

BRIEF REPORT

Kummel Disease: A Not-So-Rare Complication of Osteoporotic Vertebral Compression Fractures

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Kummel disease is the eponym for avascular necrosis of the vertebral body after a vertebral compression fracture. As our population ages, the prevalence of osteoporosis, its most common fragility fracture (vertebral compression fracture), and Kummel disease will increase. The purpose of this article is to report a prototypical case with complete radiographic and histologic workup and to provide facts regarding Kummel disease that are salient to the primary care provider. (J Am Board Fam Med 2009;22: 75–78.)

Osteoporosis afflicts more than 20% of the population older than 50, with a significant predilection for thin, white females.^{1,2} Annually, 700,000 new vertebral compression fractures (VCFs) are diagnosed in the United States. Most VCFs are stable and heal without complication. Historically, VCFs have been treated expectantly with bracing for comfort and pain medications. The advent of vertebroplasty and kyphoplasty has marked a new era in the treatment of VCFs. These cement augmentation techniques produce immediate and effective results in more than 85% of patients treated.³ These new and successful interventions have prompted a closer evaluation of VCFs presenting in the primary and urgent care settings, specifically in the form of magnetic resonance imaging (MRI) and computed tomography (CT) imaging studies. One consequence of this heightened attention has been the incidental identification of a unique and not-so-rare complication of VCFs: avascular necrosis or atrophic nonunion.

Avascular necrosis (AVN) of the vertebral body after trauma is not a new finding; in fact, its eponym, Kummel disease, was coined in deference to

the first physician to characterize this pathology in 1895. Since then, AVN of the vertebral body, specifically after VCFs, has been reported sporadically in spine specialty journals.⁴ The incidence of this finding is difficult to accurately report because multiple synonymous terms have been used to describe this pathology: posttraumatic vertebral osteonecrosis or AVN, vertebral pseudarthrosis, intervertebral vacuum, cleft or gas, delayed vertebral collapse, and VCF nonunion.^{3–8} Regardless of terminology, the true incidence is actually quite high (7% to 37%), especially among the elderly population.^{4,5} For consistency we will refer to this pathology as Kummel disease in this article. The importance of Kummel disease to the primary care physician is highlighted in the following prototypic case.

Case History

A 78-year-old man with known osteoporosis was admitted urgently to our medical center for severe acute progression of his chronic low back pain and neurogenic claudication. A CT scan was interpreted and showed a pathologic fracture of the L3 vertebral body secondary to osteomyelitis with associated psoas abscess. Routine laboratory studies were negative. CT-guided drainage of the apparent psoas abscess yielded nonpurulent serosanguinous fluid; cultures of the fluid were negative. Throughout the hospital stay, the patient was afebrile and hemodynamically and neurologically stable.

The spine surgery service was consulted and the diagnosis of Kummel disease was established based on characteristic imaging findings (Figure 1). Subsequently the patient underwent a posterior de-

