Management of Osteoporotic Vertebral Compression Fractures: A Review

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Abstract

Osteoporotic vertebral compression fractures are increasing in prevalence as the general population ages. The consequences of these fragility fractures are significant and include pain, impaired normal physical functioning, decreased lung capacity, kyphosis of the spine, loss of appetite, depression, and increased mortality. Early aggressive treatment of these fractures can prevent the ripple effect of morbidity associated with these fractures. Prevention and treatment of the underlying osteoporosis forms the foundation of the treatment paradigm for these fractures. A review of epidemiology, consequences, diagnosis, and treatment options for osteoporotic vertebral compression fractures is presented.

Introduction

Osteoporosis is a systemic skeletal disease characterized by compromised bone strength, which predisposes the affected bone to fracture.[1] The osteoporotic bone has a reduced number, thickness, and connectivity of trabecular rods. This results in increased fragility of the bone and thereby predisposes the patients to have a fracture with relatively little trauma. The lack of a universally accepted definition of vertebral fracture, the continually emerging data on these fractures, and the large proportion of undiagnosed fractures contribute to an evolving understanding of the epidemiology of these fractures. As the geriatric population increases, the incidence of osteoporotic vertebral fractures has been increasing. An estimated 1.4 million osteoporotic vertebral fractures come to receive clinical attention worldwide.[2]

Epidemiology

Osteoporosis is a public health problem worldwide. The occurrence of an atraumatic vertebral compression fracture (VCF) is generally sufficient to establish a diagnosis of osteoporosis. Vertebral compression fractures occur spontaneously or more commonly occur as a result of minimal trauma from day-to-day activities, such as bending forward, twisting, lifting objects, and even sitting from a standing position onto a low chair.[3] It is known that the risk of vertebral fractures rises rapidly with age for both men and women. In the United States and Europe, women are two to three times more likely than men to experience a vertebral fracture.[3] In a population-based study, the age adjusted incidence of clinically diagnosed vertebral fracture was 145 per 100,000 person years in women compared to 73 per 100,000 person years in men.[3]

Many of these fractures go undiagnosed. It has been shown that only one-third of all vertebral compression fractures in postmenopausal women in the United States are brought to clinical attention.[4] A prevalent vertebral compression fracture is defined as a fracture that exists at a discrete point in time. An incident vertebral compression fracture is a fracture that has occurred between two points in time. The lifetime prevalence of clinical vertebral compression fracture among Caucasians is approximately 15% for women and 5% to 9% for men, based on epidemiological data from the USA and Sweden.[5, 6] Prevalence of vertebral compression fractures increases with age.[7]

More recently, trials, such as the Vertebral Efficacy with Risedronate Therapy (VERT) trial, showed that there is a 1 in 5 risk of subsequent vertebral compression fracture within 12 months following an incident fracture among postmenopausal women.[8] It confirmed that postmenopausal osteoporotic women with prevalent vertebral compression fractures are more likely to experience incident vertebral compression fractures than do postmenopausal osteoporotic women without vertebral compression fractures.[8] These data clearly point to the urgency of intervention for anyone who sustains a vertebral compression fracture.

Consequences

Although acute vertebral compression fracture pain can be severe, it is self-limiting and responds to simple measures such as analgesic medications, activity modification or temporary limitation of physical activities, and braces. However, there can be permanent long-term side effects of these fractures. Due to the anterior compression of the vertebral body, the center of gravity moves forward, thereby creating a large bending moment. This increased bending moment must be counterbalanced by the posterior muscles and ligaments, which results in muscle fatigue and pain. Also, because of the anterior translation of the bending moment, there are larger compressive forces on the osteoporotic anterior part of the vertebral body. Degenerative changes in the disk result in uneven transmission of load to the vertebral end plates, which along with the loss of disk height and the kyphotic posture result in further increase in the compressive loads on the vertebral body. The knees bend and the pelvis tilts forward to counteract the forward bending of the kyphotic spine. This results in a muscle fatigue, gait abnormalities, decrease in gait velocity, and consequently an increased risk of falls and additional fractures.[9] Additionally, the decreased activity leads to worsening of the osteoporosis. The posture results in protrusion of the abdomen. It is evident that the kyphotic deformity, and not the acute pain, accounts for the long-term consequences of vertebral compression fractures.

