Although complications related to vertebroplasty or kyphoplasty are few, we treated 2 patients with vertebroplasty or kyphoplasty for pain, presumed to be due to vertebral compression fractures, which were subsequently found to be due to occult osteomyelitis/diskitis. The onset of their infections appeared to have preceded their vertebral body augmentation procedures and was possibly due to prior interventional procedures for histories of back pain.

An 86-year-old woman had had 3 prior kyphoplasty procedures for fractures at T10, T11, and L1. She reported continued severe pain, and subsequent magnetic resonance imaging was misinterpreted for another fracture at T12, resulting in her fourth kyphoplasty. She became septic and had some improvement with antibiotics, but she declined specialty care and died. A 74-year-old man with chronic back pain had recently undergone lumbar facet joint injections. Computed tomography and subsequent bone scan found uptake at both L2 and L3. Despite abnormal erythrocyte sedimentation rate and C-reactive protein level and normal radiographic vertebral height, he underwent a vertebroplasty. His pain increased, and subsequent workup found L2-3 diskitis. He recovered with antibiotics and specialty care. Similar to prior reports of spondylodiskitis, both patients had multiple medical comorbidities.

This article emphasizes the need for clinical reevaluation and scrutiny in the interpretation of imaging studies, including for infection in patients with continued pain after spinal procedures. The differential diagnosis of infectious etiology is an important consideration prior to vertebral cement augmentation for presumed fragility fracture.

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Vertebroplasty and kyphoplasty are popular in the treatment of acute osteoporotic vertebral compression fractures and have a low complication rate.1-3 This article describes 2 patients with unusual complications. They were referred for severe disabling pain after a recent vertebroplasty/kyphoplasty performed elsewhere. They were found to have diskitis, but unlike prior reports, they had multiple spinal procedures, and the onset of their diskitis could not be definitely identified.

CASE REPORTS

Patient 1

An 86-year-old woman presented to the author’s institution after she underwent 4 kyphoplasty procedures elsewhere. Her medical history was significant for atrial fibrillation and secondary stroke, hypertension, osteoporosis, gastritis, coronary artery disease, atherosclerosis, aortic aneurysm, constipation, depression, and rheumatoid arthritis.

Initially she presented to her local clinic and was referred to a local orthopedic spine surgeon. She had a 1-month history of thoracolumbar back pain unrelated to trauma, and magnetic resonance imaging (MRI) was diagnostic for a compression fracture at T10. She underwent kyphoplasty at T10 after preoperative antibiotic of intravenous cefazolin. Due to persistent pain over the following 2 months, a repeat MRI scan identified a new compression fracture at T11, for which she underwent a kyphoplasty procedure with preoperative cefazolin antibiotic. Postoperatively, she had minimal pain improvement.

Approximately 1 month later, the patient had a fall with recurrent severe back pain and a left wrist fracture. Plain radiographs and MRI revealed a new compression fracture at T11, for which she underwent a kyphoplasty procedure with preoperative cefazolin antibiotic. Postoperatively, she had minimal pain improvement.

Over the next 2 months, and despite no new history of trauma, the patient’s symptoms progressed. Spinal bracing was again attempted, but she remained confined to a wheelchair because standing was intolerable. An MRI scan was interpreted as a new compression fracture at T12 (Figure 1), for which the patient underwent her fourth kyphoplasty with preoperative intravenous cefazolin. The patient’s symptoms remained severe, and 6 weeks after her T12 kyphoplasty (8 months after her first kyphoplasty), she presented to the author’s institution for a second opinion.

At presentation, the patient gave a vague history of fevers, chills, sweats, and an approximately 20-pound weight loss. She rated her back pain as 10 on the visual analog scale (VAS) for pain (range, 0 [no pain] to 10 [worst pain possible]) when she was not lying down, with radiation from the thoracolumbar region to the lumbar region and into both thighs. Her Oswestry Disability Index (ODI) was 93. She was unable to stand or ambulate due to severe pain back pain; however, she was neurologically intact. Given the worrisome clinical picture of possible sepsis, hospitalization was advised. The patient declined admission to our tertiary referral center and was instead admitted to her local community hospital, where she consulted with hospitalists.

Her blood tests revealed a normal white blood cell count of 8.2 and highly elevated erythrocyte sedimentation rate (ESR) of 98 and C-reactive protein (CRP) level of 7.8. Updated radiographs and MRI revealed diskitis with bony erosion at T12-L1 and evidence of osteomyelitis of the adjacent vertebrae (Figure 2). The patient underwent a computed tomography (CT)-guided biopsy of the T12-L1 disk, which grew methicillin-sensitive Staphylococcus aureus. She was treated with a brace; antibiotics (nafcillin for 6 weeks); multiple opioids for pain; and teriparatide, vitamin D, and calcium for her osteoporosis. After nafcillin antibiotic administration, the patient’s ESR was 31.

The patient’s pain remained severe, and her activity level consisted of bed-to-chair mobilization. The patient declined follow-up spine MRI and radiographs or
Case Report

spine specialty care. Two months after discharge, the patient was readmitted for hypotension and somnolence. She was found to have a clostridium difficile colitis with secondary metabolic acidosis and electrolyte abnormalities, a urinary tract infection, anemia, and hypoxemia. She was treated with vancomycin for 2 weeks. Despite a persistently elevated ESR (range, 56-79), recommended spine evaluation was again declined, and she returned to hospice care for continued opioid treatment of her back pain. Shortly thereafter (4 months after she presented to the author’s institution), she died. The extent to which her diskitis resolved was not known.

