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REVIEW ARTICLE

Evaluation and Management of Vertebral Compression Fractures

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Abstract

Compression fractures affect many individuals worldwide. An estimated 1.5 million vertebral compression fractures occur every year in the US. They are common in elderly populations, and 25% of postmenopausal women are affected by a compression fracture during their lifetime. Although these fractures rarely require hospital admission, they have the potential to cause significant disability and morbidity, often causing incapacitating back pain for many months. This review provides information on the pathogenesis and pathophysiology of compression fractures, as well as clinical manifestations and treatment options. Among the available treatment options, kyphoplasty and percutaneous vertebroplasty are two minimally invasive techniques to alleviate pain and correct the sagittal imbalance of the spine.

Introduction

Vertebral compression fractures (VCFs) of the thoracolumbar spine are common in the elderly, with approximately 1.5 million VCFs annually in the general US population.[1] Approximately 25% of all postmenopausal women in the US get a compression fracture during their lifetime.[2] The prevalence of this condition increases with age, reaching 40% by age 80.[3] Population studies have shown that the annual incidence of VCFs is 10.7 per 1000 women and 5.7 per 1000 men.[4] Men older than age 65 years are also at increased risk of compression fractures. However, their risk is markedly less than that of women of the same age.[4-6] Vertebral compression fractures are as common in Asian women as in Caucasian women, and less common in African-American women.

Although less severe than hip fractures, VCFs can cause severe physical limitations. Chronic back pain, which is associated with these kinds of fractures, leads to functional limitations and significant disability. Multiple adjacent VCFs can lead to progressive kyphosis of the thoracic spine, resulting in a number of comorbidities, such as decreased appetite resulting in poor nutrition and decreased pulmonary function.[5,7,8] The progressive decline in health status likely contributes to increased morbidity and mortality in patients with VCF compared to the general population.[8,9] VCFs also significantly increase medical costs: the estimated annual cost of VCFs in the US is $746 million.[10,11]

Etiology of Vertebral Compression Fractures of the Spine

The most common etiology of VCFs is osteoporosis, although trauma,[12] infection, and neoplasm can also lead to VCFs.[13,14] Postmenopausal women have the greatest risk because of hormonal changes that can lead to osteoporotic bone. Decreased bone mineral density because osteoporosis disrupts the bone microarchitecture and alters the contents of noncollagenous proteins in the bone matrix.[15,16] This structural deterioration of the tissue leads to fragile bones that are prone to fractures. It is estimated that approximately 44 million Americans have osteoporosis and that an additional 34 million Americans have low bone mass.[17]

Studies have suggested that having 1 VCF increases the risk of future VCFs. Lindsay et al reported that, irrespective of bone density, having 1 or more VCFs leads to a 5-fold increase in the patient's risk of developing another vertebral fracture.[18] Other studies have also found that having 1 compression fracture increases the risk of another compression fracture by 5 fold, and having 2 or more compression fractures increases the risk of having another fracture by 12 fold.[19-21] The relative risk for developing VCFs also increases with decreased bone mineral density: if bone mineral density is decreased by 2 standard deviations, the risk of developing a VCF increases by 4 to 6 times.[19]

Presentation and Complications From Vertebral Compression Fractures

Compression fractures of the thoracolumbar spine have a flexion compression mechanism of injury. This mechanism usually involves the first column (anterior longitudinal ligament and anterior half of the vertebral body). Pain is the main symptom (Table 1); neurologic deficits tend to be quite infrequent, because such a fracture does not involve retropulsion of bone fragments into the vertebral canal. Compression fractures of the vertebral bodies are particularly worrisome in patients with severe osteoporosis. Fractures occur in these patients during trivial events, such as lifting a light object, a vigorous cough or sneeze, or turning in bed. It has been hypothesized that fractures in vertebral bodies occur because of an increased load on the spine cause by contraction of paraspinal muscles.[16,22,23] It has been suggested that approximately 30% of compression fractures in patients with severe osteoporosis occur while the patient is in bed.[24,25] Patients with moderate osteoporosis can injure their spine by falling off a chair, tripping, or attempting to lift a heavy object. The most likely cause of a spinal compression fracture in those without osteoporosis is severe trauma, such as an automobile accident or a fall from a great height. When patients younger than age 55 years present with compression fractures, malignancy should be considered as a possible cause of the fracture.[26]

