29 Vertebral Hemangioma

Mara Kunst

INTRODUCTION

Vertebral hemangiomas (VHs) are benign vascular tumors frequently encountered as incidental findings on computed tomography (CT) or magnetic resonance imaging (MRI). The vast majority are quiescent, with classic imaging features that are reassuring to radiologists and patients, and are called *typical hemangiomas*. VHs that are clinically quiescent but do not demonstrate classic imaging features are frequently referred to as *atypical hemangiomas*. These may produce a diagnostic challenge, requiring additional diagnostic tests and potentially even confirmatory biopsy but incur little risk to the patient. A small percentage of VHs can display active behavior, demonstrating aggressive imaging features that can mimic primary bone malignancies or metastases. These "aggressive hemangiomas" are frequently clinically symptomatic, resulting in pain or neurologic compromise. Treatment options vary by patient and institution, but most commonly include radiotherapy and/or surgery.

EVOLUTION: OVERVIEW

According to a large autopsy and radiography study,[1] VHs are present in 10% to 12% of the adult population, but this is likely an underestimate because MR can now detect an extremely large number of small VHs. Histologically, VHs are composed of capillary or cavernous blood vessels, lined by a single layer of flat endothelial cells set in a loose, edematous stroma. These vessels permeate bone marrow and surround preexisting trabecula. Secondary reactive phenomena, such as fatty or fibrous involution of bone marrow, frequently occur (Fig. 29.1).[1,2] Although the exact mechanism for hemangioma formation and growth has yet to be discovered, studies have cited the importance of local tissue ischemia and estrogen signaling in promoting vasculogenesis.[3]

Typical hemangiomas reflect the histology, demonstrating low attenuation interspersed between vertically oriented bony trabecula on CT, and hyperintense signal on T1- and T2-weighted MR images due to their fatty content (Fig. 29.2). These are considered incidental and inconsequential, requiring no further diagnostic work-up or routine follow-up. Atypical hemangiomas have less fatty and greater vascular content, thereby demonstrating reduced T1 signal.[1,2,4] Although this may produce a diagnostic challenge, CT showing vertically oriented trabecula will frequently aid in establishing lesions as benign. If discovered incidentally and clinically asymptomatic, atypical VHs also require no further work-up. Aggressive VHs comprise a small subset (1%) of all VHs, in which fat becomes replaced by vascular stroma. On imaging, this results in background soft tissue attenuation on CT and hypointense T1 signal on MR. Additional aggressive imaging features may also be present and include involvement of the entire vertebral body, extension into the neural arch, cortical expansion, and an associated soft tissue mass.[1,2,4] Patients with aggressive VHs frequently become symptomatic, with lesions producing pain (55%) or neurologic deficits (45%).[5] Recognizing remnant bony trabeculation on CT will provide the best chance at arriving at the correct preoperative diagnosis (Fig. 29.3).

EVOLUTION: IN GREATER DEPTH

It must be emphasized that VHs without aggressive imaging features may still produce pain and can continue to grow (Fig. 29.4).

Alternatively, VHs that demonstrate aggressive imaging features can exhibit vastly differing rates of growth (Fig. 29.5).

This underscores the need for accurate reporting of individual lesion characteristics and change over time, should patients become symptomatic.

Progression to symptoms can be the result of mechanical compression from tumor growth (producing either bony or soft tissue expansion), intratumoral hemorrhage, or compression fracture due to weakened bone. VHs can also produce local redistribution of blood flow, resulting in a relative cord ischemia (Fig. 29.6).[6,7] It is reasonable to conclude that this ischemia may contribute to the sudden development of symptoms in patients whose VHs are less aggressive appearing and/or stable over time.

