Vertebral Compression Fractures

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ABSTRACT

Fragility fractures, particularly in the hip and spine, are the most common complication of osteoporosis. In the US, approximately 1–1.5 million vertebral compression fractures (VCFs) occur annually. While patients may present with sudden onset of low back pain and limited mobility, more than two-thirds of VCFs are asymptomatic and are detected incidentally. X-rays are the standard imaging modality for diagnosis, with CT and MRI indicated if neurological deficits are present or a malignant cause is considered. Initial management is often non-surgical, with medications, physical therapy, and bracing. Surgical management in the form of cement augmentation (kyphoplasty or vertebroplasty) or instrumented fusion can be considered after failure of non-operative treatment, cases of deformity, or neurologic deficits. Subsequent VCFs occur frequently, and risk factors for refracture include advanced age, low bone mineral density, and low BMI. Treatment of primary VCFs with anti-resorptive medication is essential to reduce the risk of subsequent fractures.

KEYWORDS: vertebral compression fractures; osteoporosis; secondary fractures

INTRODUCTION

Osteoporosis is the most commonly encountered metabolic bone disease, which affects 200 million people worldwide.[1] The disease is defined as a progressive loss of bone mineral density (BMD) as measured by dual-energy x-ray absorptiometry (DEXA). A score more than 2.5 standard deviations below the population average (T-score) indicates osteoporosis.[2] Due to the drop in estrogen after menopause and the consequent imbalance between bone resorption and formation, postmenopausal women have the greatest risk of developing osteoporosis.[3–5] Other risk factors include malignancy, low BMI, use of steroid medication, use of alcohol or tobacco, physical inactivity, and calcium deficiency.[2,6–8]

VCFs are the most reported fragility fracture in patients with osteoporosis. Approximately 1 to 1.5 million VCFs occur each year in the US, with an incidence rate of 40% in women over 80 years old.[9,10] With an aging population, the incidence of VCF will continue to grow, and therefore clinicians should be mindful of the presentation and management of these patients. Furthermore, patients with VCFs are at high risk of subsequent fractures, and it is important to consider bone density optimization for these patients and reduction of the risk factors for the development of additional fragility fractures. Previous studies have demonstrated that one prior VCF increases the risk of subsequent VCFs by 5-fold, and 2 previous VCFs increases the risk of future VCFs by 12-fold.[11] Analysis of data across 373 centers found that among 381 patients who had a VCF, the incidence of a new VCF in the following year was 19.2%.[12] A systematic review investigated the risk factors of secondary fractures after vertebroplasty, which included history of prior fractures, advanced age, reduced bone marrow density, and bone cement leakage.[13] Low BMI and the number of treated vertebrae were also established as moderate risk factors for refracture in another systematic review.[14]

CLINICAL FEATURES

The most common cause of a VCF is osteoporosis, although a diagnosis of malignancy should be considered in patients under 50 years old without history of trauma.[15] Patients with osteoporosis may develop a VCF after minor events, including coughing, sneezing, and lifting. In patients with severe osteoporosis, an estimated 30% of fractures occur when the patient is in bed.[11,16]

Risk factors for VCF can be modifiable or non-modifiable, which can guide clinicians in lifestyle optimization and identifying higher-risk patient groups. Nonmodifiable factors include advanced age greater than 70 years, female sex, history of steroid use, and Caucasian race. Modifiable factors include alcohol and tobacco use, physical inactivity, low BMI, and dietary deficiency of calcium and vitamin D.[11] The first step in preventing VCFs is the management of modifiable risk factors including treatment for osteoporosis.

The classically described symptom of a VCF is sharp or dull pain that is aggravated by movement or positional changes.[11] In many patients, this pain can be mild and attributed to another cause. Furthermore, 66% of patients with osteoporotic VCFs are asymptomatic, and their VCFs are discovered incidentally when imaging studies are performed for other reasons.[17] Red flags which may suggest a pathological

fracture (e.g., due to malignancy) include weight loss, other systemic symptoms, and persistent non-resolving pain.[18] The physical examination is often normal in patients, but midline tenderness with percussion over the spine and excessive thoracic kyphosis can indicate the presence of a VCF.[19] Kyphotic deformity with loss of height is more commonly seen with multiple fractures.[20] Neurological deficits, such as sensory or motor deficits, tend to be rare in osteoporotic VCF patients with minimal trauma as these fractures do not typically cause retropulsion of bony fragments into the vertebral canal.[11] The presence of neurological deficits should prompt evaluation with an MRI and possibly CT, and consideration of a more severe fracture or pathologic process.