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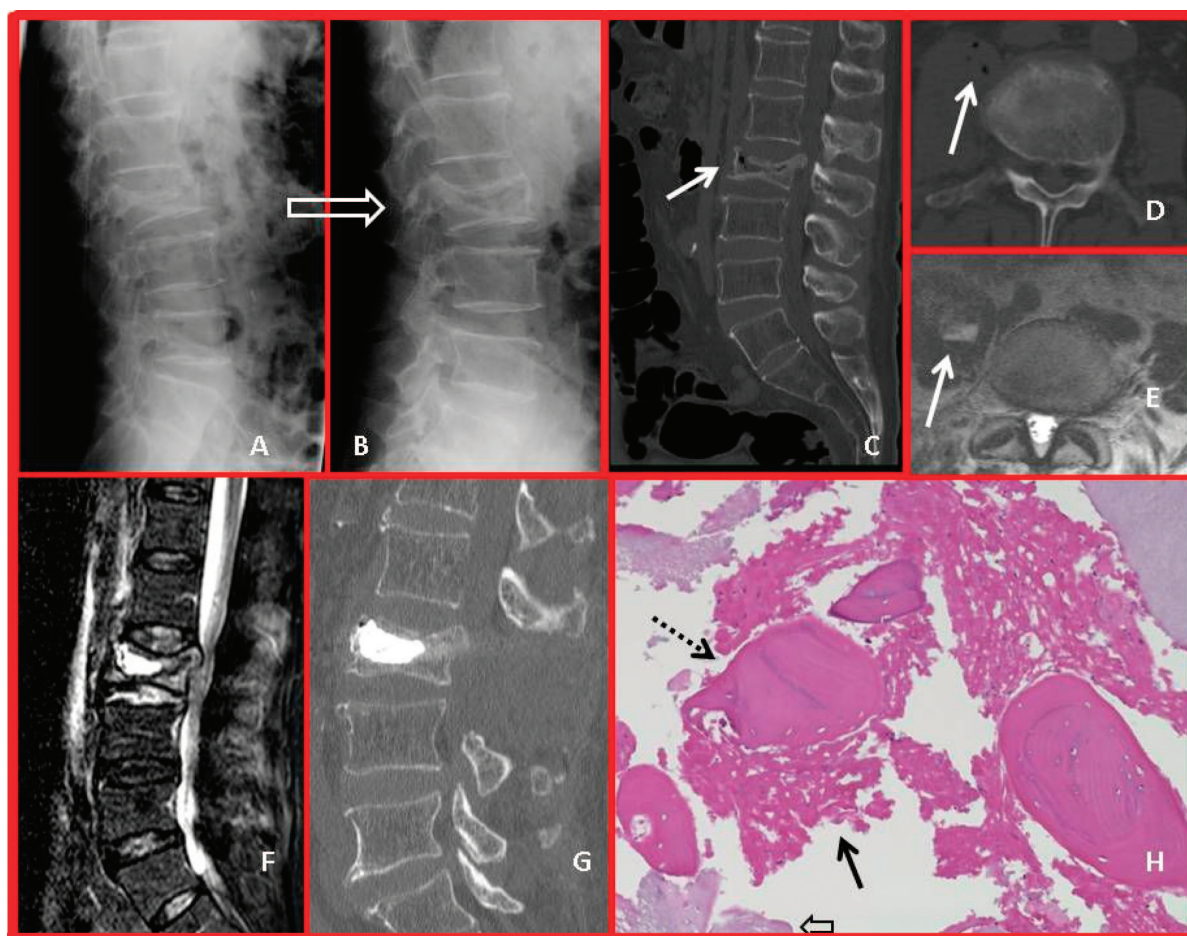


Figure 1. The following sequence of images illustrates the salient radiologic and pathologic findings in Kummel disease. In flexion, the fracture compresses through the avascular zone directly beneath the superior endplate, demonstrating the maximal degree of height loss, but in extension, the fracture plane gaps open (A and B). This motion (pseudarthrosis) is responsible for the vacuum effect, which pulls transudative fluid and nitrogen gas (white arrow, C) into the fracture site. Intervertebral gas is a radiographic and computed tomographic finding indicative of Kummel disease. In this case, the fluid and gas escaped from the fracture and descended along the right psoas muscles, creating the false appearance of a psoas abscess (D). Confirming this explanation of the fluid collection over the right psoas, the fluid collection is benign in appearance; there is no associated soft-tissue edema on T2 magnetic resonance images (MRIs) (E). Sagittal STIR MRI, the image modality of choice for diagnosing acute VCFs, shows fluid (homogeneously bright white area) in the fracture cleft, with air bubbles (black dots). Fluid filled clefts on MRIs are characteristic of Kummel disease and occur because patients undergoing MRI lie supine for ≥ 40 minutes, which seems to be enough time to allow transudative fluid to replace intervertebral gas. Note, the vertebral body height is greater on the MRI (F) than on the flexion radiograph (A), and this is because the supine position extends the spine. The most important finding on the MRI is that the adjacent level endplates and vertebral bodies (L2 and L4) are completely normal. Spondylodiscitis or vertebral osteomyelitis would have destroyed and/or inflamed the adjacent disk spaces and endplates. The pathopneumonic finding in this sequence of images, is the intervertebral gas, pseudarthrosis and lack of adjacent inflammation. The postoperative sagittal CT-recon image shows the results of open kyphoplasty, with laminectomy (note that the L3 spinous process is absent) and improvement in L3 vertebral height (G). H: The final image is a high power view (original magnification, $100\times$) of the biopsy obtained at surgery, which shows areas of necrotic bone (dotted arrow); immature, woven bone (solid arrow); and nests of cartilage, all which are pathopneumonic for Kummel disease.