One known risk factor for development of neurologic symptoms in quiescent hemangiomas is pregnancy.[6–9] Increased abdominal pressure in the third trimester leads to increased flow in the vertebral venous system. Redistribution of blood flow can limit the tenuous blood supply of the thoracic spinal cord.[6,7] Finally, estrogen also influences endothelial growth and hemangioma enlargement.[9,10]

Treatment options for symptomatic VHs vary according to patient and institution, but most commonly include radiotherapy and/or surgery.[11] Radiotherapy is an effective treatment for VHs

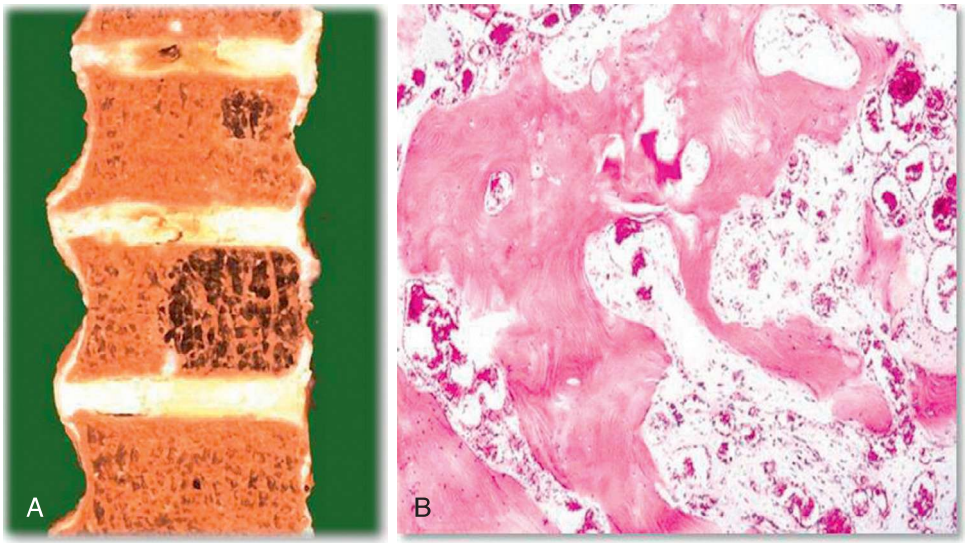

Figure 29.1. (A) Gross photograph of hemangiomas of the vertebral bodies shows two well-demarcated, coarsely trabeculated red lesions, clearly demarcated from the normal cancellous bone. (B) Photomicrograph of hemangioma in which vascular channels of various sizes and shapes can be seen. The thickened bone appears immature, with increased cellularity and irregular architecture (H&E, × 4 obj.). *(Reprinted with permission from Bullough PG. Benign nonmatrix producing bone tumors. In:* Orthopaedic Pathology. *5th ed. St. Louis: Mosby [Elsevier]; 2010:549.)*

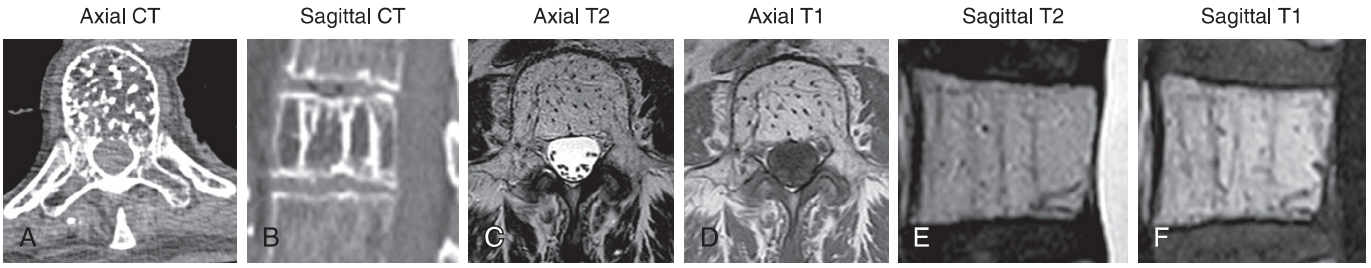

Figure 29.2. "Typical" imaging appearance of a hemangioma. The radiologic diagnosis is straightforward when a nonexpanded vertebral body demonstrates coarsened and thickened vertically oriented internal trabeculation. The vertical orientation of vertebral body coarsened trabeculation results in the pathognomonic "polka dot" or "spotted" pattern on axial CT images (A) and "corduroy" or "jail bar" pattern on sagittal or coronal CT images (B). Note the diagnostic T1 and T2 hyperintense fat signal intensity surrounding the coarsened trabeculations on MRI (C–F).