Normal curvature of the thoracic spine in adults is 20 to 40 degrees. Multiple vertebral compression fractures can result in kyphosis of the thoracic spine with measurements exceeding 50 degrees. This can result in a loss of overall height of the individual. As a result of this kyphotic angulation of the spine, the twelfth rib may rest on the iliac crest and the gap between the lower ribs and ilium may narrow. As a consequence, the abdomen is compressed, resulting in a loss of appetite, distension, eructation, and constipation. Studies have shown that the extent of spinal deformity significantly correlated with intensity of pain, physical limitations, and changes in mood.[10]

Chronic pain and discomfort are often present in persons with multiple vertebral compression fractures.[11] The chronic pain is often the cause of significant psychological and social consequences including social isolation, lower self-esteem, increased anxiety, problems in relationships, increased dependence on others, insomnia, and depression.[9, 12]

Vertebral compression fractures in the thoracic spine reduce the pulmonary function. One thoracic vertebral compression fracture causes an approximately 10% loss of forced vital capacity.[13] In patients with co-existing lung disease, the loss of lung capacity can be particularly significant.[13] Women with one or more VCF have an age-adjusted relative risk of mortality related to pulmonary causes of 2 times to 2.7 times higher compared to women without VCF.[14]

The economic impact of osteoporosis and the VCF is significant. Each VCF is estimated at $8,000 to $16,600 in hospitalization costs.[15] The indirect costs of vertebral compression fractures, including lost productivity, lost activities of daily living, and lost productivity of caregivers, have not been adequately studied for either hospitalized or non-hospitalized patients with VCF. The indirect costs of osteoporotic fractures have been estimated at between $4.5 billion and $6.4 billion in the United States.[16] Of all the types of osteoporotic fractures, clinical lumbar fractures are associated with the lengthiest need for help from caregivers.[17]

Diagnosis

Osteoporosis can be primary or secondary. Secondary osteoporosis can be due to several underlying medical conditions or medications. Secondary causes are usually identified through patient history and laboratory testing. A detailed patient history can often identify the risk factors for osteoporosis. The most common risk factors identified by history alone for secondary osteoporosis include use of oral glucocorticoids (>7.5 mg/day), early menopause, and malnutrition or unintentional weight loss.[18] Underlying medical conditions include diabetes mellitus, hyperparathyroidism, renal failure, chronic obstructive pulmonary disease, rheumatoid arthritis, alcoholism, hepatic disease and disorders, multiple myeloma, and metabolic disorders. The use of tobacco products, barbiturates, and heparin are also potential causes of secondary osteoporosis. Other possible causes of VCF include multiple myeloma, metastatic bone disease, and Paget's disease. Because of the complex health issues often seen in this patient population, a comprehensive medical history and general physical examination should be complemented with a complete blood count, comprehensive metabolic panel, erythrocyte sedimentation rate, and serum and urine protein electrophoresis as needed, to assist in the initial detection of underlying infectious, metabolic, or malignant processes.

Vertebral compression fractures may be asymptomatic and hence can be missed clinically. However, the patient may present with severe and chronic back pain. Often, it may be necessary to differentiate chronic back pain from unrelated causes, such as lumbar spinal canal stenosis and degenerative lumbar spondylosis, from pain caused by a VCF. Because of the complex etiology of back pain, osteoporotic VCF may not be suspected or even considered by the clinician, even in the presence of severe back pain not attributable to any other cause. It must be noted that a fairly significant proportion of patients will present with pain radiating along the ribs or, in many cases, sternal or chest pain or abdominal pain.

Typically, the acute back pain associated with osteoporotic VCF subsides as the fracture heals over a period of approximately three months.[22] A closer analysis reveals that not all fractures heal. Even if the fracture has healed, back pain can persist, and this has been attributed mostly to the resulting kyphotic deformity of the spine.

