What Is Your Diagnosis?

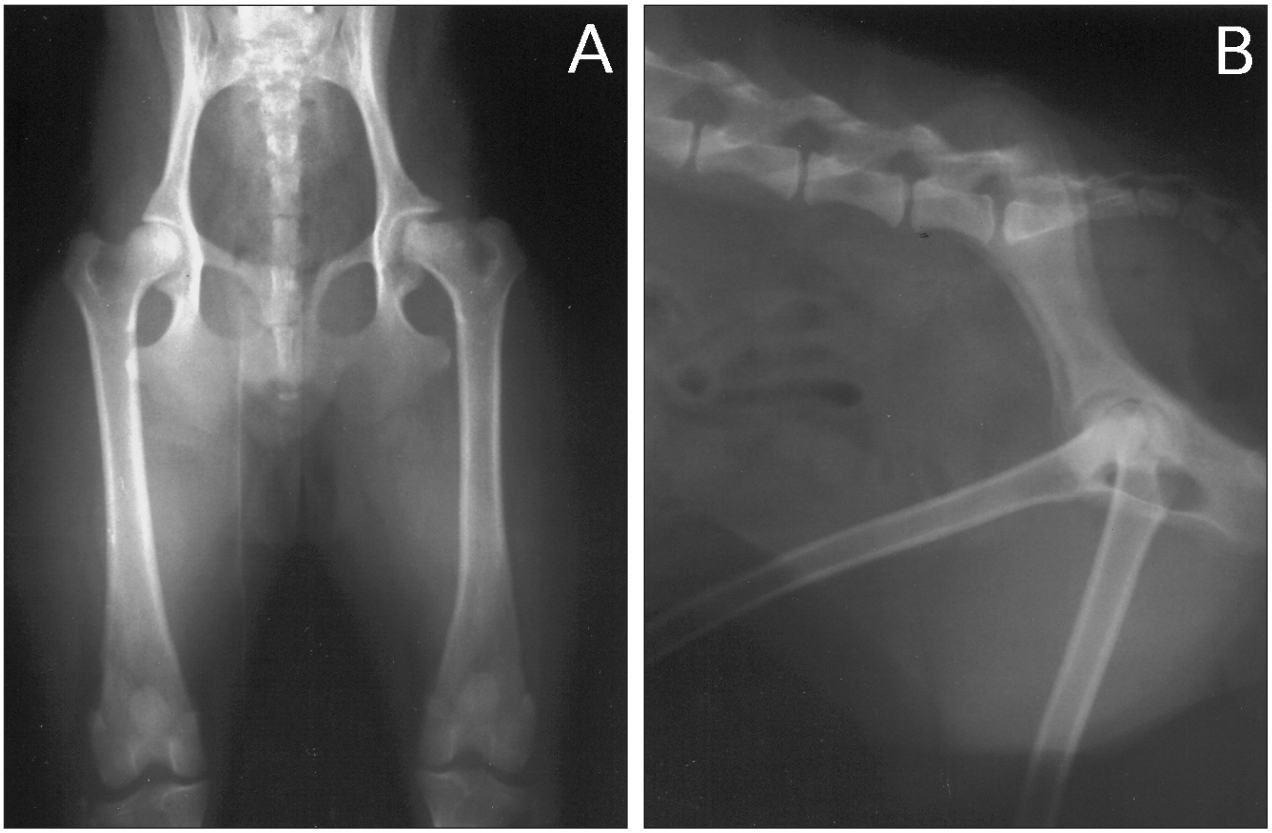

Figure 1—Extended ventrodorsal (A) and lateral (B) radiographic views of the pelvis of a 13-month-old Miniature Pinscher with a 4-month history of progressive lameness of the left hind limb. The dog had been treated surgically 3 months previously to correct left medial patellar luxation.

History

A 13-month-old spayed female Miniature Pinscher was evaluated because of a left hind limb lameness that had progressed in severity to a non–weight-bearing lameness over the previous 4 months. The affected limb had been shut in a door when the dog was 12 weeks old. However, the dog recovered fully from that injury, and mild intermittent left hind limb lameness did not develop until several months later. Three months prior to the present evaluation, left trochleoplasty and tibial tuberosity transposition were performed to repair a grade II medial patellar luxation, but the lameness continued to worsen after surgery. On physical examination, the left hind limb musculature was atrophied, and abduction of the left hip joint elicited signs of pain. A grade II medial patellar luxation was detected in the right hind limb. Manipulation of the left stifle joint and patella did not elicit signs of pain. Radiographs of the pelvis were obtained (**Fig 1**).

Determine whether additional imaging studies are required, or make your diagnosis from Figure 1—then turn the page ⏵

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Diagnosis

Radiographic diagnosis—Avascular necrosis of the left femoral head with secondary osteoarthritis of the hip joint (**Fig 2**).