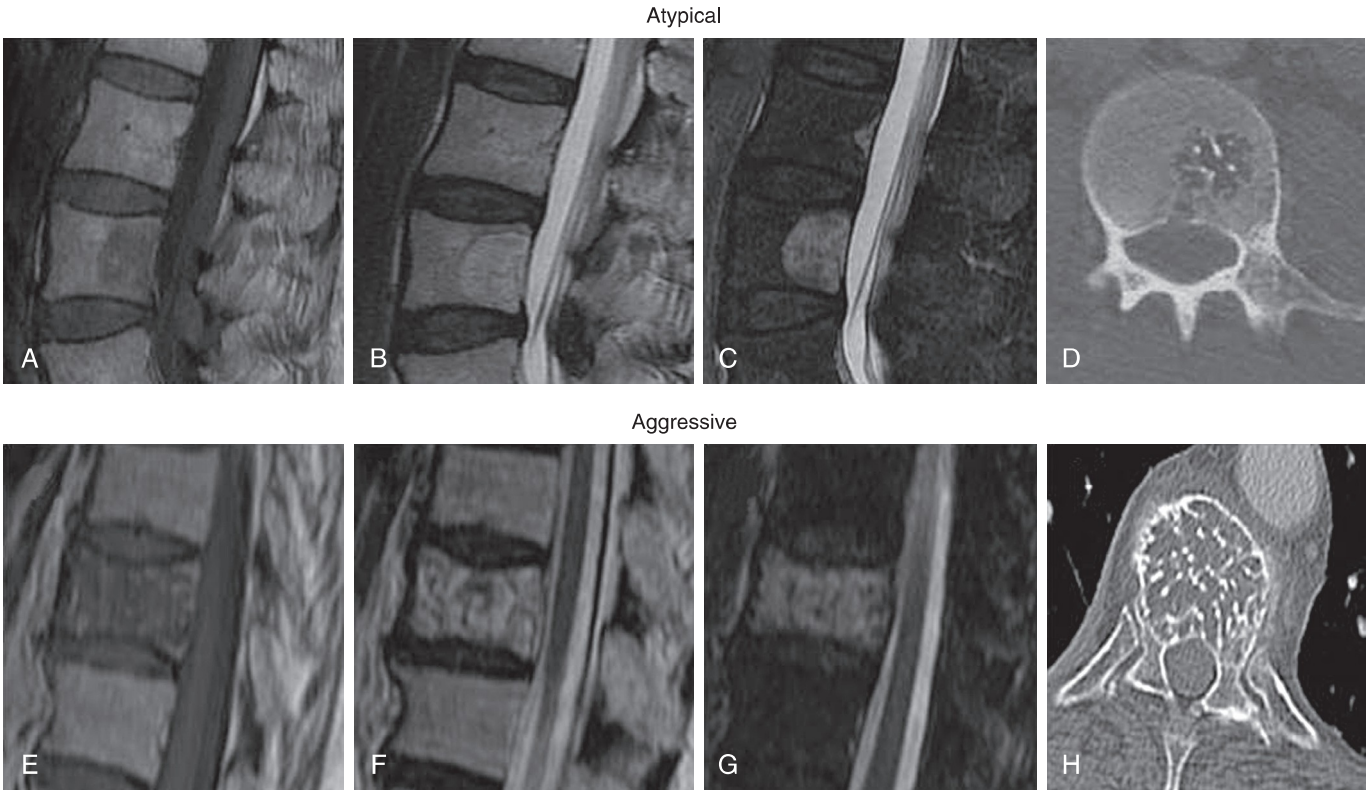

Figure 29.3. Atypical and aggressive vertebral body hemangiomas. (A–D) Atypical hemangiomas have reduced T1 signal (A) due to a relative paucity of intralesional fat and remain hyperintense on T2 (B) and T2 fat-saturated (C) sequences. Thickened vertical trabecula should be considered a key feature for the diagnosis (D). (E–H) Aggressive vertebral body hemangiomas show low T1 (E) and high T2 (F) and fat-saturated (G) signal as further evidence of replacement of marrow fat by vascular stroma. Aggressive vertebral body hemangiomas frequently have an atypical radiologic appearance on any imaging modality. Both MRI and CT should be obtained to look for the classic appearance (H) to help narrow the differential—however, ultimately biopsy may be needed.

