25 Kummel Disease

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INTRODUCTION

Kummel disease was first described by Hermann Kummell in 1891 as delayed posttraumatic vertebral collapse occurring weeks or months after an often minimal injury. The mechanism is still debated. These lesions are most widely believed to be secondary to delayed osteonecrosis of the vertebral body, potentially related to vascular disruption of the anterior watershed territory of the vertebral body following trauma. It is a rarely reported entity, which likely occurs more frequently than recognized. Typically, only one vertebral level is involved within the lower thoracic or upper lumbar spine. Kummel disease most commonly affects middle-age or elderly patients. Unlike the more commonly seen osteoporotic compression fracture, delayed vertebral osteonecrosis (Kummel disease) has a typical clinical and radiographic course. The imaging finding of the "intravertebral vacuum cleft" (Fig. 25.1) has been described on plain film, computed tomography (CT), and magnetic resonance (MR) imaging. Although vertebral clefts are often associated with Kummel disease, the finding is not exclusive to this disease process.

STAGES OF EVOLUTION: OVERVIEW

Initial imaging in patients that develop Kummel disease is classically radiographically occult. This is one of the defining clinical features of the entity. However, many patients who develop Kummel disease are not imaged at the time of initial injury, typically due to the minor severity of the trauma, such as a fall from standing.

Following an asymptomatic period or sometimes minor persistent pain, the patient will experience worsening of pain severity, typically greater than after the initial pain episode with or without neurologic symptoms, such as radiculopathy or bowel/bladder dysfunction.

Imaging at this delayed time point will demonstrate either a vertebral compression deformity or intravertebral "cleft" dependent upon patient positioning during imaging, weight-bearing versus supine. This is the result of a potentially dynamic (unstable) cavity that has developed within the vertebrae. Flexion or weight-bearing radiographs may demonstrate vertebral compression, while extension or supine radiographs may demonstrate a "cleft." Nitrogen-predominant gas may fill the cleft because of the lower pressure within the cavity causing the radiographically described "intravertebral vacuum cleft" (IVC). Although IVC was initially attributed to Kummel disease, this sign has proven not to be specific to this disease process. Other conditions with IVC include intravertebral disc herniations, subacute fractures that violate the vertebral endplate, and even pathologic compression fractures.

IVCs are easily identified on CT; however, they may create confusion or misinterpretation on MRI. On MRI, one might expect hypointense signal related to the trapped gas. However, more typically T2 hyperintense fluid will fill the cavity due to prolonged supine positioning required for MRI allowing time for fluid accumulation. T1 signal is variable, as it depends on the protein concentration of the fluid. There may be an associated "double line sign" in the adjacent vertebral marrow, similar to the appearance of osteonecrosis at other sites (such as the femoral head). This finding likely represents a sclerotic rim with adjacent reactive bone surrounding the cavity.

The five clinical stages are summarized in Box 25.1 and morphologic changes to the vertebral body depicted in Fig. 25.2.

KUMMEL DISEASE EVOLUTION: IN GREATER DEPTH

Kummel disease has been associated with the presence of preexisting vascular risk factors, which has raised the suspicion that the delayed osteonecrosis is related to avascular necrosis. For example, chronic administration of corticosteroids stimulates hyperinsulinemia, which increases intramedullary fat deposition, resulting in crushing of the intramedullary vessels and vascular disruption of medullary arterioles. Chronic microfractures related to osteopenia are other potential eliciting mechanisms of avascular osteonecrosis and subsequent vertebral body collapse (see Fig. 25.1). However, cases that lack a history of trauma should not be referred to as Kummel disease, but instead related to the underlying disorder that has caused avascular osteonecrosis. See imaging examples of sickle cell disease related vertebral osteonecrosis and radiation induced osteonecrosis below.

One study found that IVCs are identifiable with standing lateral radiography, supine cross-table radiography, and MRI, at 14%, 64%, and 96%, respectively. However, several IVCs remain undetected, until they are filled with opaque cement at the time of vertebroplasty. This supports the dynamic nature of the lesion and may explain the underreporting of this entity.

MIMICS AND DIFFERENTIAL DIAGNOSIS

Pathologic compression fractures (Fig. 25.3), sickle cell disease (Fig. 25.4), and radiation therapy (Fig. 25.5) are other causes of osteonecrosis that are distinct from posttraumatic delayed osteonecrosis of Kummel disease.