The majority of VCFs occur in the mid-thoracic or thoracolumbar zone of the spine.[20] In patients with severe kyphotic deformity, pressure of the thoracic cavity on the pelvis and abdomen can result in reduced pulmonary function, atelectasis and pneumonia, and decreased appetite resulting in poor nutrition.[11] The osteoporotic thoracic kyphotic deformity frequently results in a restrictive pattern of pulmonary function. Lombardi et al reported that, when compared to women with osteoporosis alone, women with osteoporotic vertebral compression fractures had a lower forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV1).[21] Furthermore, the severity of the kyphotic angle has been demonstrated to negatively correlate with predicted FVC and FEV1.[22] One study revealed that patients with osteoporosis had a lower FVC when compared to a control group of patients with chronic low back pain, with the reduction in lung function correlating with clinical measures of osteoporosis including height reduction.[23] A systematic review by Harrison et al identified four case-control studies that established an association between osteoporosis-related kyphosis and reduction in vital capacity.[24] These studies had several limitations, however, and the authors recommended future investigation with standardized outcome measures and longitudinal follow-up. Harrison et al note that pulmonary function tests are reported in comparison to reference values (based upon age, gender, and height) which can have variations depending on the formulas utilized. Additionally, kyphosis can be measured by height, rib-pelvic, or wall-occiput measurements, as well as radiographically.[24]

The restrictive component of thoracic kyphosis may lead to detrimental respiratory complications. Lee et al performed a retrospective review of 51 patients with thoracic hyper-kyphosis who visited the respiratory department.[22] Of these patients, 35 were hospitalized due to respiratory complications including lower respiratory tract infection, acute respiratory failure, and exacerbation of a chronic airway disease. For patients with severe hyperkyphosis, surgical intervention may improve pulmonary function with younger patients exhibiting greater improvements in FEV1 compared to older patients.[25] Similarly, patients with osteoporotic vertebral compression fractures demonstrated partial improvement in lung function subsequent to kyphoplasty.[26]

Other complications of vertebral compression fractures include chronic pain, constipation, increased risk of venous thrombosis, and prolonged immobility, which can result in reduced functional ability and psychological issues.[10,16,27,28] In addition to these complications, VCFs also have a detrimental burden on healthcare expenditure, with an annual medical cost of $746 million per year in the United States (**Table 1**).[11]

Table 1. Symptoms and complications of vertebral compression fractures

Symptoms
• Sudden onset low back pain, which can occur after a low energy event such as sneezing or turning in bed
• Increased pain while walking or standing
• Limited spinal mobility
Complications
• Chronic pain
• Kyphosis, predominantly thoracic
• Height loss
• Loss of mobility: Resulting in pressure sores, risk of deep venous thrombosis, pneumonia, and psychological distress
• Gastrointestinal complications: Including constipation which can cause subsequent decreased appetite, nausea, and poor nutrition
• Decreased pulmonary function: Leading to pneumonia and exacerbation of chronic airway disease

(Adapted from reference 28 with permission.)