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Vertebral compression fractures have an insidious onset and may produce only low-grade back pain. Over time, multiple fractures may lead to progressive loss of stature and continuous contraction of the paraspinal musculature to maintain posture. This combination results in fatigued muscles and pain that may continue even after the original compression fractures have healed.[27]

Patients with multiple compression fractures and progressive loss of vertebral body height may develop excessive thoracic kyphosis and lumbar lordosis.[16,28] In severe cases of kyphosis, pressure exerted by the thoracic cavity on the pelvis can cause impaired pulmonary function, a protuberant abdomen, and early satiety and weight loss. Other complications of compression fractures include constipation, bowel obstruction, prolonged inactivity, deep vein thrombosis, increased osteoporosis, progressive muscle weakness, loss of independence, kyphosis and decreased height, crowding of internal organs, respiratory disturbances (eg, atelectasis, pneumonia, and prolonged pain), low self-esteem, and emotional and social problems; these patients are also more likely to be admitted to a nursing home.[29,30] Patients with compression fractures have a 15% greater risk of death compared to those who do not have a compression fracture.[21,29,31]

VCFs can lead to segmental instability when the vertebral body collapse is more than 50% of the initial height. With one segment collapsed to the point of instability, the adjacent levels have to support the additional load. This increased strain on the adjacent segments may result in degeneration of the spine and/or additional VCFs.[32]

A significant majority of fractures, 60% to 75%, occur around the thoracolumbar region. This segment is between T12 and L2 and is considered a transition zone from the more rigid thoracic vertebral column to the relatively mobile lumbar vertebral column. This anatomic relationship makes the thoracolumbar junction more prone to fractures than the rest of the spine.

Risk Factors for Vertebral Compression Fractures

The most important risk factor for VCF is osteoporosis, but there are a number of others, both modifiable and nonmodifiable[33] (Table 2). Modifiable risk factors include activities and behaviors that the patient can change, such as alcohol consumption, tobacco use, osteoporosis, estrogen deficiency, early menopause or bilateral salpingo-oophorectormy, premenopausal amenorrhea for more than one year, frailty, impaired eyesight, insufficient physical activity, low body weight, dietary calcium deficiency, and dietary vitamin D deficiency[33,34] (Table 2). Nonmodifiable risk factors include advanced age, female sex, Caucasian race, dementia, susceptibility to falling, history of fractures in adulthood, history of fractures in a first-degree relative, previous steroid treatment,[35] and previous treatment with anticonvulsants (Table 2). Managing modifiable risk factors, including treatment for osteoporosis, is the first step in preventing VCFs.[33]

Interestingly, obesity is protective against fractures, as it decreases the risk of bone loss: high stress on the bone induces a stronger bone remodeling response.[29] In addition, obesity leads to increased quantities of sex hormones, especially estrogen, which promotes osteoblast activity. The hyperinsulemia associated with obesity leads to decreased production of insulin-like growth factor binding protein-1 (IGFBG-1), thus increasing levels of IGF-1 protein, which stimulates the proliferation of osteoblasts.[29]

Detecting Osteoporosis

The most reliable method of detecting osteoporosis, and thereby identifying patients at risk for compression fractures, is to measure bone mineral density.[36] Currently, the standard method of measuring bone mineral density is dual-energy x-ray absorptiometry.[36] This test has become the gold standard because it can measure central bone mass and has excellent specificity. Bone mineral density T scores represent the standard deviation from the mean peak value in young adults. According to the World Health Organization, a T score less

Table 1. Symptoms and complications of vertebral compression fractures[11,21,30-31,42]

Symptoms	Complications
Sudden onset of back pain	Continuous low-grade back pain
Intensity of pain increases during standing or walking	Thoracic kyphosis and lumbar lordosis
Intensity of pain decreases when lying on the back	Impaired pulmonary function
Pain increases during palpation over the affected level	Protuberant abdomen, and early satiety and weight loss
Decreased spinal mobility because of pain	Increased osteoporosis because of inactivity
	Deep vein thrombosis because of inactivity
	Decreased respiratory capacity because of kyphosis, which in turn leads to atelectasis pneumonia
	Low self-esteem and emotional and social problems

Table 2. Risk factors for vertebral compression fractures[7,21,30]

Modifiable	Nonmodifiable
Alcohol consumption	Advanced age
Tobacco use	Female sex
Osteoporosis	Caucasian race
Estrogen deficiency	Dementia
Early menopause	Susceptibility to falling
Bilateral salpingo-oophorectormy	History of fractures in adulthood
Premenopausal amenorrhea for more than one year	History of fractures in a first-degree relative
Frailty	
Impaired eyesight	
Insufficient physical activity	
Low body weight	
Dietary calcium deficiency	
Vitamin D deficiency	

than -2.5 indicates osteoporosis, while T scores from -1 to -2.5 indicate osteopenia or decreased bone density,[36] and T scores greater than -1 are normal.