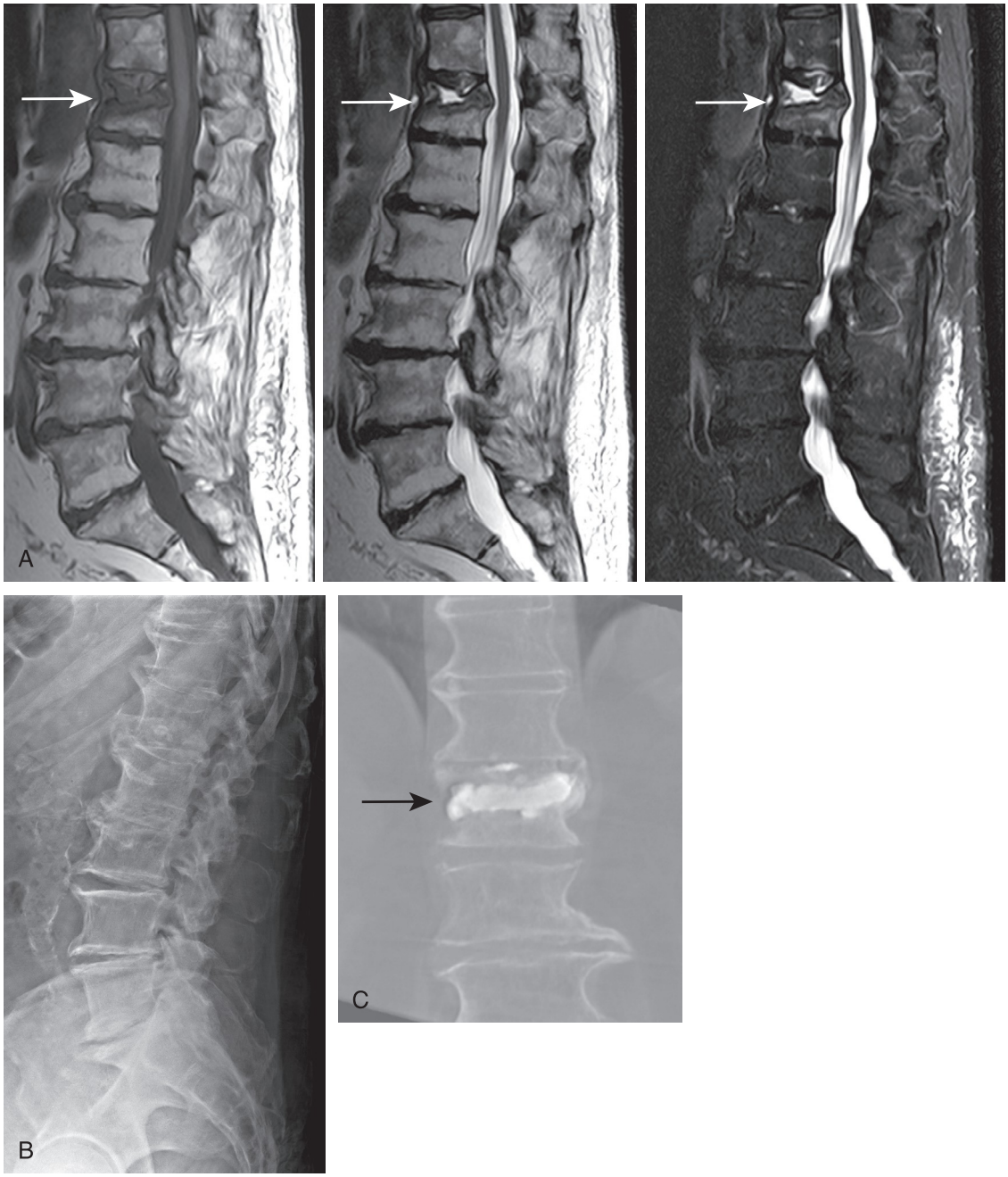

Figure 25.1. Kummel disease. 86-year-old male with reported fall from standing position 8 months prior, now with positional pain aggravated by sitting, and noted to walk in a stooped position. (A) Sagittal T1, T2, and T2 fat-saturated sequences demonstrate fluid-filled cavity within the upper T12 vertebral body with mild vertebral collapse and posterior cortical buckling *(arrows)*. Thick sclerotic changes are also seen along the margin of the cavity without marrow edema. (B) Normal T12 vertebra on lateral lumbar radiograph at time of initial injury 8 months prior. (C) Coronal DynaCT reconstruction demonstrates filling of the osteonecrotic cavity after vertebroplasty *(arrow)*.