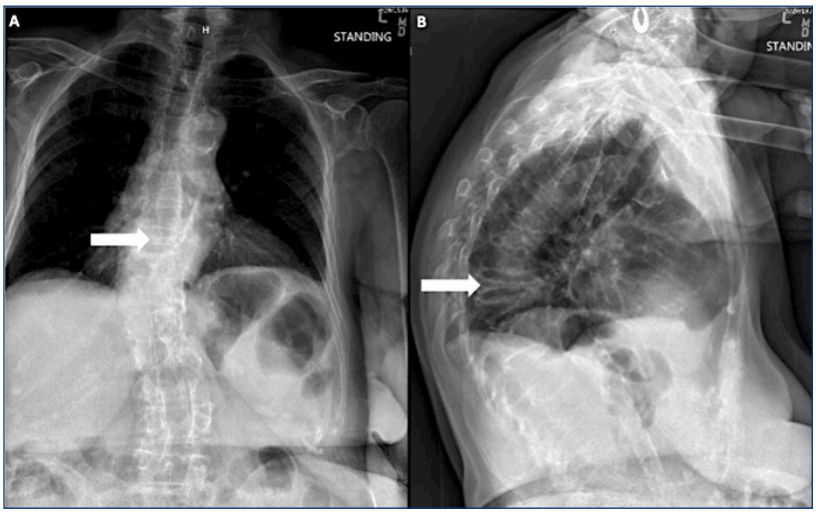
IMAGING

History and physical examinations, including a neurological assessment to evaluate arms, legs, and bladder and bowel function are the initial steps in evaluation, followed by imaging. Compression fractures can often be diagnosed with plain radiography, including lateral and anteroposterior views.[27] Clinicians should have a low threshold for imaging studies since inciting events are often low-energy and more than two-thirds of patients are asymptomatic.[27] If not previously recorded, DEXA scans should be acquired soon after the diagnosis of a VCF to evaluate for underlying osteoporosis and determine disease severity.[27]

A normal radiograph of the vertebral column should demonstrate a similar size and shape of the vertebrae across adjoining levels with horizontal endplates. A VCF is characterized by a reduction in vertebral height of 20%, or 4-mm loss from the baseline.[27] The Genant classification is commonly utilized to grade vertebral fractures based upon their morphology and height loss. Loss of height is graded from 0 (normal) to 3 (severe fracture), and morphology is reported as wedge, biconcave, or crush (**Figure 1**).[29]

Advanced imaging (CT or MRI) is rarely required but may be indicated to differentiate between benign versus malignant and acute versus chronic fractures.[15,27] Patients with

Figure 1. A: AP and **B**: lateral radiographs showing an osteoporotic compression fracture (arrows).



new or progressing neurological deficits merit advanced imaging to identify a retropulsed fracture where a bony fragment extends to the spinal column causing compression. MRI is typically the imaging of choice, as the characteristic signal intensities and enhancement patterns are well described for malignancy and a more recent fracture will demonstrate edematous changes. Radiological guidance should be sought when there are diagnostic concerns. For example, the intra-trabecular hemorrhage in an acute fracture may mimic a malignant cause and require further interpretation.

MANAGEMENT

Treatment for a VCF can be non-surgical or surgical. The goals of management are to achieve adequate pain relief, restore mobility, and prevent future fractures through addressing the underlying cause. In most cases, this involves careful evaluation of bone health and optimization of osteoporosis. Clinicians should discuss the benefits and risks of non-surgical and surgical treatment with a consideration for patient preferences and co-morbidities.

Non-surgical

Pain is a common presenting symptom of VCFs, and patients can describe this as intense.[30] Achieving adequate pain relief is important to prevent prolonged bed rest and encourage early mobility.[20] Although many patients experience pain relief over the first 6–8 weeks as fracture healing occurs, some patients have chronic pain.[31]

Subsequent to a VCF, a number of different medications can be used for pain relief including non-steroidal anti-inflammatory drugs (NSAIDs), opioids, calcitonin, and muscle relaxants.[27] NSAIDs are a common first-line therapy due to their ease of accessibility and low cost. Despite their effectiveness and overall safety, patients should be aware of risks, including peptic ulceration, gastrointestinal bleeding, and kidney disease.[20,27] This class of medication should also be used carefully in the elder population who generally have reduced creatinine clearance and are less tolerant of NSAIDs. Whenever possible, the patient's primary physician should be involved in the decision to use this class of medication. When NSAIDs are insufficient or contraindicated, opioids and muscle relaxants can be beneficial, but their use in the geriatric population is also cautioned due to sedative effects, constipation, nausea, and potential for dependency.[20,27] Calcitonin is a medication that has been used in the past for osteoporosis treatment, but also can provide acute relief of bone pain. A systematic review investigating its use in VCFs found strong efficacy for the management of acute back pain, but insufficient evidence for chronic pain due to older fractures.[30] Calcitonin is available intranasally and adverse effects include dizziness, flushing, and gastrointestinal disturbance.[27] Additionally, the use of calcitonin may be limited due to its relatively higher cost.[32] A review of US, UK, and Canadian national guidelines found inconsistent guidance on the use of these medications, with several stating weak evidence.[10]

For non-pharmacologic options, patients pursuing non-surgical management may consider the use of bracing, physical therapy, and nerve root blocks.[27] Physical therapy can strengthen the axial musculature and improve posture, which will assist with early mobilization and reduce the long-term likelihood of falls.[20] Rehabilitative exercise is also beneficial for all osteoporotic patients. Bracing can be used for a period of 4–12 weeks, although the evidence for its effectiveness is limited.[9,27] Braces are also not without risks and can cause muscular atrophy and deconditioning when used for an extended period.[20]

Patients may commence with a trial of non-surgical management, but careful follow-up should ensue to monitor for pain relief or progression of symptoms, as well as to observe for adverse effects.