Patient 2

A 74-year-old man with chronic back pain underwent a left L4-5 diskectomy 12 years prior to presentation to the author’s institution. His medical history was significant for type 2 diabetes mellitus, chronic obstructive pulmonary disease, coronary artery disease, paroxysmal atrial fibrillation, ischemic cardiomyopathy (for which he has a cardiac defibrillator), hypertension, aortic stenosis, peripheral vascular disease, gastroesophageal reflux, hypercholesterolemia, anxiety, and depression.

Despite 2 months of physical therapy, traction, and opioid pharmacological treatment for recurrent moderate low back pain, his pain progressed, at which point he was hospitalized. Tests revealed a normal white blood cell count of 5.6 and an elevated ESR of 45 and CRP of 14.1. Radiographs and a lumbar CT scan revealed all levels to have mild facet arthritis and mild diffuse annular bulge, a calcified disk herniation, and laminctomy defect at L4-5, but no fracture. After spine consultation at another institution, the patient underwent separate left L4-5 and L5-S1 facet joint injections, after which he was transferred to a transitional care facility.

Due to continued pain, the patient sought a second opinion at a different institution. Although repeat radiographs were interpreted as normal for age, a bone scan and single-photon emission CT revealed increased uptake on immediate blood pool, midphase, and delayed images. The scans were interpreted to show compression fractures at both L2 and L3 (Figure 3). The patient underwent a trial of bracing and then vertebroplasty at L2 and L3 but with a small amount of cement extravasation into the disk space (4 months after the onset of back pain) at the other institution. There was no documentation as to whether preoperative antibiotics were administered.

After 2 days, the patient’s pain again became intolerable (VAS=8), and he was again hospitalized and treated with high-dose parenteral narcotics. Subsequent blood tests found a normal white blood cell count of 4.6 but a markedly elevated ESR of 91 and CRP of 11.2. Radiographs and repeat CT identified vertebroplasties at L2 and at L3, as well as endplate erosion and a loss of disk height at L2-3 as compared to the CT scan taken 6 weeks earlier (Figure 4). The author’s institution was consulted for a third opinion.

Percutaneous needle biopsy of the L2-3 disk space isolated coagulase-negative S. aureus. His diagnosis of L2-3 diskitis and osteomyelitis was treated with intravenous vancomycin and ceftriaxone for 8 weeks, followed with oral doxycycline for an additional 6 weeks and bracing again after adjusting the fit. At follow-up, the patient had dual-emission X-ray absorptiometry bone density testing, which revealed osteoporotic T scores of −3.0. The patient was started on teriparatide, vitamin D (50,000 units/week), and calcium citrate (800 mg twice daily). During the subsequent year, the patient’s L2-3 disk space autofused, his pain completely resolved, he discontinued all pain medication, and he reassumed all normal activities of daily living. His VAS back pain score was 0.5 and his ODI score was 4.

Discussion

Both patients reported here had osteoporosis and presumed compression fractures despite no plain radiographic loss of vertebral height or endplate discontinuity. Common to both patients was incapacitating pain at presentation, numerous comorbidities, cement extravasation into the adjacent disk space, and a delay in diagno-
six. Reports of 10 prior cases of infection subsequent to vertebroplasty commonly found that patients presented with severe disabling pain and had comorbidities such as diabetes, immunocompromise, prior lumbar surgery, or recent infection. These are also risk factors for spondylodiskitis unrelated to vertebroplasty.17-21

The patients in the present article differed from prior reports in 1 important aspect: variable histories of back pain due to noninfectious etiology overlapped or masked their diskitis pain (ie, the onset of diskitis in our patients was not clear) because they had back pain for other concurrent reasons including additional fragility fracture, spondylolysis, degenerative disk disease, and stenosis. Additionally, unlike the prior reports where a single prior procedure (vertebral body cement augmentation) could be identified as a point of contamination, our patients had multiple and various spinal procedures (prior kyphoplasties and spinal steroid injections), which made identification of the onset of their infection uncertain.

Compounding the diagnostic challenge of persistent back pain after recent intervention was the misinterpretation of the imaging studies just prior to their vertebroplasty/kyphoplasty. A reinterpretation of patient 1’s MRI prior to the most recent kyphoplasty was consistent with diskitis with fluid adjacent to her prior L1 kyphoplasty cement, and signal changes in the adjacent vertebral body.16 The 3-phase bone and single-photon emission CT scans in patient 2, who was unable to undergo MRI, had increased uptake in all 3 phases in 2 adjacent vertebral levels with no plain radiographic evidence of loss of vertebral height. Although highly sensitive but not always specific, differential diagnosis for the bone scan interpretation, with an elevated ESR, includes an infectious process.17-21

The 2 cases reported here serve as a reminder to practitioners that despite the relative success of vertebroplasty or kyphoplasty, the enthusiasm for these techniques should be tempered in the frail elderly patient with multiple comorbidities. The patients in this report demonstrate the difficulty in the diagnosis of back pain in frail patients who have failed vertebroplasty or kyphoplasty for presumed compression fracture. Failed vertebroplasty requires rigorous re-evaluation for other potential sources of pain. Additionally, multiple procedures for back pain may confound the clinical picture and mask an underlying diskitis. An additional factor complicating patient care for both cases was that the physicians performing the vertebral cementing procedures were not involved in follow-up care of the patient. Not uncommonly, patient follow-up care may be with the family physician or other non-spine health care provider, which has the potential to delay recognition of complications and their appropriate treatments. Delayed treatment may result in poor outcomes.22-23

This article is the first to present vertebral body cement augmentation in the face of an occult diskitis. Conservative treatment with a well-fitting brace combined with aggressive medical treatment of osteoporosis may still be a prudent option for frail patients with presumed acute vertebral compression fractures while they undergo the appropriate workup to confirm the diagnosis for their back pain.

REFERENCES


