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# Diagnosis and Management of Vertebral Compression Fracture

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#### ABSTRACT

Vertebral compression fractures are the most common complication of osteoporosis, with 700,000 cases reported every year in the United States. Vertebral compression fractures typically present with abruptonset low back pain with or without a history of trauma, although more than two-thirds are detected incidentally. Diagnosis is confirmed using plain radiographs, while computed tomography and magnetic resonance imaging may be required to evaluate for a malignant cause or if there are neurological deficits on examination. Magnetic resonance imaging is also the modality of choice to determine if the fracture is acute vs chronic in nature. Patients can be managed with a combination of nonsurgical modalities including medications, bracing, and physical therapy, although when indicated, kyphoplasty or vertebroplasty may be considered to provide symptom relief.

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## INTRODUCTION

Osteoporosis is the most frequently encountered bone disease.<sup>1</sup> It is characterized by a progressive loss of bone mineral density, defined as a score more than 2.5 standard deviations below the population average (T-score), as measured by dual-energy x-ray absorptiometry (DEXA).<sup>1</sup> The global prevalence of osteoporosis is estimated at 18.3%,

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and it more commonly affects women; with a prevalence of 19.6% in US women over 50 years of age, compared with 4.4% in men.<sup>1,2</sup> Postmenopausal women have the greatest risk; the decrease in estrogen after menopause results in increased bone turnover and an imbalance between bone resorption and formation.<sup>3-5</sup> Other factors that influence the development of osteoporosis are systemic and endocrine diseases, malignancy, use of glucocorticoids, low weight, use of alcohol or tobacco, physical inactivity, and calcium deficiency.<sup>1,6-8</sup>

Vertebral compression fractures are the most common complication of osteoporosis, with 700,000 reported cases every year in the United States.<sup>9-11</sup> The annual incidence of vertebral compression fractures has been shown to be 10.7 per 1000 women and 5.7 per 1000 men.<sup>10</sup> The prevalence of vertebral compression fracture increases with age; therefore, with an aging population demographic, clinicians must be mindful of the presentation and management of vertebral compression fractures. Vertebral compression fractures are estimated to occur in 25% of postmenopausal women at some point in their lifetime, and in 40% of women over 80 years old. Men over 65 years old are also at an increased risk for vertebral compression fracture, but this risk is lower than for women of similar age.<sup>12</sup> Vertebral

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compression fractures may present with ambiguous symptoms such as nonspecific atraumatic back pain, but may lead to severe physical impairment and morbidity.<sup>10</sup>

The aim of this review is to describe the presentation, diagnosis, and management of vertebral compression fractures, including nonsurgical and surgical care. Physicians and medical professionals across all specialties should be

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every year in the United States.

aware of this condition, as the burden of osteoporosis continues to grow.

## **CLINICAL FEATURES**

Vertebral compression fractures are most commonly due to osteoporosis, but they can also occur due to trauma, infection, or neoplasms.<sup>13</sup> In patients under 50 years old without a history of trauma, malignancy should be high on the differential diagnosis.<sup>14</sup> In osteoporotic vertebral compression fractures, fractures often occur during trivial events such as lifting light objects, coughing, sneezing, or turning in bed, although falls are also a common cause. It has been suggested that approximately 30% of compression fractures in patients with

severe osteoporosis occur while the patient is in bed.<sup>10,13</sup>

Previous vertebral compression fractures are associated with an increased risk of future vertebral compression fractures. Having one previous vertebral compression fracture increases the risk of a future one by fivefold, and having 2 previous vertebral compression fractures increases the risk of future such fractures by 12-fold.<sup>10</sup> Furthermore, with a decrease in bone mineral density of 2 standard deviations, the risk of a vertebral compression fracture increases by four- to sixfold.<sup>10</sup> Modifiable risk factors for vertebral compression fractures include factors that are also associated with an increased risk of osteoporosis, such as early menopause, bilateral salpingo-oophorectomy, alcohol use, tobacco use, calcium or vitamin D deficiency, and physical inactivity.<sup>12,13,15</sup> Nonmodifiable risk factors include advanced age >70 years, white or Asian race, female sex, history of steroid use, and history of treatment with anticonvulsants.<sup>1,13,15</sup> Obesity is protective against vertebral compression fractures due to the increased bone remodeling and formation induced by physical stress on bones, increased estrogen production by adipose tissue, and hyperinsulinemia leading to increased insulin-like growth factor 1, which stimulates bone formation.<sup>10</sup>