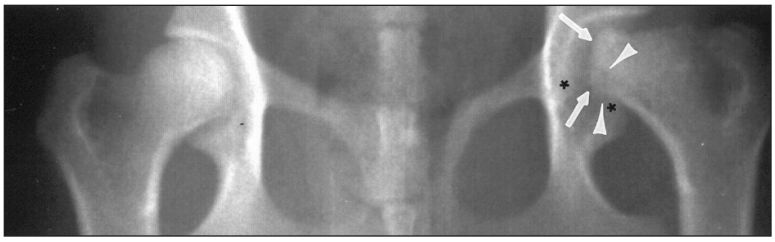

Figure 2—Panoramic enlargement of the hip joints on the ventrodorsal view depicted in Figure 1. The left femoral head is flattened (arrows), and subchondral erosions and sclerosis are evident (arrowheads). Osteophytes are apparent on the left acetabulum (asterisks), and the left femoral neck is thickened and the joint space is widened, compared with the right side.

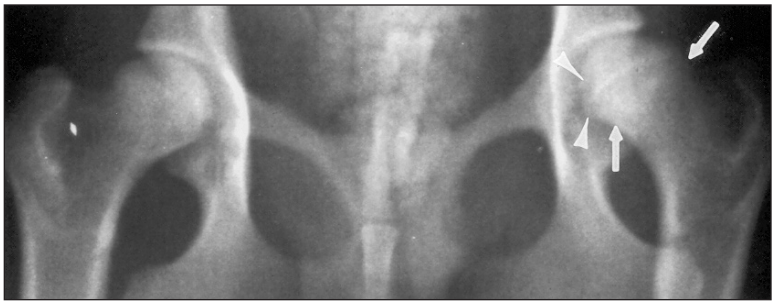

Figure 3—Panoramic enlargement of the hip joints on an extended ventrodorsal radiographic view of the pelvis of the dog described in Figure 1. This radiograph was obtained when the dog was 10 months old, 1 month after initial onset of mild intermittent left hind limb lameness. Subtle lesions are apparent, including mild sclerosis and thickening of the left femoral neck (arrows) and radiolucencies of the left femoral head (arrowheads). These lesions are suggestive of early avascular necrosis of the femoral head.

Comments

The left thigh muscles were mildly atrophied, and the left femoral head was flattened. Subchondral erosions and sclerosis of the left femoral head were apparent, and osteophytes were evident on the left acetabulum. In addition, the left femoral neck was thickened and the joint space was widened, compared with the right side. These radiographic changes are classic findings of avascular necrosis of the femoral head, or Legg-Calvé-Perthes disease, particularly considering this dog's age and breed. Avascular necrosis of the femoral head is often diagnosed in toy, terrier, and other small breeds of dogs weighing less than 12 kg (26.4 lb) and primarily develops unilaterally; bilateral involvement is reported in only 12 to 16.5% of cases.[1,2] Age of onset is between 3 and 13 months, with most affected dogs identified between 5 and 8 months of age.[1,2] The condition is caused by loss of blood supply to the femoral head and neck, resulting in necrosis and collapse of subchondral bone and collapse and cracking of associated cartilage.[1,2] Subchondral bone remodeling follows.[1,2] Initially, radiographic changes of avascular necrosis of the femoral head include flattening of the dorsal aspect of the femoral head with lucencies in the subchondral bone.[1,2] As the disease progresses, the femoral head becomes deformed, and signs of degenerative joint disease may become apparent.[3]

The etiopathogenesis of avascular necrosis of the femoral head is not completely understood.[1,3] It is thought that small-breed dogs that spend much of their time jumping excitedly on their hind limbs may, over time, cause enough vascular damage to collapse the epiphyseal vessels,[1,3] resulting in localized ischemia of the femoral head.[1,3] An autosomal recessive gene has also been proposed as the cause of this disease, so owners should be advised to spay or castrate affected dogs.[3]

Definitive diagnosis was made difficult in this case by several potentially confounding factors. Important differential diagnoses for avascular necrosis of the femoral head include medial patellar luxation and physeal trauma.[3] Although the left hind limb of the dog of this report was injured at 12 weeks of age, progressive lameness did not develop until several months later. Even so, the initial trauma cannot be ruled out as a predisposing cause of avascular necrosis of the left femoral head. Also, left medial patellar luxation was diagnosed when the dog was 10 months old, and this abnormality was initially considered the cause of lameness. However, because the medial patellar luxations were bilateral and of similar severity, they were not likely to be the cause of a unilateral lameness. Additionally, we did not detect left patellar subluxation on the original radiographs of the stifle joint obtained when the dog was 10 months old just prior to left trochleoplasty and tibial tuberosity transposition or on follow-up radiographs obtained 3 months later. In fact, early subtle lesions suggestive of avascular necrosis of the femoral head were apparent on reexamination of pelvic radiographs obtained at 10 months of age (**Fig 3**).

Femoral head and neck ostectomy (FHO) is the definitive treatment of avascular necrosis of the femoral head.[1] Virtually 100% of dogs treated by FHO resume normal, pain-free activity.[2] However, in this case, the owners declined surgery and elected to treat the dog conservatively. Conservative treatment consists of 4 to 6 months of cage rest and administration of anti-inflammatory agents, but progression of degenerative joint disease and continued lameness is common.[1] After 6 months of conservative treatment, the dog described in this report was still lame. Radiography revealed that lesions had increased in severity, and the dog was readmitted for FHO. It recovered well after surgery and was able to resume normal activity.

References

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3. Hulse DA, Johnson AL. Management of joint disease. In: Fossum TW, ed. *Small animal surgery*. St Louis: The CV Mosby Co, 1997;956–957.