

# Case 2

## Multilevel Vertebroplasty

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### Clinical Presentation

A 78-year-old woman presented with new, severe back pain. This patient was a frail, thin person with chronic obstructive lung disease, congestive heart failure, atrial fibrillation, hyperlipidemia, hypertension, asthma, a 50 pack year smoking history osteoporosis, and she was taking oral steroids. The patient had undergone a prior L3 compression fracture and subsequent percutaneous vertebroplasty (PV). This resulted in good pain relief for the acute symptoms.

### Imaging Findings and Procedures

A magnetic resonance image (MRI) (taken November 5, 2003) revealed a new compression fracture at L2 with chronic compressions of L3 (with prior PV) and L4 (Case Figure 2.1). Based on the finding of new compression fracture and a consistent pain location, PV at L2 was performed.

This patient again got good pain relief from the PV at L2. However, within days new fractures occurred, and by December 4, 2003, the patient had undergone PV at T11 and T12 and was complaining of new pain in the low lumbar area. A repeat MRI revealed marrow edema (low signal) in L4 and L5 consistent with additional compression fractures (Case Figure 2.2).

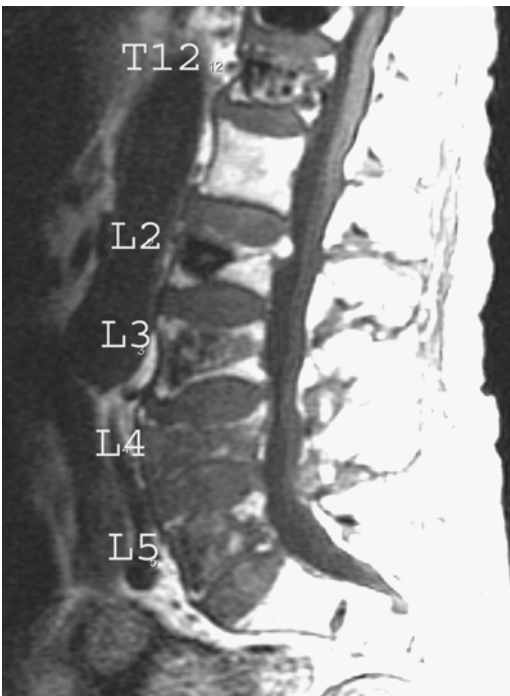
Over a period of approximately 1 year, the patient experienced nine vertebral compression fractures, sacral insufficiency fracture, and right hip fracture. The vertebral compression fractures and the sacral insufficiency fracture were all treated percutaneously with good pain relief and no clinical complication (Case Figure 2.3).

### Discussion

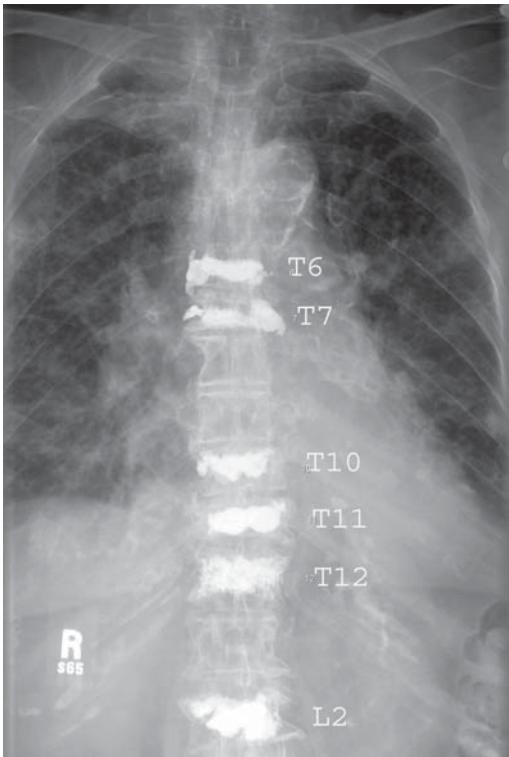
This patient demonstrates the cascade of progressive compression fractures and other osteoporotic fractures that some individuals can experience. It is important for all patients experiencing a compression