A vertebral compression fracture typically presents with abrupt-onset low back pain with position changes, weight bearing, or lying supine. However, in some cases this pain is only mild and attributed to another issue like a muscle spasm. In fact, more than two-thirds of vertebral compression fractures may be detected incidentally when patients undergo imaging for other reasons.<sup>9</sup> Patients without significant trauma with vertebral compression fractures rarely have neurological deficits, but they may have substantial functional disability.<sup>1,10</sup> Any patient at risk for osteoporosis with back pain or kyphosis should be assessed for vertebral compression fracture. Important red flags to con-

sider for a pathological fracture include history of malignancy, older age, weight loss, and persistent nonresolving back pain.<sup>16</sup> Physical examination findings are often normal, but may demonstrate kyphosis and midline spine tenderness upon percussion of the spine.<sup>17</sup> Patients also may complain upon rotation and positional changes.<sup>9,15</sup>

Although vertebral compression fractures rarely require hospital admission, they can lead to severe pain and functional impairment. Vertebral compression fractures affecting the thoracic spine can lead to progressive kyphosis of the thoracic spine with compensatory lumbar lordosis. These derangements can result in numerous problems such as a loss of height and mobil-

ity, decreased appetite resulting in poor nutrition, and decreased pulmonary function.<sup>9,12</sup> Vertebral compression fractures can also lead to chronic pain, increased risk of pressure sores, constipation and bowel issues, prolonged immobility leading to increased risk of deep vein thrombosis, pneumonia, and psychological distress.<sup>9,12,13</sup> All these issues may cause decreased independence and increased morbidity (Table). Patients with vertebral compression fractures are more likely to be admitted to nursing homes, and have a 15% greater risk of 1-year mortality compared with similar patients without vertebral compression fractures.<sup>10</sup> Patient population studies report that patients with osteoporotic vertebral compression fractures have an

Table	Symptoms	and	Complications	of	Vertebral	Compression
Fracture	es					

Symptoms Sudden onset low back pain Increased pain while walking or standing Limited spinal mobility
Complications
Kyphosis
Height loss
Loss of mobility leading to pressure sores, risk of deep venous thrombosis, pneumonia and psychological distress
Stomach problems, decreased appetite and poor nutrition
Breathing problems
Chronic pain
Constipation and bowel issues

increased mortality rate, which correlates with the number of involved vertebrae. These outcomes also have a significant impact on the US health care system—the annual medical cost associated with vertebral compression fractures in the United States is \$746 million per year.<sup>2,10</sup>

#### IMAGING

Over two-thirds of patients are diagnosed with vertebral compression fractures incidentally on plain radiography, and therefore it is important to consider imaging after a thorough history and physical examination.<sup>9</sup> Back pain and radiographic confirmation of a compression fracture indicates a symptomatic acute compression fracture, unless proven otherwise. Thus, it is necessary to confirm the diagnosis of vertebral compression fracture and exclude other causes of back pain. Sixty percent to seventy-five percent of vertebral compression fractures occur between T12 and L2 due to the change in rigidity between the rigid thoracic spine and the more mobile lumbar spine.<sup>10</sup> Clinicians should have a low threshold for radiological imaging, because many fractures evade diagnosis due to being dismissed as muscle strains, arthritis, or as a normal part of aging, and sometimes compression fractures are associated with metastatic disease, which can help diagnose malignancy. Furthermore, some clinicians may be reluctant to order radiological assessment, as the inciting event for fracture is often unclear or low energy.<sup>18</sup>

