patients with midtarsal injuries, but none had isolated cuboid dislocations.

Five reports can be found in the literature describing cuboid dislocations, all with plantar medial displacements of the cuboid. In all cases, the force has been directed medially and plantarly on the outside of the foot. This mechanism is similar in the case described.

If the hindfoot is in valgus, the axis of the talonavicular and calcaneocuboid joints will be parallel, allowing some motion. A plantar-medial force on the cuboid will allow the cuboid to be dislocated under the calcaneus, while slight rotatory subluxation will take place in the talonavicular joint. At the time of impact, slight displacement will also occur in the subtalar joint, giving rise to small chip fractures in that joint, but without dislocation.

Cuboid dislocations are rare and can easily be overlooked on a radiograph. Three of the five cases in the literature had delayed diagnoses. When recognized, immediate reduction should be performed. Closed reduction under general anesthesia can be attempted, hanging the foot with the toes in a finger trap and applying force in the opposite direction as the trauma. This was not successful in our case, but was in two of five cases in the literature. Open reduction is usually necessary, and pin fixation of the joint for a short period of time can be performed to secure the reduction. Routine exploration of other subtalar joints is not recommended.

REFERENCES

EDITORIAL COMMENT

While this is an unusual injury, it is important for it to be recognized. One should have a high index of suspicion when assessing a patient with a history of major trauma and significant foot swelling. As emphasized by the author, there is frequently a delay in diagnosis. While this particular patient appeared to have a fair result with 1-year follow up, this is not the case in a large proportion of patients with major midtarsal injuries. The long-term results of these injuries is not known, as the literature is replete with case reports and other clinical studies reporting the outcome of a variety of injury patterns.

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DISCOGENIC VERTEBRAL SCLEROSIS:
A POTENTIAL MIMIC OF DISC SPACE INFECTION OR METASTATIC DISEASE

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Guo Wang, MD
Richard Whitehill, MD

Sclerosis at the vertebral endplate is a commonly encountered plain film radiographic abnormality, caused by primary or secondary tumors, infection, aseptic necrosis, inflammatory arthritis, trauma, or degenerative disease. The most frequently encountered lesion is probably degenerative, appropriately called discogenic vertebral sclerosis (DVS). The exact cause for the sclerosis is unknown, but this entity has received much attention in the radiologic literature and has been referred to by a variety of names: non-neoplastic sclerosis, pseudoinfection of the intervertebral disc and adjacent vertebrae, discogenic vertebral sclerosis, atypical degenerative lesion, hemispherical spondylosclerosis, idiopathic segmental or vertebral sclerosis, vertebreal rim lesions, or degenerative spondyloarthropathy. However, the disease has been reported infrequently in the orthopedic literature.

Patients with DVS may show a variety of radiographic changes on plain radiographs, ranging from minimal sclerosis around a Schmorl's node, to intense vertebral body sclerosis resembling the "ivory" vertebra of metastatic disease. The sclerosis may also involve primarily two adjacent vertebral endplates and be mistaken for infection.

We report three patients with DVS on plain films and describe other radiographic abnormalities which may be encountered in this entity. The appropriate radiographic work-up in patients suspected of having DVS is also included. Orthopedists and radiologists should be familiar with this entity because of its mimesis of more sinister pathology.
**CASE REPORTS**

Case 1. A 53-year-old woman was evaluated at the gynecology clinic for cervical dysplasia. Physical examination was unremarkable. She had sustained spine trauma 20 years previously and at the time of examination had vague nonradiating low back pain. A lumbar spine series showed an area of sclerosis in the anteroinferior border of L4, with osteophytes at the L4-L5 interspace (Fig 1). The endplates were preserved, and no further work-up was performed. Follow up at 5 years showed little change in the sclerosis, and the patient’s pain
Fig 4: Posterior view of radionuclide bone scan in case 2 shows moderate increased nuclide localization at L1-L2 (arrow).

Fig 5: Single lateral film from tomography shows sclerosis of the vertebral bodies with preservation of the endplates.

Fig 6A: Coned-down lateral view of case 3 shows intense sclerosis of the L4 and L5 vertebral bodies. Lucency in central portion of vertebral endplate probably represents a Schmorl's node.

Fig 6B: AP radiograph of case 3 shows intense sclerosis of L4 and L5, but no significant endplate irregularity.

has been well-controlled with antiinflammatory, nonsteroidal analgesics.