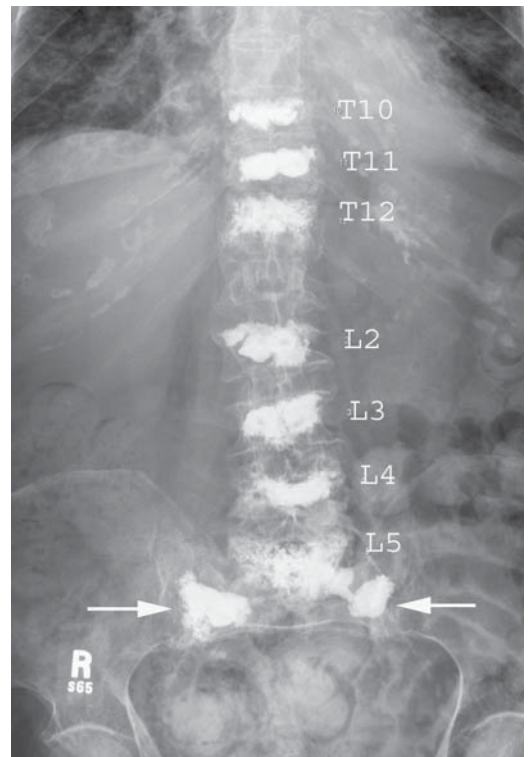
**Case Figure 2.1.** Lateral T1 MRI reveals a new compression fracture at L2 (white arrow). L3 shows central low signal consistent with a prior vertebroplasty (white arrowhead). The bone cement creates a signal void. L4 has experienced a prior compression. The bright signal indicates that this fracture is chronic.



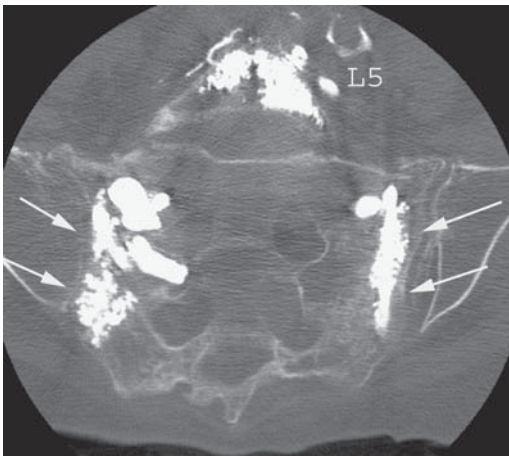
**Case Figure 2.2.** Lateral T1 MRI now showing vertebroplasties at T11, T12, L2, and L3. New compression fractures are present at L4 and L5.



A



B



C

**Case Figure 2.3.** (A,B) Anteroposterior radiographs show nine vertebral levels that have been treated with percutaneous vertebroplasty as well as sacroplasty (white arrows). (C) Coronal CT images of the sacroplasty (white arrows).

fracture to undergo appropriate workup and pharmacologic therapy in an attempt to avoid future fractures. Even with this help, progressive debility due to additional fractures can occur. The additional fractures can usually be treated with PV or kyphoplasty. It is possible that, for such patients, prophylactic vertebral therapy would help avoid future compressions, but we presently have no way of detecting vertebrae that will actually fracture in the future. Also, reimbursement guidelines specifically exclude vertebrae that are not fractured.

Finally, we have no data about the physiologic outcomes following multilevel therapy. For example, older individuals make their blood products progressively in the central marrow space (largely the spine). Will filling these vertebrae result in chronic anemia or force the body to provide blood precursors through extramedullary hematopoiesis (also a pathologic condition)?

Multilevel therapy, although controversial for many reasons, is nonetheless indicated for patients who present with multiple fractures or experience repeated fractures over time. The risk of cardiopulmonary complications increases as the number of vertebrae treated increases (during a single session). This happens because every vertebroplasty (or kyphoplasty) pushes marrow fat and blood precursors out of the bone as cement is introduced. This material ultimately is filtered by the lungs, creating pulmonary emboli. Most patients tolerate these events well and have no clinical complications. However, these emboli can result in cardiopulmonary compromise and even death in patients with poor pulmonary or cardiac function. We recommend that patients be assessed for an underlying cardiopulmonary disease that would put them at increased risk from PV or kyphoplasty. Procedures should be minimized for these individuals (limiting the number of vertebrae and amount of cement injected).

Even for patients with normal cardiopulmonary function, the number of levels that can be performed safely is not known. For this reason, we recommend limiting the number of levels treated at one setting to three in patients with normal function. For high-risk patients, more stringent criteria should be considered. Remember, compression fractures do not constitute a medical emergency, and therefore treatment should be timed to maximize safety.

Presently, there are no scientific data to support prophylactic therapy with vertebroplasty or kyphoplasty, and reimbursement is only for compression fractures resulting from osteoporotic or malignant disease. Prophylactic treatment of noncompressed vertebrae should be undertaken only with internal review board approval and with specific discussions with and consent obtained from the patient.