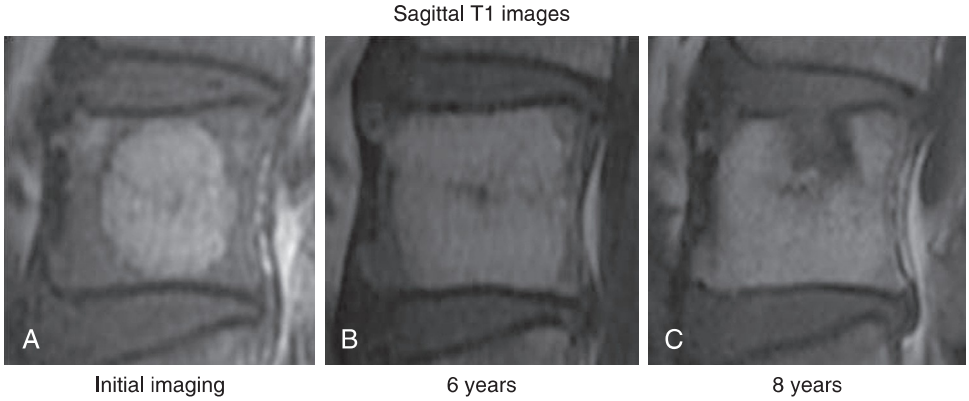

Figure 29.4. Although "typical" hemangiomas are overwhelmingly asymptomatic and quiescent lesions, they can occasionally exhibit slow growth. Sagittal T1 images of a fat containing L4 vertebral body "typical" hemangioma at the time of initial imaging (A), 6 years (B), and 8 years (C) later demonstrate slow, gradual enlargement of the hemangioma. An associated prominent Schmorl node is evident 8 years after initial imaging, presumably secondary to bone density reduction and weakened bone.