Plain film radiographs are typically the initial imaging study used for investigating a possible vertebral compression fracture.<sup>19</sup> Anteroposterior and lateral views of the

thoracolumbar spine are particularly important (Figure 1). If not previously recorded, DEXA scans should be acquired soon after the diagnosis of vertebral compression fracture to evaluate for underlying osteoporosis and determine disease severity.<sup>9</sup>

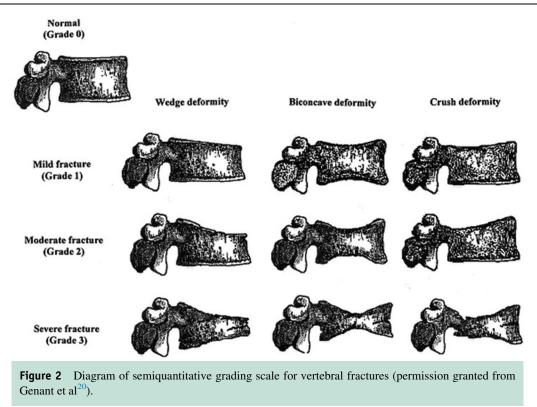
In a normal radiograph, the endplates are horizontal, and the vertebral shape and size is similar across adjoining levels. The common radiographic findings associated with vertebral compression fractures include a wedge deformity, linear zone of condensation, a step defect (which represents failure of the anterior/superior vertebral cortex), and disrupted vertebral endplate. Due to pain and concurrent narcotic medication, abdominal ileus may also be visible.<sup>18</sup> Any loss of vertebral height more than 20% with altered appearance and presence of end-plate deformities should be considered a fracture.<sup>19</sup>

There are several classification schemes for vertebral compression fractures. The Genant classification is commonly reported in literature, and has good diagnostic utility.<sup>20</sup> Fractures are classified according to their morphology and height loss. The vertebral shape is reported as either wedge, biconcave, or crush, with more than half of vertebral compression fractures described as wedge.<sup>10</sup> Wedge vertebral compression fractures classically occur in the midthoracic region with compression of the anterior segment of the vertebral body. Loss of height is graded from 0 to 3 (Figure 2<sup>20</sup>).

More detailed imaging techniques may be indicated when assessing if the vertebral compression fracture is of a benign osteoporotic cause, or if it represents malignancy.<sup>14</sup> Magnetic resonance imaging (MRI) is classically the imaging technique of choice because the characteristic



**Figure 1** Anteroposterior and sagittal radiographs showing an osteoporotic compression fracture at T8, showing focal kyphosis and mild coronal deformity through the fracture.



morphologic features, enhancement patterns, and signal intensities for cancerous lesions are well described. Interpretation may require radiological guidance due to diagnostic challenges—for instance, acute and subacute osteoporotic fractures may closely mimic malignant fractures in terms of MRI signal alteration due to resulting intertrabecular hemorrhage and edema. Computed tomography and MRI imaging may also be indicated if a patient presents with neurological deficit, including symptoms of spinal cord compression or progressive neurological decline.<sup>18</sup>

Follow-up imaging should be considered when treating patients with vertebral compression fractures. Protocol at follow-up can include anteroposterior and lateral radiographs to assess for any progressive kyphosis or coronal plane deformity 2-4 weeks following diagnosis. If neuro-logic symptoms develop, repeat MRI should be considered.<sup>15,21</sup> These symptoms may indicate an infection, tumor, or additional fracture. One prior study evaluated radiological follow-up for patients treated for vertebral compression fractures and found that new compression fractures were found in one-third of cases—half of these occurring with 3 months of treatment at the adjacent vertebral body.<sup>21</sup>

#### MANAGEMENT

Treatment aims for vertebral compression fractures include restoration of functional mobility, pain relief, and prevention of future fractures through addressing the underlying cause. Treatment strategies can be nonsurgical or surgical.

