up compared with 12% when such leakage did not occur [44].

Recurrent pain after apparently successful vertebroplasty has been reported as occurring in up to 25% within the first 6 months of treatment [44]. In many cases, a new fracture is identified at another vertebral level. Occasionally, no new abnormality is recognized, and the possibility of recurrent pain at the treated level must be considered. Although the underlying mechanism remains unclear, re-fracture around the PMMA cement or inadequate fracture healing are possibilities. In these cases, plain radiographs may reveal increased collapse, bone scans may demonstrate increased tracer uptake, and magnetic resonance and computed tomographic imaging may reveal diffuse edema. In a retrospective study of 250 vertebroplasty patients, six patients with recurrent pain meeting the above criteria were identified [45]. Repeat vertebroplasty was associated with greater than 50% pain reduction in two-thirds of these patients.

Proposed contraindications to vertebroplasty include absence of pain, severe cardiopulmonary disease, infection, and uncorrectable coagulopathy. Relative contraindications include severe rigid vertebral collapse, significant retroplacation of bone into the spinal canal, or presence of a neurologic deficit [46]. Clinical success rates for vertebroplasty in terms of pain relief and functional improvement have been reported between 78% and 90% for vertebral compression fractures [46–49].

Balloon tamp reduction

The technique of balloon tamp reduction is similar to vertebroplasty, but includes the step of percutaneous insertion of a balloon tamp through the pedicle into the involved vertebral body. Balloon inflation purportedly achieves compaction of surrounding cancellous bone and varying degrees of elevation of the compressed vertebra. Potential advantages of balloon tamp reduction over vertebroplasty include the possibility of improved deformity correction and decreased potential for cement leakage.

Rates of pain relief and functional improvement with short-term follow-up have been at least equivalent to vertebroplasty [50,51]. Higher rates of kyphosis reduction are reported to occur if patients are treated within 3 months of the actual fracture [41,52]. However, the amount of kyphosis...
reduction related specifically to postural positioning as opposed to balloon elevation of the vertebral end plates is unclear. Studies have reported 90% satisfactory pain relief and functional improvement after this procedure. Significant complications have been reported at a rate of 0.7% per level treated and include hypoxia, epidural hematoma, paraparesis, and anterior cord syndrome.

Dynamic fracture mobility

Dynamic fracture mobility has been observed in a significant percentage of osteoporotic vertebral compression fractures and is defined as any measurable change in vertebral body height between standing lateral radiographs and cross-table lateral supine radiographs as observed by unaided vision [53]. The amount of change varies significantly among patients and can allow substantial height restoration with vertebroplasty alone. A retrospective review of 73 fractures treated with vertebroplasty revealed significant improvements in kyphosis and vertebral wedge angle averaging 19% and 44%, respectively [54]. A 29% average anterior height restoration was observed. Height restoration was more pronounced in fractured vertebra containing gas. These findings make it unclear to what degree balloon tamp reduction provides additional height restoration beyond what is achievable through dynamic fracture mobility alone. A well-designed randomized prospective study comparing the two techniques would be beneficial and should control for the phenomenon of dynamic fracture mobility. An unanswered question is whether there is any benefit from “height restoration” apart from cement injection in patients undergoing these treatments.

Surgical management

Indications for surgical intervention in the setting of osteoporotic compression fractures have not been strictly defined. The current consensus includes progressive neurological loss, severe unrelenting pain, and significant deformity.

Neurologic deficit typically occurs in association with spinal canal compromise resulting from “two-column” fracture associated with retropulsion of bone into the canal in association with severe deformity. These patients have traditionally been treated with anterior decompression either through a thoracotomy or retroperitoneal approach and reconstruction of the involved level using a structural graft. In a retrospective study [19] of 497 osteoporotic vertebral compression fracture patients, 10 presented with neurologic deficits and evidence of spinal cord compression and underwent anterior decompression and stabilization procedures. Although all patients regained independent ambulatory status postoperatively, none regained full lower extremity strength. Bowel and bladder function was reportedly satisfactory.