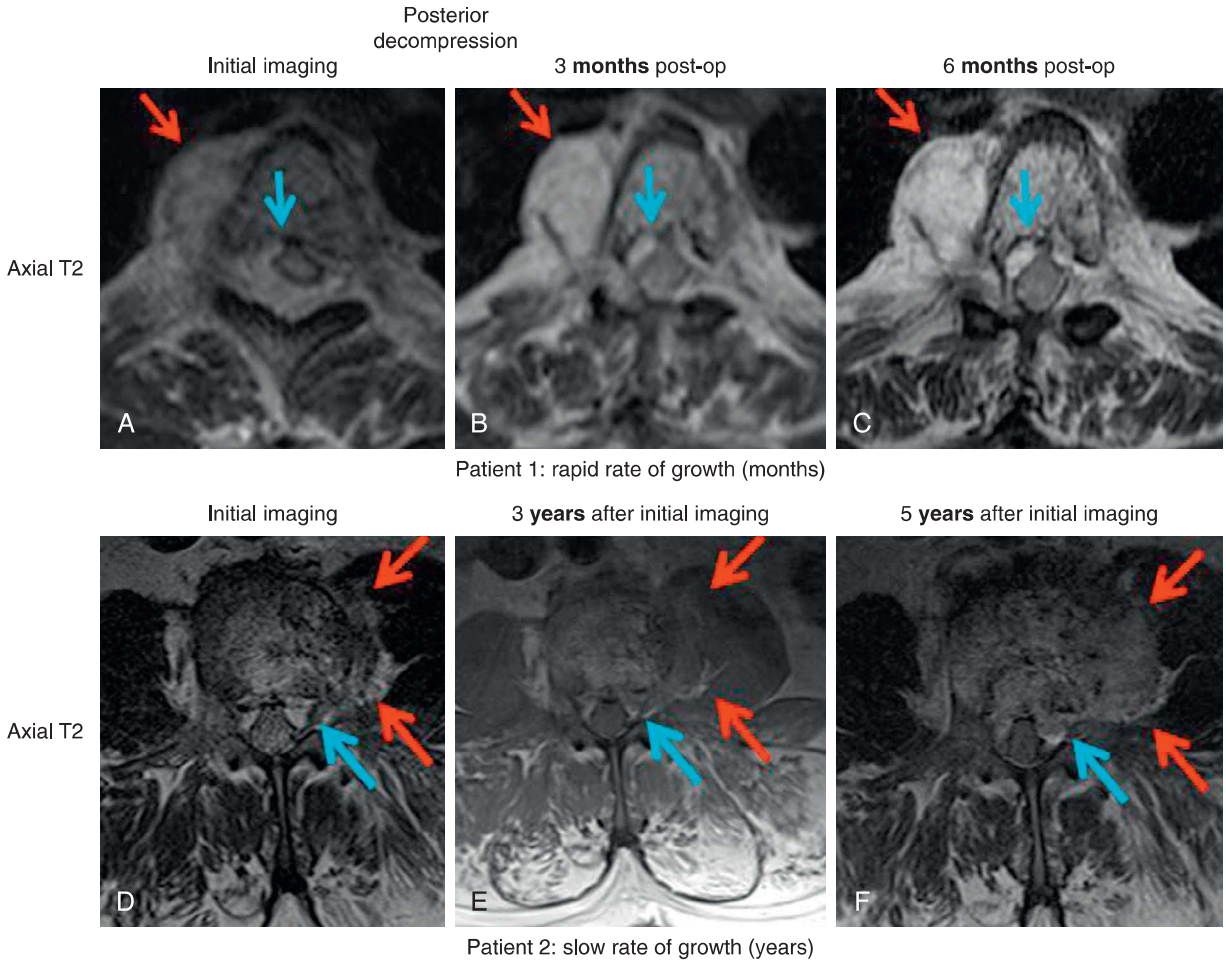

Figure 29.5. Hemangiomas with "aggressive" features may demonstrate a wide spectrum of growth rates. Axial T2 images in patient 1 (A–C) demonstrate a rapid appreciable rate of growth of right paravertebral *(red arrows)* and ventral epidural hemangioma components *(blue arrows)* over the 3-month imaging intervals. Axial T2 images in patient 2 (D–F) demonstrate a much slower rate of growth of left paravertebral *(red arrow)* and ventral epidural components *(blue arrows)* over imaging intervals spanning several years.

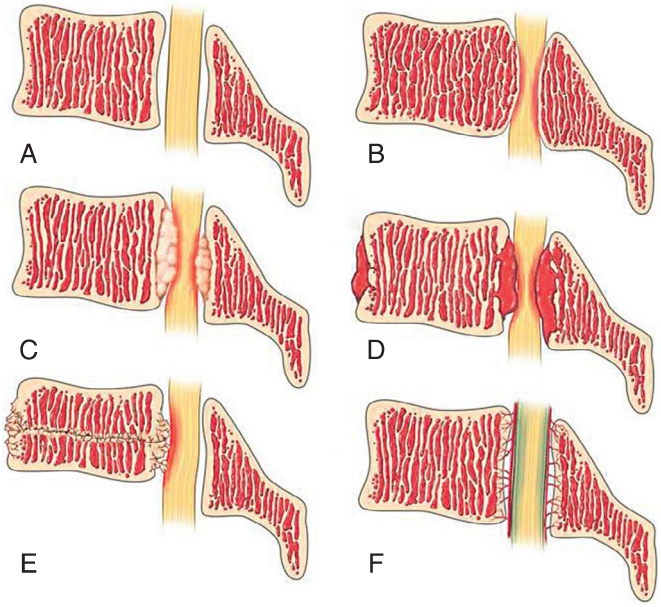

Figure 29.6. Graphic representation of individual factors that can contribute to rapid progression on vertebral hemangiomas, producing pain or neurologic compromise. (A) Vertebral body hemangioma with vertical striations of bony trabecula, (B) osseous expansion producing cord compression, (C) soft tissue expansion producing cord compression, (D) epidural hemorrhage, (E) compression fracture, and (F) redistribution of blood flow producing relative cord ischemia.