BOX 25.1 Five Clinical Stages of Kummel Disease

1. Initial injury: Often minimal trauma or trauma ranging from mild to severe. (Imaging typically not performed, although normal if performed.)
2. Posttraumatic period: Characterized by minimal or minor residual back pain without loss of function (continuation of activities of daily living).
3. Variable latent stage: Patient usually feels well, although minor symptoms may be present.
4. Recrudescent stage: Characterized by increasingly severe back pain. (Imaging at this stage will show vertebral abnormalities of vertebral collapse or an air or fluid cleft depending on patient positioning, i.e., flexion or extention/supine. See Fig. 25.2.)
5. Terminal stage: Vertebral body collapse and kyphosis with potential for neurologic deficit due to instability and spinal canal compromise, if untreated.

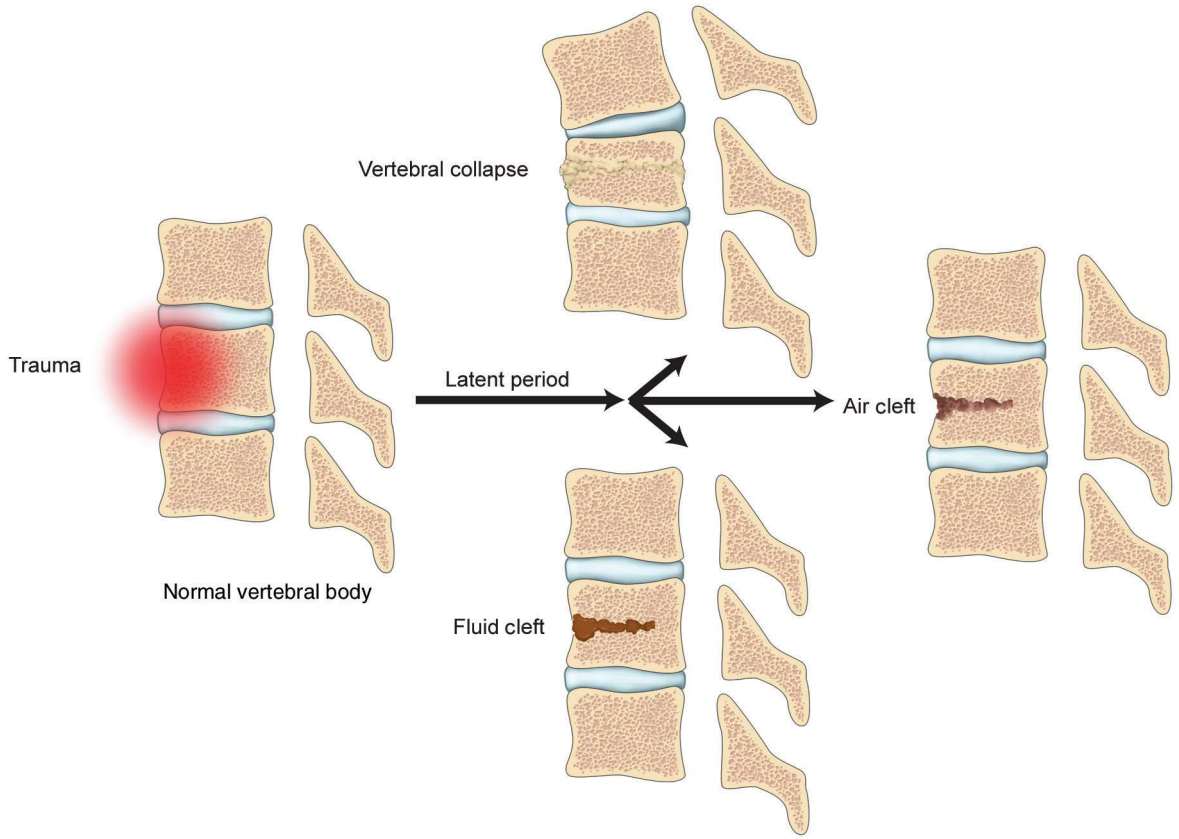

Figure 25.2. Morphologic changes of Kummel disease. Following typically minor trauma to the spine and a variable latent period, vertebral osteonecrosis will manifest as either vertebral collapse or as an air or fluid cleft depending on patient positioning, i.e., flexion or extension/supine. Air cleft is most commonly seen on supine or extension radiographs and supine CT, while fluid cleft is most commonly seen on supine MR related to the longer acquisition time required for MR imaging that allows for fluid to enter the cleft. Notably, small air bubbles may remain in the non-dependent portion of the cleft on earlier acquired MR sequences, which may subsequently dissipate on later sequences.