Classification of Vertebral Compression Fractures

VCFs can be classified in three categories: wedge, biconcave, and crush. Wedge fractures are the most common, accounting for more than 50% of all VCFs.[37] These fractures occur in the midthoracic region and are characterized by compression of the anterior segment of the vertebral body (Figure 1a and 1c). Biconcave compression fractures are the second-most common, accounting for approximately 17% of all VCFs[37] (Figure 1b and 1c). In these fractures, only the middle portion of the vertebral body is collapsed, whereas the anterior and posterior walls remain intact. The least common VCFs are crush compression fractures. They account for only 13% of VCFs.[37] In these fractures, the entire anterior column, including anterior and posterior margins, is collapsed. Complex fractures account for the remaining 20% of VCFs.

Imaging Modalities

Several imaging modalities are available for evaluation of patients with suspected compression fractures. Plain radiographs are the initial diagnostic modality (Figure injuries should have a complete spine series. This helps to avoid overlooking injuries, especially when patients present with other life-threatening injuries.[26] Multiple VCFs are found in 5% to 20% of patients presenting with compression fractures. Loss of vertebral height, disruption in alignment along anterior and posterior vertebral body lines, facet dislocation, and an increase in interpedicular and interspinous distance (>7 mm) are indicators of vertebral disruption.[2] The major disadvantage of radiographic films is their inability to detect ligamentous injuries.[38] Measurement of posttraumatic kyphotic angulation is useful for assessment of fracture progression, especially for fractures managed conservatively. Kyphotic angulation is measured as the angle between the superior end plate one level above and the inferior end plate one level below the injured segment. Typically, upright films are used to measure kyphotic angulation and to monitor changes in and progression of kyphosis in patients with VCFs.

Another imaging modality used to evaluate VCFs is computed tomography (CT) scan (Figure 1b). CT scans are primarily used for areas where plain films suggest there may be injury. They can help detect instability of an anterior wedge compression fracture, and occult bony injuries. CT is ideal for imaging complex fractures and determining the degree of vertebral

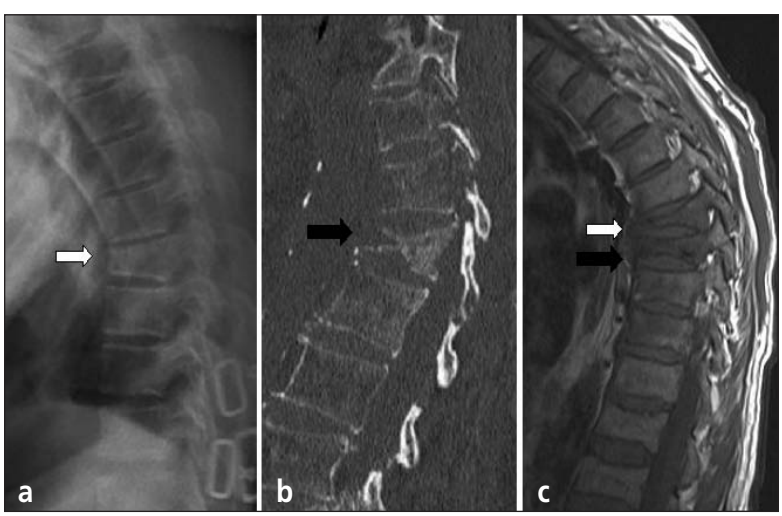

Figure 1. X-ray images of vertebral compression fracture: a) x-ray images of vertebral compression fracture with anterior wedging (white arrow) b) computed tomography scan of biconcave vertebral compression fracture (black arrow) c) T2 weighted magnetic resonance images of wedge vertebral compression fracture (white arrow), and biconcave vertebral compression fracture (black arrow).