#### Nonsurgical

Patients can suffer from intense pain following a vertebral compression fracture, therefore, adequate pain management is essential.<sup>22</sup> Many patients achieve pain relief over 6-8 weeks as the fracture heals, but some experience persistent pain and disability.<sup>23</sup> Opioids, nonsteroidal anti-inflammatory drugs, and calcitonin have all been previously utilized for pain relief following these fractures. Nonsteroidal antiinflammatory drugs are frequently first-line medications for pain relief due to their perceived safety profile, low cost, and accessibility.<sup>24</sup> However, for elderly patients, there are notable side effects, such as gastrointestinal bleeding and renal impairment. Similarly, opioids can have sedating effects-which may be dangerous for the elderly population-as well as uncomfortable side effects including constipation and nausea. Opioid pain medication may be beneficial in acute pain management, although long-term use can lead to dependence and tolerance, and should not be routinely utilized for vertebral compression fracture.<sup>25</sup>

Calcitonin is a 32-amino acid polypeptide available as a nasal spray, subcutaneous or intramuscular injection, or rectal suppository. It may act as an analgesic by directly stimulating calcitonin binding receptors in the central nervous system and increasing beta-endorphin plasma levels.<sup>26</sup> Side effects are commonly mild, including flushing and diarrhea. A systematic review and meta-analysis found that calcitonin had strong efficacy in the management of acute back pain associated with vertebral compression fracture, but insufficient evidence for older fractures causing chronic pain.<sup>22</sup> One potential limitation of its use is the relatively high cost of calcitonin.<sup>27</sup>

Despite these findings, a systematic review of national guidelines across the United States, United Kingdom, and Canada found inconsistent guidance and weak evidence for the use of these medications.<sup>12</sup> In the face of mixed data, patients and clinicians may initiate nonsurgical management, but with substantial follow-up and attentiveness to when surgical intervention is necessary. Further studies are required to assess functional outcomes with calcitonin and other medication use, such as length of hospital stay and quality of life.<sup>28</sup>

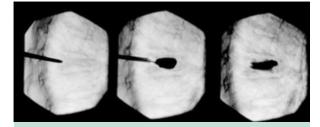
Nonsurgical prevention strategies to avoid future compression fractures includes adequate management of the underlying cause. In osteoporosis, this involves obtaining a DEXA scan to monitor bone mineral density; physical therapy/exercise to strengthen antigravity muscles; and prescription of appropriate medications.<sup>29</sup>

Normalizing calcium and vitamin D levels can help prevent progression of osteoporosis. The Institute of Medicine recommends that post-menopausal women with osteoporosis take 1000 mg of calcium daily through their diet and through supplements and 600 international units of vitamin D daily.<sup>30</sup> Bisphosphonates, such as alendronate, are taken orally and are a first-line treatment for osteoporosis.<sup>31-33</sup> They bind to the bone mineral matrix and inhibit osteoclast function, decreasing bone turnover, which results in an increase in bone mass. Side effects include upper gastrointestinal tract discomfort and, in rare cases, osteonecrosis of the jaw.<sup>34</sup> Due to accumulation in the bone, a drug holiday is required after they have been taken for 3-5 years.<sup>34</sup> Other medications that may be considered include denosumab (a receptor activator of nuclear factor- $\kappa$ B ligand inhibitor), raloxifene (a selective estrogen receptor modulator), and teriparatide (recombinant human parathyroid hormone).<sup>33</sup> Pharmacologic treatment of osteoporosis should be strongly considered in all patients with a vertebral compression fracture.

## Surgical

Determining when to refer for surgical management of a vertebral compression fracture is a challenge for physicians. One consensus panel of primary care physicians noted that leg pain or weakness in context of a vertebral compression fracture can warrant immediate referral to the surgical team.<sup>29</sup> Primary care physicians should also consider referral for surgical management if the level of pain, disability, or kyphotic deformity is substantial, or if there is no response to nonsurgical management over 4-6 weeks. Additionally, if there is progressive kyphosis on follow-up radiographs, spine surgeon referral should be initiated.