that cause pain but without neurologic deficits or spinal canal invasion on imaging. The effective dose is 35-40 Gy in 20 fractions over a 4 week period, a dose at which potential complications such as radionecrosis, radiation-induced myelitis, and secondary malignancy are rare.[12–14] Surgery is reserved for patients with neurologic deficits, or pain and evidence of spinal canal invasion requiring rapid decompression. Additional therapies, including kyphoplasty, embolization, or percutaneous sclerotherapy, may have a secondary therapeutic role in specific cases.[11,14]

DIFFERENTIAL DIAGNOSIS

Aggressive VHs have a heterogeneous imaging appearance, which makes their diagnosis complex. Particularly on MR, their appearance may mimic that of osteolytic metastases, Paget disease, lymphoma, and solitary bone plasmacytoma.[15] Chordoma can also appear similarly but most commonly occurs in the clivus and sacrum, whereas aggressive VHs most commonly occur in the mid thoracic spine.[12] Solitary plasmacytoma is a rare condition that may enter higher on the differential based on clinical grounds. The most commonly cited differential diagnostic considerations are therefore osseous metastases, Paget disease, and lymphoma. CT is the easiest and potentially most effective technique to further narrow the differential.

Although aggressive VHs may still show a remnant organized trabecular pattern, demonstrating a classic "white polka dot" or "corduroy" appearance on CT, metastases and lymphoma tend to replace or destroy bone. Paget disease may also be distinguishable by CT, demonstrating thick cortex, thick trabecula, and wide bone (Fig. 29.7). If CT is not helpful, FDG-PET may be used because