More complex imaging modalities, such as CT myelography and magnetic resonance imaging (MRI) are not necessary unless the patient has a neurologic deficit. In special cases where the compression fracture is because of an infectious or malignant process, more advanced MRI techniques can be used. MRI is helpful for better visualization of cord compression and ligamentous disruption. High signal intensity indicates cord injury. MRI is also useful in evaluating the age of the VCF. New injuries can be identified by a T2 signal because of an increased signal intensity from water in the vertebral body. CT myelography for assessment of cord compression is indicated when MRI is contraindicated, such as in patients with a pacemaker. Imaging modalities other than plain films should always be used in patients with neurologic deficits, as multiple compression fractures can cause enough kyphotic angulation to lead to cord compression and progression to complete loss of neurologic function.

Treatment of Osteoporosis

Prevention and treatment of osteoporosis is one of the first steps in managing VCFs. Postmenopausal women with osteoporosis should be treated with 1500 mg calcium and 400 IU vitamin D daily.[16,21] Serum testosterone should be tested in men with compression fractures to rule out hypogonadism.[16,21] Osteomalacia should be suspected if alkaline phosphatase level is elevated. Cigarette smoking should be discouraged, and alcohol should only be consumed in moderation.[21] A daily weight-bearing exercise program should be recommended.[16] Newer treatment options like bisphonates have been shown to reduce the risk of fractures.[15,21] In randomized clinical trials, alendronate has been found to reduce the risk of vertebral fractures by 50% in postmenopausal women.[3] Other agents with clinical evidence of efficacy include raloxifene, parathormone, and calcitonin.[39]

Nonsurgical Treatment

Nonsurgical management is one of the preferred approaches for treatment of VCFs.[18,28] Conservative management includes a short period of bed rest followed by gradual mobilization with external orthoses.[39] Since VCFs are flexion-

compression injuries, a hyperextension brace is used. These braces are usually beneficial for the first few months, until the pain resolves. Although younger patients tolerate bracing well, elderly patients generally do not,[28] because of increased pain with bracing. Thus, elderly patients tend to require more bed rest. Immobility predisposes patients to venous thrombosis and life-threatening complications such as pulmonary embolism. It can also lead to pressure ulcers, pulmonary complications, urinary tract infections, and progressive deconditioning. In addition, it has been reported that bone mineral density decreases 0.25% to 1.00% per week in patients who are on bed rest.[23,40] To reduce pain and thus promote early mobilization with conservative management, appropriate analgesics should be prescribed. Narcotics should be reserved for patients who receive inadequate relief from regular analgesics. A major concern with narcotics is physical dependence and other adverse effects, like gastrointestinal dysmotility and cognitive deficits. Physical therapy and rehabilitation are also important factors that expedite healing.

For patients with pathologic compression fractures, a course of radiotherapy may be indicated if the tumor is radiosensitive. Radiotherapy provided pain relief in approximately 50% of patients with VCFs due to myeloma or prostate or breast cancer.[41,42]

Operative Management

Operative management of VCFs has gained popularity, as it produces rapid, significant, and sustained improvements in back pain, function, and quality of life.[43] Surgical intervention is indicated for those patients with intractable back pain failing conservative therapy or where there is evidence of impending or existing neurologic deficit, or where the spinal deformity is extremely severe.[25,28] However, operative management of elderly patients does carry increased risk because of comorbidities.[16,25]

There are several surgical options for the management of painful osteoporotic fractures. Vertebral augmentation through minimally invasive techniques such as kyphoplasty and percutaneous vertebroplasty are among the most popular.[25,31] Other methods include use of the OsseoFix Spinal Fracture Reduction System (AlphaTec Spine; Carlsbad, CA) and internal bracing. More invasive techniques, such as anterior and posterior decompression and stabilization with placement of screws, plates, cages, and rods are also available. These procedures, however, are challenging because it is difficult to achieve adequate fixation in osteoporotic bone.[23,25]

Percutaneous vertebroplasty is one of the favored methods of treating painful VCFs.[25] It encompasses augmentation of the vertebral body by injection of polymethylmethacrylate (PMMA).[25] This method has been successful in treating pain, even eliminating the need for pain medication in some cases. Short-term results indicated that 75% to 100% of patients can have good to moderate pain relief after vertebroplasty,[1,6] which also increases functional ability by stabilizing the fracture and preventing further vertebral collapse.[44,45] Vertebroplasty is most effective in compression fractures less than 6 months old. Its objective is not to restore the height of the vertebral body; in static fractures the average increase in anterior body height is only 2.5 mm. Contraindications of this procedure include infection of the vertebral body, coagulopathy, bone fragment retropulsion, and allergy to any of the substances used during the procedure, including PMMA cement and sometimes contrast agent. A number of potential serious complications of intraosseous injection of bone cement have been reported in the literature. One such complication is cement leakage, which ranged from 3% to 75%.[22] Leakage into the spinal canal may result in neurologic deficit, such as radiculopathy or spinal cord compression. In addition, there was an increased incidence of new VCFs in the adjacent segments after vertebral body augmentation procedures.[22] This is currently thought to be because of the increased stiffness of the treated vertebra compared to the adjacent vertebral bodies.