Two common minimally invasive treatments utilized in the surgical management of vertebral compression fractures are kyphoplasty and vertebroplasty, in which bone cement, or polymethylmethacrylate, is injected into the fractured vertebral body (cement augmentation) (Figure 3). These have become common treatment options and can be

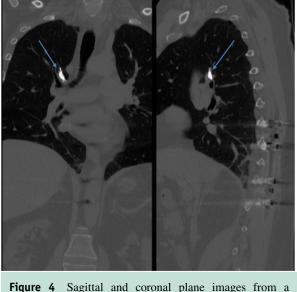


**Figure 3** T8 kyphoplasty. Intraoperative images demonstrating balloon inflation and injection of cement during a one-level kyphoplasty with partial restoration of vertebral body height and angulation.

undertaken as an inpatient or outpatient procedure. Physicians can refer to one of several specialties for these procedures, including orthopedics, neurosurgery, interventional pain management, and interventional radiology.<sup>35</sup> Regional variations in practice patterns may dictate which specialty to refer to for cement augmentation. Indications include recent painful fracture due to osteoporosis or pathological causes including myeloma, lymphoma, or metastasis. Contraindications for these procedures include complete vertebral collapse (vertebra plana), coagulation disorders, and unstable fractures.<sup>35</sup>

In a vertebroplasty, physicians use image guidance, typically fluoroscopy, to percutaneously inject cement into the cancellous bone of the fractured vertebral body, with the aim of alleviating pain and preventing deformity and further loss of height.<sup>36</sup> Kyphoplasty was later introduced as a modification of vertebroplasty by inflating a balloon tamp to create a cavity within the vertebral body. Theoretically, this reduces the pressure needed to inject the cement and minimizes extravasation.<sup>36</sup> Additional benefits of kyphoplasty include restoration of the vertebral body height and reduced kyphosis, although clinical studies have found both procedures to be similarly effective and there are no differences among patient-reported outcome measures.<sup>36,37</sup> Overall complications are rare, ranging from <2% if performed for osteoporosis and up to 10% for pathological fractures caused by malignancy.<sup>36</sup> Most notably, extravasation can occur, which may result in compression of neural elements, leading to radiculopathy or embolization events. Further compression can also be caused if the fracture pattern is burst, instead of compression, and therefore cement may push the posterior vertebral body into the spinal canal. Figure 4 demonstrates a patient suffering from a pulmonary embolism due to cement extravasation, which was asymptomatic. Kyphoplasty is associated with a reduced risk of cement leakage and therefore, this may be a preferred treatment option for patients in whom this is a great concern.<sup>37</sup>

To ascertain the efficacy of these procedures, a randomized control trial was performed to compare vertebroplasty with a control therapy (simulated procedure without cement). The study reported that the improvements in pain and disability were similar.<sup>38</sup> However, this study was severely limited, as patients were allowed to cross over to the other study group after 1 month if adequate pain relief



**Figure 4** Sagittal and coronal plane images from a patient suffering a pulmonary embolism (shown by blue arrow) secondary to cement extravasation.

was not achieved. By 3 months, 51% of patients in the control group switched to vertebroplasty, and therefore, this complicated the interpretation of long-term outcomes as an intention-to-treat analysis was performed and the results of this study should not be utilized as an argument against cement augmentation. Halvachizadeh et al<sup>39</sup> performed a systematic review and meta-analysis of randomized control trials that focused on the outcomes of kyphoplasty, vertebroplasty, or nonoperative management of osteoporotic vertebral fractures. They reported favorable evidence for surgical intervention with superior pain outcomes compared with nonoperative management. Based on these studies, surgical treatment may be the preferred approach if pain is impacting mobility.

## CONCLUSION

Vertebral compression fractures are the most common complication of osteoporosis. Although many patients are asymptomatic and diagnosed incidentally, several experience debilitating pain and disability. Diagnosis is often initially made with plain radiographs, although computed tomography and MRI imaging may be required to evaluate for subtle bony changes, non-contiguous fractures, malignancies, or if there are neurological deficits on examination. Patients can be initially managed non-surgically, but if this fails and results in persistent pain, kyphotic deformity, pseudoarthrosis, or neurological deficits, cement augmentation via vertebroplasty or kyphoplasty should be considered.

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