Case 2. A 48-year-old woman presented with low back pain and radiation to the anterior aspect of the left thigh of 6 months duration. There was no apparent history of trauma, and physical examination and laboratory studies were normal. A lumbosacral spine series done 2 years before her referral to our hospital demonstrated intense endplate sclerosis at the L1-L2 interspace with osteophytes (Figs 2A-B). These changes showed progression at the time of her presentation, and a radionuclide bone scan showed mildly increased uptake at the L1-L2 level (Figs 3-4). The orthopedists consulted radiology to perform a percutaneous biopsy to exclude infection. After review of the films, the musculoskeletal radiologist asked that linear tomography be performed before biopsy to assess the status of the vertebral...
endplates. Tomography demonstrated preservation of both endplates, and no biopsy was obtained (Fig 5). The patient is well 1 year later without evidence of infection.

Case 3. A 36-year-old woman complained of low back pain which had been present for 2½ years. There was no radicular component to the pain. Physical examination was normal, except for diffuse lumbar paravertebral spine pain on deep pressure. Her laboratory evaluation included a normal complete blood cell count with differential, calcium, phosphate, alkaline phosphatase, uric acid, and erythrocyte sedimentation rates. Serum protein electrophoresis was normal. Plain AP and lateral radiographs demonstrated sclerotic lesions of L4 and L5 (Fig 6), and a radionuclide bone scan showed mild increased localization at this level (Fig 7). A lumbar myelogram, followed by a computed tomography (CT) scan, showed only a mild disc bulge at the L4-L5 interspace (Fig 8). An intravenous pyelogram, mammograms, and a gallium scan were all normal.

The patient underwent a closed Craig needle biopsy under general anesthesia. Bone from the L4 and L5 vertebral bodies and disc material from the L4-L5 interspace were obtained. The biopsy specimens were sterile, and no bacteria were seen on gram stain. The final pathological diagnosis was necrotic bone with new bone formation, pathogenesis unknown.

Postoperatively, the patient experienced back and radiating pain in the distribution of her left third lumbar nerve root. The radicular pain slowly resolved over a 6-week period, although she continues to have low back pain as before.

**DISCUSSION**

The first report of DVS was by Ackermann and Schwarz in 1958. They presented three patients with primary malignancies who developed intense segmental vertebral sclerosis. Biopsy or autopsy in each case showed no tumor, and they called the lesion “non-neoplastic sclerosis.” In 1966, Lowman and Robinson reported three examples of vertebral endplate sclerosis and irregularity, thought to be infection, at laminectomy sites. Biopsy showed no inflammation, and aseptic necrosis was considered a possible cause for these findings. Two years later, Williams et al. reported similar findings in patients who had not had surgery, naming the entity, “pseudoinfection of the intervertebral disc and adjacent vertebrae.” All of these early reports were probably DVS, and later reports have used a variety of other designations for the disease.

Battikh et al. described three radiographic patterns of vertebral sclerosis in DVS: dome-shaped sclerosis arising from the vertebral endplates, band-like sclerosis across the vertebral
endplate, and diffuse vertebral sclerosis. Katz et al. expanded this concept into six classes of vertebral endplate sclerosis.

Patterns A-C represent varying degrees of severity and distribution of endplate sclerosis, all of which are most commonly seen in DVS. Pattern D is more extensive sclerosis and, when the endplates are smooth, is most likely due to DVS. This pattern is exhibited in case 2. If the endplates are irregular and eroded, infection is a more likely cause for this pattern of erosion. Pattern E, diffuse sclerosis involving over 80% of a vertebral body in both endplates, is exhibited in case 3. These changes are rarely caused by DVS, but the presence of a Schmorl’s node, as seen in our patient, suggests DVS as the cause of the sclerosis. Pattern F is any lesion which cannot be placed into any of the other categories.

The majority of patients with DVS are middle-aged to elderly women who present with vague low back pain and have no predisposing factors for spondylitis or metastatic disease. Alternatively, intervertebral sclerosis is noted as an incidental finding on lumbar spine films obtained for other reasons. Any of the lower thoracic or lumbar vertebrae may be involved, but the most common location is the L4-L5 level. The diagnosis of DVS is first suggested by the plain films. Case 1 represents the common findings of helmet-shaped sclerosis centered at the anterior and inferior aspects of the vertebrae associated with intervertebral disc space narrowing at the affected level. More extensive sclerosis, often accompanied by a Schmorl’s node, as seen in case 2 and case 3, is less common. When more pronounced changes are encountered, however, alternative diagnoses may be considered.