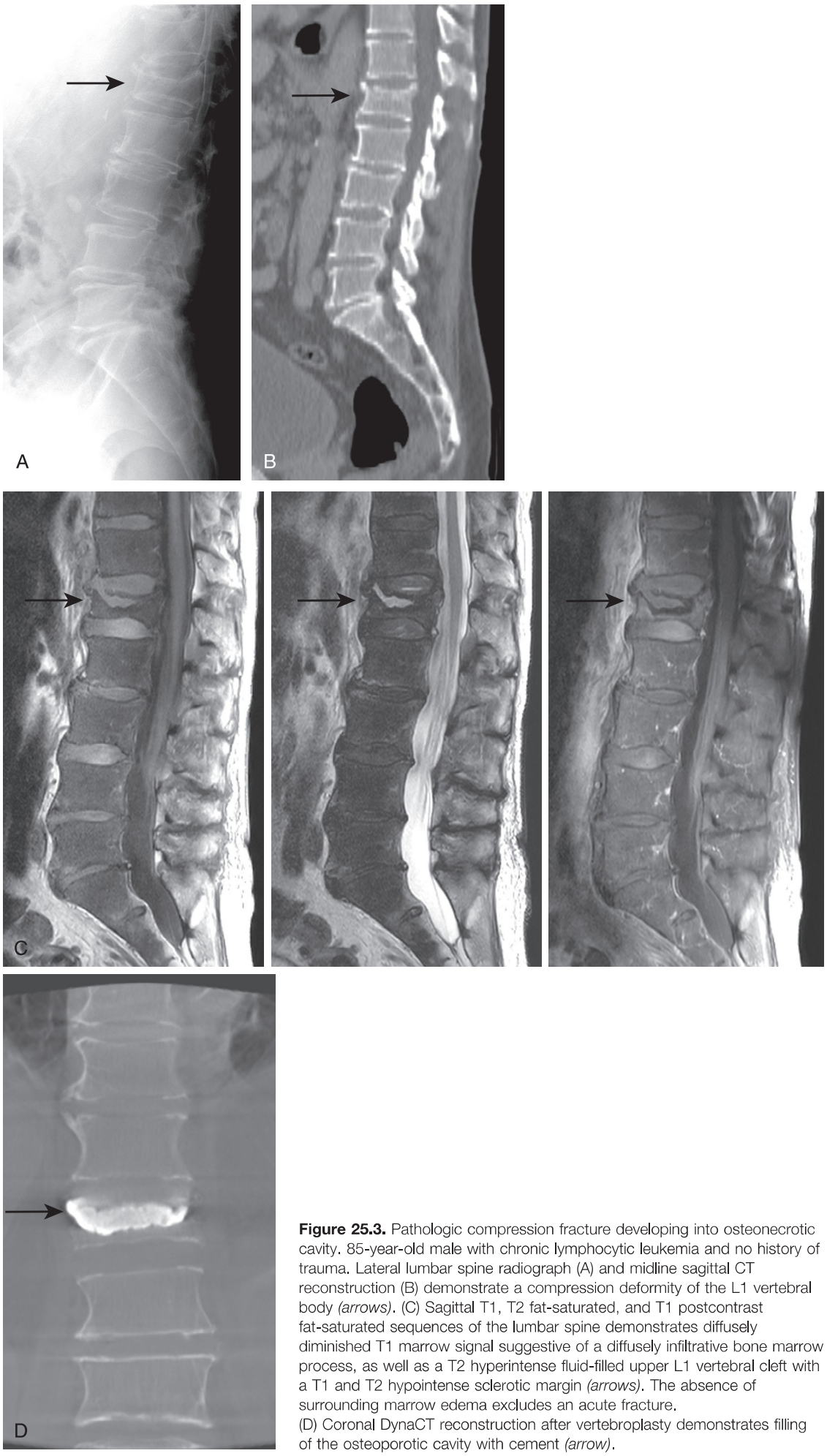

Figure 25.3. Pathologic compression fracture developing into osteonecrotic cavity. 85-year-old male with chronic lymphocytic leukemia and no history of trauma. Lateral lumbar spine radiograph (A) and midline sagittal CT reconstruction (B) demonstrate a compression deformity of the L1 vertebral body *(arrows)*. (C) Sagittal T1, T2 fat-saturated, and T1 postcontrast fat-saturated sequences of the lumbar spine demonstrates diffusely diminished T1 marrow signal suggestive of a diffusely infiltrative bone marrow process, as well as a T2 hyperintense fluid-filled upper L1 vertebral cleft with a T1 and T2 hypointense sclerotic margin *(arrows)*. The absence of surrounding marrow edema excludes an acute fracture. (D) Coronal DynaCT reconstruction after vertebroplasty demonstrates filling of the osteoporotic cavity with cement *(arrow)*.