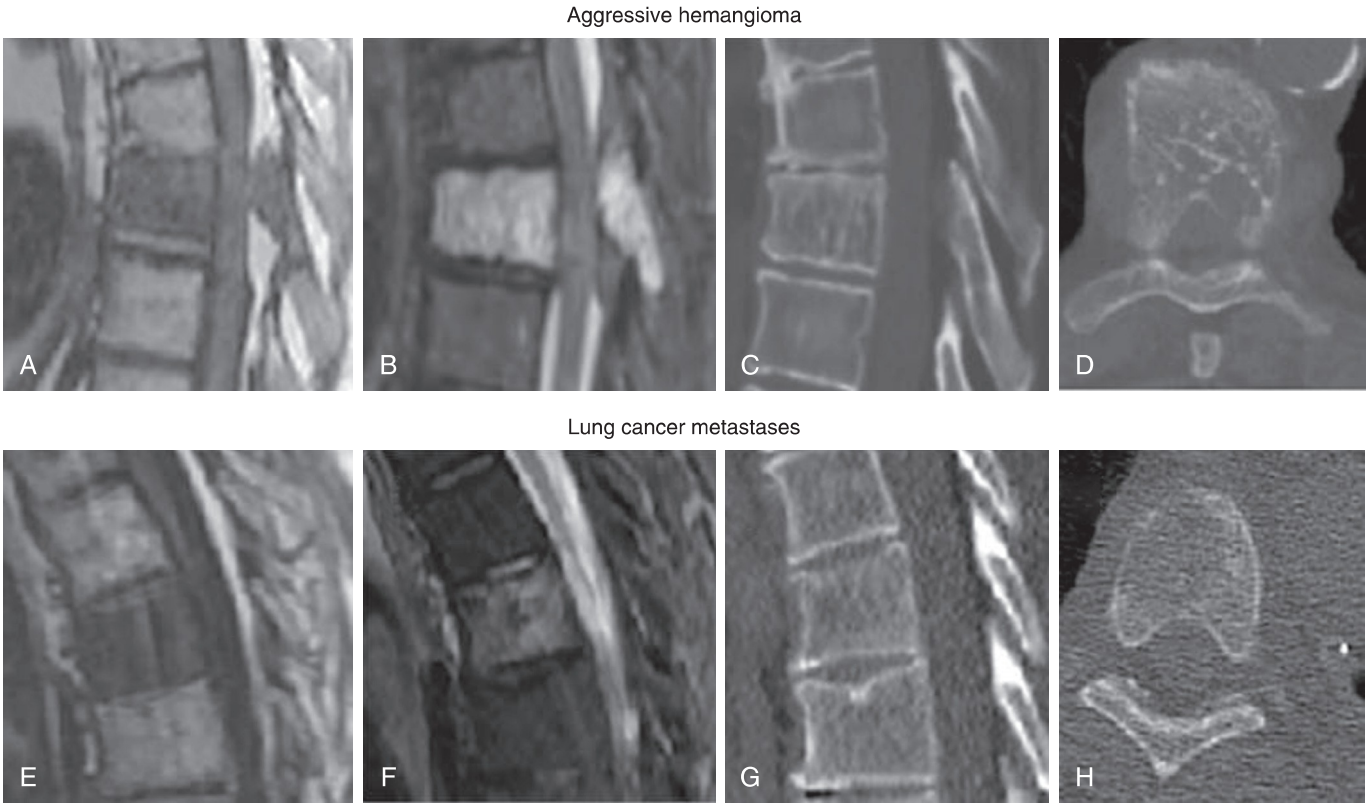

Figure 29.7. Differential diagnosis: images A through D are of an aggressive vertebral body hemangioma. The lesion is hypointense on T1 (A) and hyperintense on T2 with fat saturation (B) and shows classic vertical striations on sagittal CT (C) and stippled appearance on axial CT (D). Images E through H show images of a 76-year-old male with lytic metastases from lung cancer. This lesion is hypointense on T1 (E) and hyperintense on T2 with fat saturation (F) and has a permeative appearance with cortical destruction on sagittal (G) and axial (H) CT imaging.

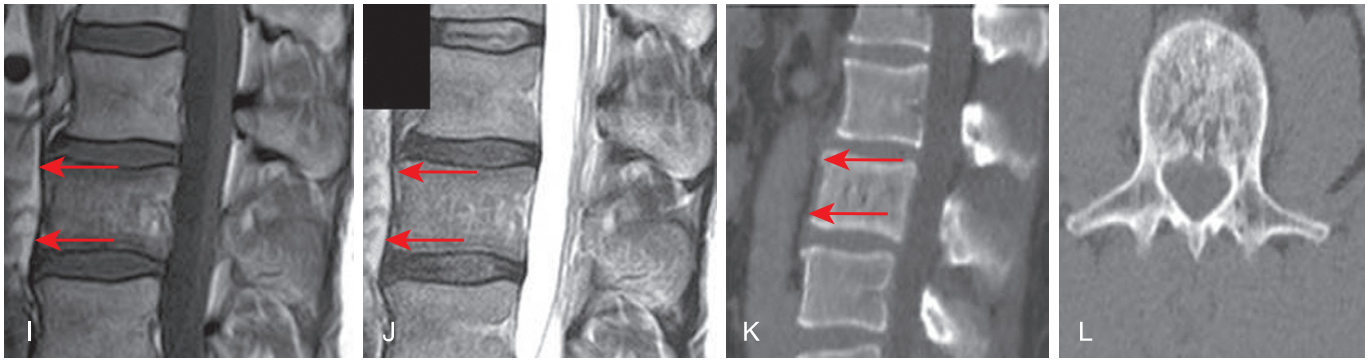

Figure 29.7., cont'd Images I through L are from a 67-year-old male with Paget disease of bone. The affected vertebral body is heterogeneously hypointense on T1 (I) and hyperintense on T2 (J). Sagittal MRI (I and J) and sagittal and axial CT (K and L) images demonstrate the classic triad of Paget disease: thickened cortex *(arrows)*, thickened trabecula, and wide bone (notice the vertebral body is slightly larger than those adjacent).

metastases and lymphoma are FDG avid. Paget disease is typically not FDG avid but will demonstrate increased radiotracer uptake on bone scan.[16]

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