In patients who present with a sudden onset of severe back pain with little or no trauma, a lateral spine radiograph may be a good screening tool to diagnose VCF. However, while back pain is the most common symptom from VCF, an analysis of a cohort of women (n=2,992) aged 65-70 years, indicated that 38% of

women with moderate or severe back pain showed no signs of any vertebral deformity on initial radiographs.[19] The height loss associated with vertebral fracture may be gradual rather than immediate. The most common location for osteoporotic VCF is the midthoracic region (T7-T8) and the thoracolumbar junction (T12-L1). These locations correspond to the most mechanically compromised regions of the spine. In the thoracic region, where the thoracic kyphosis is most pronounced and loading during flexion is heightened and the thoracolumbar junction where the relatively rigid thoracic spine connects to the more freely mobile lumbar segments.[3] Morphologically, VCF types include wedge, crush, or biconcave fractures. Wedge-shaped fractures are the most common. In general, radiographic screening for osteoporotic VCF is warranted with a new onset or worsening of back pain in men or women with osteoporosis, those receiving oral glucocorticoid therapy, or postmenopausal women aged 55 years or older, loss of two or more inches in height,[20] prominent kyphosis in postmenopausal women aged 55 years or older, and decreased bone mineral density (BMD).[21] Radiographs of the spine cannot, however, differentiate between new VCF and old or healed VCF.

The presence of a malignant neoplasm is always of concern in elderly patients with non-traumatic vertebral compression fractures, especially when the fracture occurs cephalad to the T5 level or if there are significant constitutional symptoms or failing health. An MRI scan is the single most useful imaging modality in the evaluation of osteoporotic VCF. Acute intravertebral edema, indicating a fracture, is easily identified on the MRI as a high-intensity signal on T2 weighted and fat suppression or short tau inversion recovery (STIR) sequences. MRI is also useful in differentiating osteoporotic VCF from other causes of VCF, including malignant neoplasms. Involvement of the pedicle or posterior elements should alert the clinician to the presence of an underlying malignant neoplasm or infection. In patients who cannot have an MRI scan due to contraindications such as the presence of a cardiac pacemaker, they may be evaluated with a nuclear scintigraphic bone scanning and a CT scan of the spine at the site of the fracture. Scintigraphic uptake, however, may be nonspecific and may persist for as long as two years after fracture, thereby reducing diagnostic specificity. Positron emission tomography can assist in discriminating malignant from non-malignant causes of vertebral fractures when standard uptake values are greater than 2.5.

Management

The ideal goal of treatment of a patient who has been diagnosed with osteoporotic VCF is to alleviate pain and reduce and stabilize the fracture. However, based on overall health of a patient and patient choice, various treatment modalities may have to be considered. Some patients may have minimal symptoms of pain and may not want any interventional treatment. Treatment modalities include general medical management, open surgical treatment, and percutaneous vertebral body augmentation. General medical management includes analgesic medication, activity modification, spinal brace, and medical treatment of the osteoporosis. Pharmacotherapy for established osteoporosis reduces the incidence of future fractures and must be initiated in all patients. Current medications include anticatabolic therapies (hormone replacement therapy, calcitonin, raloxifene, and aminobisphosphonates such as alendronate, ibandronate, and risedronate) or anabolic therapy (teriparatide). The shortcomings of medical management include failure to adequately control pain, failure to provide long-term functional improvement, and deformity causing persistent pain due to failure in restoring the anatomy. Open surgical treatment is indicated in those rare instances when the patient has a neurologic deficit as a result of the VCF. Open surgery involves large dissections, prolonged anesthetic times, and a high incidence of complications.

Percutaneous vertebral body augmentation procedures include vertebroplasty and kyphoplasty. In recent years there has been a dramatic growth in the adoption of these procedures for the treatment of osteoporotic VCF. Both procedures consist of percutaneous cannulation of the fractured vertebral body followed by intravertebral installation of bone cement, such as polymethylmethacrylate (PMMA). More recently, it has been possible to install allograft bone graft in a mesh as a method of vertebral body augmentation. In vertebroplasty, a low pressure injection of cement into the vertebral body is carried out without any attempt to reduce the fracture. In kyphoplasty, a balloon is inserted into the vertebral body with the dual purpose of fracture reduction and void creation. Pain relief is comparable in both vertebroplasty and kyphoplasty. The exact mechanism of pain relief is unproven but is believed to result from the stabilization of the fractured vertebra and elimination of macroscopic and microscopic motion at the fracture site. Other postulated mechanisms include chemical and thermal neurolytic effect of PMMA. Absolute contraindications to kyphoplasty include coexisting infection, pregnancy, non-painful VCF, high-velocity fractures, fractures with retropulsed bone fragments into the spinal canal, uncontrolled coagulopathy, extensive vertebral body disruption, technical factors, and medical conditions precluding anesthesia or operative intervention.[23, 24]