Surgical

Although patients may commence with non-surgical treatment, clinicians must be aware of the indications for surgical management. An immediate referral to a surgeon is merited if a patient complains of leg weakness or pain, which indicates neurological deficit and demands further evaluation.[33] Furthermore, if patients exhibit no improvement in their pain level and disability over 6 weeks of conservative therapy, then surgical management should be considered.[27,33]

Kyphoplasty and vertebroplasty are percutaneous cement-augmentation techniques to manage symptomatic

VCFs. These are both minimally invasive procedures where bone cement is injected into the fractured vertebral body.[9] The procedure can be performed either inpatient or outpatient depending on individual patient characteristics. Several specialties can perform kyphoplasty and vertebroplasty, including surgical specialties (orthopedic surgery and neurosurgery) and non-surgical specialties (anesthesia, pain medicine, and radiology). Recent trends suggest that cement-augmentation procedures are being performed increasingly by non-surgeons.[34] The indication for these procedures in osteoporotic VCF is intense and sustained pain adjacent to the fracture with failure of conservative management for a minimum of 3 weeks. These procedures can also be used for pain relief in patients with osteolytic bony metastases.[35] Hirsch et al published a clinical care pathway using the RAND/UCLA Appropriateness Method. The multidisciplinary expert panel recommended that cement augmentation procedures be considered in patients with positive findings on advanced imaging (preferably MRI) and worsening symptoms, and in patients with 2–4 of the following unfavorable characteristics: progressive height loss, vertebral body height loss greater than 25%, kyphotic deformity, or severe impact on daily functioning.[36] Contraindications for these procedures include coagulation disorders, infection, allergy to bone cement, tumor involving the spinal canal, and unstable fractures.[20,35]

In a vertebroplasty, fluoroscopic guidance is used to inject cement into the fractured cancellous bone. This can alleviate pain and prevent further loss of height (**Figure 2**).[33,37] A kyphoplasty is similar to a vertebroplasty but involves an inflated balloon tamponade to restore vertebral height and create a cavity which can be subsequently filled with cement. This has theoretical advantages over vertebroplasty since it minimizes cement extravasation, restores vertebral height, and reduces kyphosis. In practice, clinical studies have found both procedures to be effective with no differences between patient-reported outcome measures.[37,38]

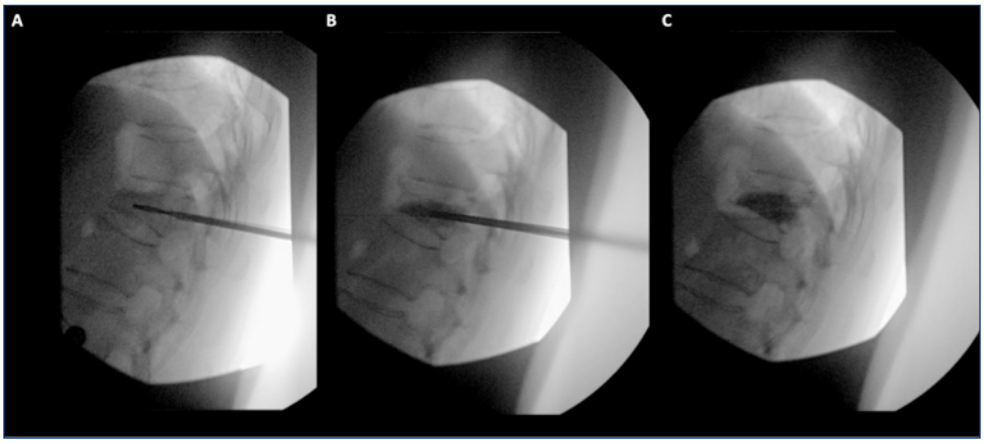
Complications of cement-augmentation procedures are low but include bleeding, infection, and neurological injury.[27] Cement extravasation is a rare yet catastrophic complication which can lead to arterial embolization or compression of neural elements. This complication is more common in vertebroplasty where cement is injected at higher pressure.[38] A systematic review and meta-analysis comparing cement-augmentation procedures to non-surgical management of osteoporotic VCF found superior pain outcomes in the surgically treated patients, demonstrating their efficacy.[39]