Surgical treatment of the osteoporotic spine is among the most challenging tasks in spinal surgery. Reliable fixation may be difficult to achieve, and structural grafts frequently subside in weak osteoporotic bone. Careful patient selection is the critical factor in achieving satisfactory surgical results. Preoperative health status must be carefully considered. Anterior surgical approaches are valuable in achieving decompression of the spinal canal or releasing anterior tethering structures in order to accomplish kyphosis correction; however, anterior instrumentation has been associated with a high rate of failure when used in isolation. A combined anterior and posterior procedure may maximize the chances for successful fusion, especially with multiple points of spinal fixation and occasionally with PMMA augmentation. Vertebral body augmentation combined with stable anterior column constructs may provide satisfactory long-term stability.

Posterior instrumentation has traditionally used sublaminar hooks, although hook migration and laminar fracture may occur in osteoporotic bone. Pedicle screw fixation provides an attractive alternative, providing three-column purchase at multiple points of fixation (multiple vertebral bodies above and below the fracture). Sublaminar hooks or wires can be used to further support in load share with pedicle screws at the proximal or distal end of constructs where the risk of pullout is greatest.

Discussion

Although most OVCFs are benign, a subset may cause significant morbidity and cost to society both economically and in decreased quality of life and productivity of our growing elderly population. Medical advances have allowed for more focused and effective treatment of osteoporosis. Prevention is the most important strategy with recognition and treatment of osteoporosis and bone loss before the fracture occurrence. Once a fracture has been diagnosed, nonsurgical management with activity modification and symptomatic medication, with or without bracing, is adequate for a majority of patients. In those who do not achieve acceptably rapid symptomatic relief with nonsurgical treatment, the judicious use of vertebral augmentation by vertebroplasty or balloon tamp reduction may provide more rapid pain relief although long-term outcomes have not been defined. At present, augmentation is performed with PMMA, which provides excellent compressive strength and immediate mechanical support. However, PMMA has a number of drawbacks because of a significant modulus mismatch with adjacent vertebrae that may lead to increased stresses at the augmented/nonaugmented junction. In larger volumes, this has been implicated as a cause for increased risk for adjacent level fractures. Biomechanical standards and safety guidelines for the use of PMMA combined with opacifying agents have not been determined for vertebral augmentation procedures [55]. Furthermore,
bone cement cures by exothermic reaction which may contribute to local tissue necrosis.

The exact indications for vertebral body augmentation of these fractures remain unclear. Certainly it is reasonable to consider vertebral augmentation in nonsurgically treated patients who fail to improve over 8 to 12 weeks after fracture. It should be kept in mind that although patients who are within 6 weeks of their fracture have the best outcome with regard to fracture reduction, many of these patients will improve clinically with nonsurgical care.

Future topics

The long-term natural history of osteoporotic vertebral compression fractures requires more detailed study. Longer term outcome studies following specific treatments such as vertebroplasty, balloon tamp reduction, and more traditional surgical reconstruction will allow refinement of appropriate indications for these different surgical procedures. Newer materials for augmentation are being introduced and may provide more favorable biomechanical properties and decreased toxicity. There may also be a role for prophylactic vertebral augmentation with biologic agents that locally improve bone density and strength.

Conclusions with key points

Osteoporotic vertebral compression fractures are becoming increasing sources of significant pain and disability as the population ages. Preventive measures remain the most clinically effective and cost-effective. When fractures occur, most patients experience rapid symptomatic resolution over the course of several weeks and require only nonsurgical treatment measures such as activity limitation, pain medication, and sometimes bracing. For those experiencing persistent pain, vertebral augmentation may provide significant symptom relief. Correctly or incorrectly, the technical aspects of these procedures are perceived as relatively easy to master, which likely contributes to their appeal. However, the role of these relatively new procedures has not been clearly defined and requires more rigorously controlled long-term follow-up studies to demonstrate efficacy in terms of long-term functional improvement in treated patients.

References

[43] Carlson GD, Smith JS, Gordon CD. Is there increased risk of adjacent segment vertebral compression fracture after kyphoplasty. Presented at: Annual Meeting Poster Presentation of the American Academy of Orthopaedic Surgeons; February 13–17, 2002; Dallas, TX.