It is well known that the degree of kyphosis correlates with poor levels of physical functioning.[14] The advantages of kyphoplasty over vertebroplasty include better correction of the kyphotic deformity by restoration of height of the vertebral body. While the magnitude of benefit obtained with kyphoplasty over simple stabilization with vertebroplasty continues to be debated, a recent study by Grohs et al. showed that kyphoplasty provided a mean correction of the kyphotic angle of 6°, while no significant reduction was achieved with vertebroplasty.[25] Optimal time for intervention with kyphoplasty again remains a subject of debate. Recent studies have supported the early intervention approach, arguing that early treatment yields more complete and reliable fracture reduction. Ortiz et al. reported that there was a 4.4° of correction of kyphosis when VCF was treated within six months of the event versus 2.7° of correction when treated after six months after the event.[26] Also, height increases of 44.7% were seen when fractures were treated with kyphoplasty within six months versus a height increase of 34.6% when the fractures were treated after six months.[26]

Pain relief associated with percutaneous vertebral body augmentation treatment is as high as 96% at a mean of 48 month follow-up.[27] Prospective studies comparing vertebroplasty with kyphoplasty did not show any significant difference in the rates of success, with 92% success for vertebroplasty versus 93% for kyphoplasty.[28] A more recent randomized controlled trial showed that in patients with acute, painful, vertebral fractures, kyphoplasty improved quality of life, function, mobility, and pain more rapidly than did non-surgical management, with significant differences in improvement between the groups at one month.[29]

Like any other interventional procedure, percutaneous vertebroplasty and kyphoplasty have complications and morbidity associated with them. Complications can be divided into medical/anesthesia problems, surgical problems, and long-term effects. Postoperative morbidity including adverse events such as myocardial infarction and pulmonary embolism have been reported to occur, although none of these adverse events were procedure or device related.[29] It must be noted that a large proportion of patients with osteoporotic VCF are elderly and have multiple medical co-morbidities. Surgical and technical complications are known to occur and include fractures of the sternum and ribs during positioning of these frail patients, improper instrument placement due to poor visualization of the pedicles on fluoroscopy, leakage of cement into the spinal canal or neuroforamen causing neurologic injury and fracture of the pedicle and transverse process. More recent studies on kyphoplasty have not reported any major post-operative surgical complications in their study subjects.[24, 30] The most important complication of both vertebroplasty and kyphoplasty procedures remains leakage of cement. Cement leakage can occur into the spinal canal or the neuroforamen. Most often the leakage occurs into the disc space or the lateral aspect of the vertebral body. Cement leakage is clinically silent in the vast majority of cases, with symptomatic leaks representing only a small portion of the total. The latter represent the main source of pulmonary and neurologic complications.[31]

There is a concern that kyphoplasty may result in an increased rate of additional fractures of the spine.[32] However, it must be noted that all osteoporotic patients with VCFs are at relatively high risk of additional fractures. Wardlaw et al. reported no significant increase in the rate of new radiographic vertebral fractures in patients who underwent kyphoplasty for the index fracture when compared to controls.[29] The low rate of significant complications and the high success rate of these procedures in achieving pain relief and deformity correction make them a suitable choice of treatment for osteoporotic VCF.

Conclusion

With an increase in the aging population, osteoporosis-related vertebral compression fractures have become increasingly prevalent. Our ability to diagnose these fractures is improving with better awareness of this condition. Referral pathways are opening up as a result of increasing awareness of these fractures among the general population as well as among primary care and emergency room physicians. Percutaneous vertebral augmentation procedures including vertebroplasty and kyphoplasty provide excellent pain relief, prevention of further collapse of these fractures, improved quality of life, function, and mobility for these patients when compared to non-surgical treatment options. Irrespective of treatment modality adopted for the treatment of osteoporotic vertebral compression fractue, it is imperative that medical treatment of the unerlying osteoporosis should be aggressively pursued.

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