The major alternative diagnoses for DVS with extensive sclerosis are infectious spondylitis and metastatic neoplasm (Table 1). Patients with DVS, however, rarely have predisposing factors for either of these two diseases. Furthermore, there are important differentiating radiologic features on plain films. In DVS, the vertebral body height and width are usually preserved, whereas in infection, the vertebral body is narrowed and shortened secondary to erosive changes. Often, metastatic disease also shows some loss of height. Linear tomography may be a helpful next step and should show normal or only slightly irregular endplates in DVS, in contrast to the deeper endplate erosions and loss of vertebral height seen in spondylitis. This feature was helpful in case 2.

Patients with DVS may also have linear gas collections within the substance of the in-

Table 1

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Plain Films/Tomography

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Computed Tomography

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Magnetic Resonance Imaging

Fig 9A: Coronal T1-weighted MRI image of 38-year-old woman with back pain shows intervertebral joint space narrowing and decreased signal intensity at anterior borders of L2 and L3 (arrowhead).

Fig 9B: T2-weighted image shows increased signal intensity in same areas. This is an example of Type I changes.
Fig 10A: Lateral lumbar spine of 37-year-old woman with back pain shows intervertebral disc space narrowing, and sclerosis and osteophyte formation at L4-L5 interspace (arrow).

Fig 10B: Coronal T1-weighted MRI image shows slight increased signal intensity at the anterior endplates of L4 and L5 (arrowhead).

Fig 10C: Coronal T2-weighted image in same patient shows slight increased signal intensity in same areas compatible with Type II changes (arrowhead).

Intervertebral disc space representing nitrogen gas formation secondary to a degenerative process, an extremely rare feature in infectious spondylitis or neoplasm. Computed tomography may also be helpful in equivocal cases. In infection, CT should show a destructive process centered on the disc space with extensive vertebral erosions, marked loss of the intervertebral space, and a prevertebral soft tissue mass. In metastatic neoplasm, CT may show more extensive posterior element involvement and a smaller prevertebral soft tissue component. In contrast, CT findings of DVS are mild intervertebral disc bulging, joint space narrowing, subchondral cyst formation, and absent soft tissue mass at the affected level, as seen in case 3.

Radionuclide bone scanning (RBS) is another test which may be considered in problem cases. However, because new bone formation may occur in DVS, metastatic disease, or spondylitis, RBS will show increased accumulation in each disorder. The intensity of uptake of the radionuclide should be more intense in infection than in DVS, but this feature will not reliably distinguish between the two entities. A finding of multiple axial and appendicular lesions with intense uptake, however, should suggest metastatic neoplasm as the possible cause of the sclerosis.

Finally, magnetic resonance imaging (MRI) has shown promise in distinguishing DVS from other discovertebral lesions. Modic et al characterized the MRI features of the vertebral body and intervertebral disc in degenerative disc disease imaged by high field strength magnets. In the vertebral body, Type I changes, seen in approximately 4% of patients undergoing MRI for suspected lumbar disc disease, show decreased signal on T1-weighted and increased signal on T2-weighted images (Fig 9). Histologically, discs with Type I characteristics show disruption and fissuring of the endplate with vascularized fibrous tissue, which probably causes a prolongation of T1 and T2 relaxation times. Type II changes, seen in approximately 16% of all MRI cases and in 30% of patients treated with chymopapain (a model for acute disc degradation), show identical signal on T1-weighted and T2-weighted images or slightly increased signal on T2-weighting (Fig 10). Histology of patients with Type II MRI characteristics shows endplate disruption with red to yellow marrow conversion, which shortens the T1 relaxation time. However, Type I and Type II changes have not been correlated with clinical symptoms and do not necessarily correspond with sclerosis on the plain films.

Type III changes, however, appear to correlate with the sclerosis on the plain film seen in DVS. These MRI features are decreased signal intensity on both T1 and T2-weighted images. This decreased signal is probably due to a combination of decreased hematopoietic elements and an increase in immobile protons in the region of intense sclerosis seen on the plain films (Fig 11). In contrast, however, Sobel et al reported three patients with DVS showing increased signal intensity, thought to be edema, hemorrhage, or absent hematopoiesis in the area.
Fig 11A: Lateral lumbar spine of 56-year-old woman with back pain shows intense sclerosis and osteophyte formation at L5-S1. Note disc bulge (arrow).

Fig 11B: Coronal T1-weighted MRI image shows decreased signal intensity in the areas of sclerosis seen on the plain films (arrow).

Fig 11C: Coronal T2-weighted image in same patient shows decreased signal intensity in areas of sclerosis on plain film characteristic of Type III changes (arrow).

Fig 12A: Coronal T1-weighted lumbar spine MRI in 62-year-old man with prostate carcinoma shows decreased signal intensity in multiple areas of metastatic involvement.