SECONDARY FRACTURES

One of the challenges following cement augmentation procedures is the risk of a subsequent VCF, which often happens at the adjacent vertebral levels to cement injection.[40] In a radiological study, new VCFs occurred in approximately one-third of patients, and in more than half of these the fracture occurred within 3 months of vertebroplasty at the adjacent vertebral level.[41] Several studies have considered if novel VCFs are the result of osteoporotic progression or the consequence of vertebral stiffness by cement augmentation. Several biomechanical studies have reported minimal changes in stresses and strains at adjacent levels to the kyphoplasty and conclude that adjacent segment fractures are more likely due to progression of osteoporosis rather than the intervention.[42,43]

Moon et al followed 111 female patients with osteoporotic VCFs who underwent kyphoplasty. The 1-year incidence rate of new compression fractures was 15.5% which is lower than the rate in natural osteoporotic progression.[40] The authors conclude that the lower incidence rate observed in their study sample could be related to a higher percentage of patients who were receiving medication for osteoporosis and recommend that spine surgeons should consider postoperative utilization of anti-osteoporotic medication to prevent novel fractures. This is supported by a meta-analysis which found that low BMD is a high-risk factor for refracture.[14] In fact, a 1% increase in BMD has been associated with a 3% reduction in risk of VCF.[13] This evidence emphasizes the role of metabolic treatment for primary VCFs to optimize treatment outcomes and reduce the risk of subsequent fractures. Furthermore, Moon et al found that one third of patients with subsequent VCFs were clinically asymptomatic, which emphasizes the importance of careful

Figure 2. A: Intraoperative image of a kyphoplasty demonstrating vertebral body access, **B**: balloon inflation, and **C**: cement injection.



follow-up. In patients with a primary VCF, follow-up imaging should be considered. This can include AP and lateral radiographs to detect progressive kyphosis or coronal plane deformity 2-4 weeks after diagnosis. A repeat MRI should be considered in the presence of new or progressing neurological symptoms, which may indicate an additional fracture, infection, or tumor.[44,41]

Due to the high likelihood of additional fragility fractures after a primary VCF is identified, such as distal radius fractures, geriatric hip fractures, or additional VCFs, many institutions have implemented the concept of a fracture liaison service to identify high-risk patients and pursue early diagnostics and potential intervention. These services have shown benefit in improving patient outcomes, and early referral should be considered if one of these services is available to patients with the new diagnosis of a VCF.[45]

CONCLUSIONS

VCFs are the most common fragility fracture affecting patients with osteoporosis. (1) Patients can present with acute pain although many are diagnosed asymptomatically after incidental imaging. (2) Plain radiographs are the modality of choice for diagnosis, while CT and MRI imaging may be required if a patient has neurological deficits, or a malignancy is a considered cause for the fracture. (3) Initial management is often non-surgical for at least 3 weeks before cement-augmentation procedures are considered. (4) A critical component in the management of VCF is the initiation of strategies for fracture prevention. If not performed recently, a DEXA scan should be ordered to monitor BMD. Patients should be educated on lifestyle changes such as smoking cessation and exercise, with referral to physiotherapy if assistance is needed to promote a regular program. (5) Pharmacologic treatment should be strongly considered for treatment of osteoporosis and fracture prevention. Medications to treat osteoporosis include bisphosphates (which are common first-line therapeutics), denosumab (a RANK ligand inhibitor), selective estrogen receptor modulators (raloxifene), and recombinant human parathyroid hormone (teriparatide). To prevent the progression of osteoporosis, it is also crucial to normalize calcium and vitamin D levels and provide dietary supplementation. (6) Early referral to a fracture liaison service or other provider who manages osteoporosis may improve outcomes in these patients and reduce risk of future fragility fractures. (7) An individual who experiences a VCF has a 5-fold increased risk of having a subsequent one, thereby justifying treatment regardless of bone mineral density.

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