Despite the early encouraging results of vertebroplasty for VCFs, in 2009 Buchbinder et al found that vertebroplasty offered no benefit to patients with fresh and painful VCFs.[46] In this placebo-controlled study, researchers performed sham surgery, which included percutaneous insertion of the needle and opening the PMMA-monomer mixture to release the odor present during the real operation.[46] MRI in 78 patients confirmed that vertebral compression fractures had been treated, and no improvement in symptoms was observed in patients who received vertebroplasty. Patients in both groups had similar, significant reductions in overall pain and similar improvement in physical functioning, quality of life, and perceived recovery.[46] A similar study also showed that vertebroplasty and a sham procedure had equivalent results.[47]

Another option for vertebral body augmentation is kyphoplasty. This involves placement of an inflatable balloon tamp in the fractured vertebral body.[27] The balloon is inflated using a contrast agent so that position and inflation can be confirmed with image-intensified fluoroscopy. The inflation creates a cavity that can later be filled with PMMA or other types of bone cement. The risks associated with this procedure are similar to those of percutaneous vertebroplasty, however lower rates of cement leakage into the spinal canal have been reported.[43] Kyphoplasty offers the potential for reversing spinal deformities: height restoration can be improved postoperatively by 50% to 70%, with a segmental kyphosis improvement of 6° to 10°.[26,48] Thus, kyphoplasty has the potential to prevent the pulmonary and gastrointestinal complications associated with severe kyphosis.[48] Kyphoplasty is most successful at restoring the height of the fractured vertebral body if it is performed within 3 months of the occurence of fracture or onset of pain.[22,23,43,49,50] Short-term results show that 85% to 100% of patients have good to moderate pain relief.[26,48] Wardlaw et al found that kyphoplasty had improved functional recovery compared with nonsurgical treatment.[51] Contraindications of kyphoplasty are similar to those of percutaneous vertebroplasty and include infection of the vertebral body, coagulopathy, bone fragment retropulsion, and allergy to any of the substances used during the procedure, including cement and contrast agent.[24,33,52] Garfin et al found that short-term complications from this procedure were related to cement extravasation

… because of increased pain with bracing. … elderly patients tend to require more bed rest. Immobility predisposes patients to venous thrombosis and life-threatening complications …

and damage from heat and pressure on the spinal cord and nerve roots.[43]

New techniques have been developed to minimize the risks of complications from kyphoplasty. Vesselplasty was developed in 2009 to decrease the rate of cement leakage: the inflatable balloon is left in the patient and filled with cement, thus reducing the risk of cement leakage.[53] Alternatives to PMMA were also explored. An expandable polymer bone tamp, Sky Bone Expander (Disc-O-Tech Medical Technologies, Ltd; Herzliya, Israel), appeared to have good initial results.[54] Cortoss (Orthovita; Malvern, PA), a bioactive, injectable, nonresorbable composite consisting of highly cross-linked resins and reinforcing bioactive glass fibers, was also found to have a more physiologic load transfer, and patients treated with Cortoss were less likely to be hospitalized for new vertebral compression fractures.[55]

Conclusion

Compression fractures affect many patients worldwide and are most common in elderly populations, especially postmenopausal women. These fractures often cause incapacitating back pain and morbidity. The most important step in treating compression fractures is prevention and treatment of osteoporosis. When vertebral compression fractures become symptomatic and cause disability, several treatment options are available, including kyphoplasty to alleviate pain and correct the sagittal imbalance of the spine. ❖

Disclosure Statement

The author(s) have no conflicts of interest to disclose.

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Osteoporosis

If the compact osseous tissue becomes porous from the widening of the Haversian canals, the condition is termed osteoporosis … In the vertebrae and in the bones of the extremities, both concentric and eccentric atrophy take place, the bony trabeculae being thereby in places thinner or even entirely absorbed.

— *A Text-Book of Special Pathological Anatomy,* Ernst Ziegler, 1849-1905, German pathologist