Fig 12B: T2-weighted image in same patient shows decreased signal in same areas typical of sclerotic metastatic neoplasm.

of sclerosis, on T2-weighted images. This discrepancy may be explained by the fact that Sobel's data were obtained on low field strength magnets.

The normal intervertebral disc is characterized on MRI by a slightly decreased signal intensity of the central nucleus pulposus relative to the peripheral anulus fibrosus on T1-weighted images. On T2-weighted images, the signals are reversed, with the inner nucleus pulposus showing high signal intensity while the anulus shows lower signal intensity. With disc degeneration and subsequent fibrosis, there is an overall decreased signal throughout the entire intervertebral disc and no extraosseous findings on either T1- or T2-weighted sequences.24,25

In infectious spondylitis, the disc and body show different MRI characteristics.26 A para-vertebral soft tissue mass, distorted disc with an abnormal configuration, and no differentiation of the disc from the vertebral endplate are seen in infection. Typically, there is low signal intensity within the disc and adjacent vertebral
In some patients, even after extensive diagnostic evaluation, the differentiation between DVS, infection, and neoplasm may not be clear cut. In these cases, depending on the clinical situation, percutaneous biopsy or follow up may be appropriate next steps. If follow up is chosen, the natural history of the diseases may aid in a definite diagnosis. DVS progresses slowly with gradual increase in the sclerosis, as shown in case 2. Patients with infectious spondylitis should show increasing symptoms, bone destruction and erosion, and increasing soft tissue mass. Patients with sclerotic metastatic disease should show increasing sclerosis, multifocal involvement, and constitutional symptoms. If symptoms or signs progress, prompt biopsy is appropriate (Table 2).

In summary, DVS is a relatively common disease with a wide range of radiographic manifestations which may be mistaken for infectious spondylitis or metastatic disease. However, there are clinical and radiographic clues which are helpful in distinguishing between these entities. Orthopedists and radiologists should be aware of the salient features of DVS, and add it to the list of differential considerations for vertebral endplate or vertebral body sclerosis in patients with low back pain.

REFERENCES

4. Williams JL, Moller GA, O'Rourke TL. Pseudoinfections of the intervertebral disk and adjacent vertebrae. AJR. 1968; 103:611-615.
ORTHOPEDICS: It has been suggested by some that the low back pain associated with discogenic vertebral sclerosis may be benefited by surgical disc excision and endplate curettage. You make no mention of this in the paper. What is your opinion?

Pope et al: We make no mention of surgical disc excision and endplate curettage in this article primarily because we are most interested in the preoperative diagnosis of discogenic vertebral sclerosis not being confused with other pathology. It would be logical to think that surgical intervention as above would diminish some of the symptoms in this disease.

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HEMANGIOMA OF THE TIBIA

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CASE REPORT

A 44-year-old man presented with posteromedial left knee pain with activity. He denied trauma, past infection, effusion, night pain, or past history of malignancy. Physical examination was normal without any evidence of cutaneous hemangiomas or warmth in the region. Radiographs were taken (Fig 1), revealing cortical striations and sclerosis in the metaphysis of the proximal left tibia without any cortical expansion. A TC99m radionuclide scan (Fig 2) showed focal uptake in this region alone.

A MRI scan (Fig 3) was also performed. There was a cystic region anteriorly with a sclerotic portion posteromedially, and irregular calcification. No cortical expansion was evident. A chest radiograph was normal. Complete blood count, blood chemistry, and erythrocyte sedimentation rates were all within normal limits.

Open biopsy of the lesion was performed. Fibro-fatty brown tissue was removed through an oval cortical window. Blood-filled regions were present with minimal active bleeding encountered. A frozen section was done which was consistent with hemangioma. Further curettage was performed, and the biopsy site was filled with polymethylmethacrylate. Histologically, the specimen consisted of somewhat thickened bony trabeculae intermixed with fibrosis and benign vascular endothelium. No hematopoietic elements were identified. A more solid portion consisted of fibrous tissue with multiple blood vessels oriented transversely and longitudinally. Final pathology was consistent with cavernous hemangioma of bone (Fig 4).

At 12 months postoperative, the patient was asymptomatic with normal functional activities.

DISCUSSION

Skeletal hemangiomas represent 1% of all lesions of bone.1-5 Hemangiomas of bone commonly occur in the fifth decade and predominantly in women. Bucy and Capp were the first authors to report on hemangiomas in the American literature,6 describing eight cases of hemangioma of bone, with only one involving a long bone (ulna). They also reviewed the literature and reported on four lesions affecting long bones with only one involving the tibia. Unni et al7 reported the Mayo Clinic’s series.