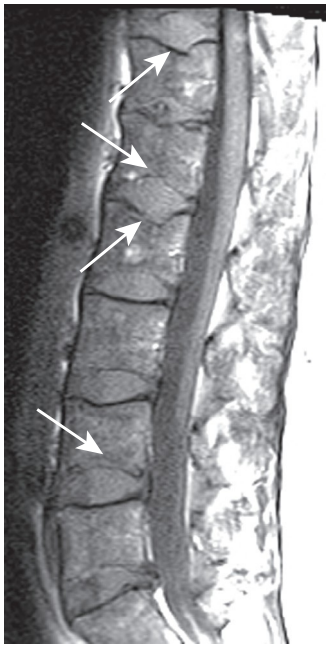

Figure 25.4. A 40-year-old with back pain. Central endplate deformities *(arrows)* in patient with history of sickle cell disease.

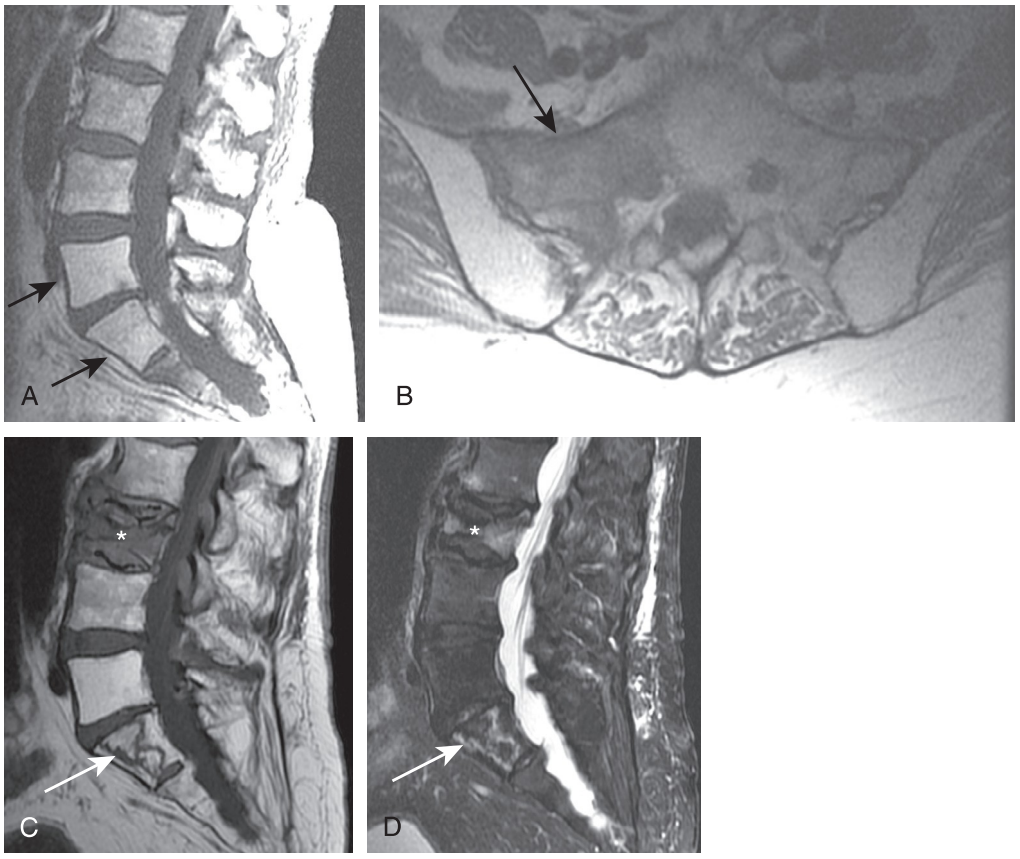

Figure 25.5. A 76-year-old female treated for cervical carcinoma with pelvic radiation. Sagittal T1-weighted imaging reveals hyperintense T1 marrow signal at L5 and S1 *(arrows)* consistent with complete fatty marrow replacement due to prior radiation (A). Axial T1-weighted image reveals a right sacral metastatic lesion *(arrow)* (B). Several years following a second round of pelvic radiation for the sacral metastasis, the patient suffered an acute L3 compression fracture *(*)*. Sagittal T1- (C) and T2-weighted, fat-saturated (D) imaging also revealed a serpiginous region of T1 hypointense signal and T2 hyperintense signal within S1 *(arrow)*, suggestive of osteoradionecrosis.

SUMMARY

An understanding of the delayed manifestations of Kummel disease should encourage repeat imaging in patients with persistent or recurrent symptoms following trauma, even after a prolonged asymptomatic period. This is especially important in patients with risk factors such as advanced age, osteoporosis, or steroid usage.

SUGGESTED READING

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