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Vertebral compression fractures: What time destroys, methylnmethacrylate may mend

WITH ADVANCES in medicine, life expectancy continues to improve, making care of the elderly a greater part of all of our practices, regardless of specialty. And as osteoporosis is a particular problem in the aged, its sequelae present numerous clinical challenges.

Dr. Mazanec and colleagues, in their article on vertebral compression fractures in this issue of the *Cleveland Clinic Journal of Medicine*,¹ should be congratulated on an excellent discussion of an increasingly important problem.

■ OSTEOPOROSIS VS TRAUMA

Mazanec et al appropriately highlight the difference between osteoporotic compression fractures and traumatic fractures of the thoracolumbar spine. This is an important distinction, as it influences management.

This distinction should be made first by history and then, by radiographic imaging.

History and presentation

Osteoporotic compression fractures generally occur in elderly and postmenopausal women after low-energy stresses, such as picking up a baby or a bag of groceries, or sneezing. However, these injuries should not be overlooked in elderly men.²

Patients with osteoporotic compression fractures sometimes present with low back pain, particularly if the lesion is in the low

lumbar region. However, fractures are more common in the lower thoracic and upper lumbar region, likely because it is a transition zone between the relatively stiff thoracic vertebrae and the more mobile lumbar segments. The clinical presentation, therefore, is more typically mid-to-low thoracic pain.

Furthermore, the pain is not necessarily mechanical in nature. While pain can be exacerbated by movement, fracture pain is generally constant and dull. Importantly, complaints of low back pain in an osteoporotic patient should alert the clinician to the possibility of a sacral insufficiency fracture. This can be diagnosed by a bone scan, which displays the hallmark “H-pattern” of increased uptake. These fractures respond to limited bed rest and progressive mobilization.

High-energy traumatic fractures can and do occur in patients of any age, male or female, though they are more common in younger men engaged in high-risk activities. They most commonly occur at the T12–L1 region.

Radiographic appearance

Osteoporotic compression fractures, by definition, occur in osteoporotic bone. On plain radiographs, the vertebrae appear to be “washed out” with loss of detail of the bony contours. It is also important to examine the sacrum and iliac wings and to note any loss of the dense cortical bone that is normally present in these broad bony structures.

But compression fractures often occur in nonosteoporotic bone as well. These fractures usually appear as a simple wedge fracture,

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This paper discusses therapies that are experimental or that are not approved by the US Food and Drug Administration for the use under discussion.



which can result in acute kyphotic deformities at that level. While some osteoporotic fractures are the simple wedge type, more often they are of two other types: biconcave (ie, both the inferior and superior vertebral end plates are pushed in) or crush (ie, uniform height loss). The latter two types rarely occur after high-energy traumatic lesions.

■ TREATMENT OF TRAUMATIC FRACTURES

Treatment of traumatic vertebral fractures is based on the amount of residual stability. This is influenced not only by the extent of fractured bone, but also by the integrity of the spinal ligaments, which is assessed with plain radiographs, computed tomography, and magnetic resonance imaging.

If a fracture is considered unstable, it is usually treated with open surgery that may consist of internal fixation with hardware. The goal is to protect the neurologic structures and to decrease pain and deformity.

■ TREATMENT OF OSTEOPOROTIC FRACTURES

Osteoporotic compression fractures are inherently stable injuries without ligamentous injury. The kyphotic deformity develops over time and is primarily from loss of vertebral height at the fracture or fractures. In most cases, the neurologic structures are not at risk from either the deformity or the fracture itself. The main complaint, and the focus of clinical management, is pain.

Kyphoplasty and vertebroplasty

The treatment of painful osteoporotic compression fractures has advanced considerably with the development of minimally invasive fracture stabilization in the form of vertebral augmentation (ie, kyphoplasty and vertebroplasty). The clinical results of these techniques have been extremely encouraging, demonstrating pain relief in 95% to 100% of patients treated.³⁻⁷

Kyphoplasty, in particular, has enjoyed considerable publicity because it can reduce vertebral compression fractures and stabilize them for pain relief. As symptomatic kyphosis is often the result of multiple compression

fractures, kyphoplasty, in contrast to vertebroplasty, can be used to correct or prevent such deformity.

It is commonly believed that correcting the deformity may biomechanically reduce further fracture risk at other levels. While this remains to be proved in a prospective clinical series, kyphoplasty is the only method of vertebral augmentation that has demonstrated the ability to correct kyphosis.^{2,4} Vertebroplasty does not have this ability and should be considered only as a method of pain relief.

Braces are important, underused

Bracing is an important component of nonoperative management of osteoporotic compression fractures, in addition to proper pharmacologic and analgesic therapy.

Various types of braces are available. A shell-type device, such as a thoracolumbosacral orthosis, offers stability during rotation, flexion, and extension. However, because of the encasing plastic shell design, patients often complain of uncomfortable itching and sweating underneath the brace.

Shell-type braces are useful in the treatment of acute high-energy traumatic fractures. While some surgeons might prescribe them for osteoporotic compression fractures, they are generally considered “overkill.” The deforming forces pushing the spine into kyphosis can be overcome by a hyperextension brace, such as a Jewett-type device, which is much less bulky, more comfortable, less expensive, and easier to apply and remove.

Unfortunately, poor overall patient compliance with brace therapy is a major factor limiting its effectiveness.

■ NEUROLOGIC SEQUELAE

Mazanec et al¹ briefly discuss the neurologic sequelae of osteoporotic spinal fractures. Neurologic compromise is due to compromise of the spinal canal.

Compromise of the thoracic spinal canal leads to myelopathy, which presents as an upper motor neuron condition. Hyperreflexia in the lower extremities, clonus, and varying patterns of motor and sensory deficit can be present.

Compromise of the lumbar spinal canal compresses the cauda equina, resulting in a

Osteoporotic fractures are most common in the lower thoracic and upper lumbar region



presentation similar to degenerative lumbar stenosis. This is a lower motor neuron phenomenon, leading to hyporeflexia in the lower extremities, which may or may not be accompanied by motor or sensory deficit.

Senile burst fractures. Neurologic injury is extremely rare with osteoporotic compression fractures, but is more characteristic of a senile burst fracture—an osteoporotic fracture that extends to the posterior aspect of the vertebral body. Not all senile burst fractures cause spinal canal encroachment, however.

On plain radiographs, senile burst fractures can appear similar to compression fractures. Computed tomography or magnetic resonance imaging can better demonstrate the retropulsion of bony fragments into the spinal canal that is characteristic of this injury.

This lesion is better treated with open anterior decompression and stabilization with hardware to maximize neurologic recovery.

It is not amenable to kyphoplasty or vertebroplasty because of the risk of cement extravasation into the spinal canal through the fracture site.

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