compression and open kyphoplasty. An open biopsy of the L3 vertebral body demonstrated osteonecrosis without evidence of infection, which confirmed the diagnosis of Kummel disease. The patient's pain reduced after surgery and he continued to do well at a 6-week follow-up.

Discussion

Kummel disease represents a failure of the fracture healing process, for which there are no good nonoperative treatments.^{5,6} An avascular zone develops below the superior endplate, eliminating healing potential and resulting in an atrophic or avascular nonunion. Patients with mobile VCF nonunion and chronic back pain (>3 months) with or without symptoms of neurogenic compression are candidates for surgical stabilization.^{6,8,9} Although kyphoplasty and vertebroplasty provide excellent and immediate pain relief by stabilizing the fracture cleft, they do not address symptoms stemming from neurologic compression.^{3,6-9} Our patient had baseline lumbar spinal stenosis, which acutely progressed. Thus, a posterior decompression was performed to relieve concomitant neurogenic claudication and potentially reduce back pain. With cement augmentation at the spinal levels being decompressed, posterior decompression is performed in an open fashion because the vertebral pedicles are visible through the operative field; otherwise, cement augmentation alone is a same-day percutaneous procedure.^{8,9} Some patients, typically those with multiple levels of previous VCFs, develop a posture that hunches forward and is referred to as fixed positive sagittal imbalance. In these cases kyphosis correction through osteotomies and instrumented spinal fusion can be performed with good to excellent outcomes.¹⁰

Plain upright anteroposterior and lateral radiographs are the standard initial study for Kummel disease. Flexion/extension radiographs may be ordered to detect pseudarthrosis (motion through the vertebral body), which, along with an intervertebral air-filled cleft, are considered classic for Kummel disease. CT scans provide improved detail of the avascular nonunion but are not always necessary to identify these characteristics. MRI scans are limited to ambiguous cases or to evaluate underlying spinal pathology, like spinal stenosis, which may alter the plan for intervention.

This case illustrates the common symptoms, patient presentation, and imaging results of Kummel

disease. The common symptoms and clinical presentation are back pain, often acute or chronic at onset, with or without neurogenic claudication in a patient with known osteoporosis. The findings from classic radiograph and CT scans in this case include pseudarthrosis, intervertebral air, and the lack of adjacent inflammatory changes on MRI.⁵⁻⁷ Knowledge of the characteristics of Kummel disease can expedite care and reduce potentially unnecessary procedures. The diagnosis of Kummel disease can reliably be made based on classic imaging findings (Figure 1). In the absence of progressive neurological deficit or signs of infection, vertebral cement augmentation can be performed expeditiously without additional workup, with the expectation of immediate and good to excellent resolution of pain.

Conclusion

AVN after VCF (Kummel Disease) is a potential complication in up to one-third of VCFs.⁵ As such, there is a reasonably good chance that most primary care providers will be involved in the treatment of a patient with Kummel disease at some point in their career. This case and the figure highlight the distinct radiographic findings (intervertebral air, pseudarthrosis, and normal adjacent levels), which reliably differentiate this condition from more morbid ones like spondylodiscitis.

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