History

An 18-week-old neutered female Mastiff with a 2-week history of acute left hind limb lameness was evaluated. The puppy injured the left stifle joint when it collided with a pile of bricks during an attempt to jump over them. The puppy had grade 3 (on a scale of 1 to 5) lameness in the left hind limb and severe stifle joint effusion was evident. Manipulation of the left stifle joint revealed severe craniocaudal instability and signs of pain on extension. With the patient conscious, it was difficult to ascertain whether a cranial or caudal drawer sign was present. Tibial thrust was not present, and stifle joint instability was not evident in the mediolateral plane. No additional abnormal examination findings were evident. On the basis of physical examination findings, initial differential diagnoses included traumatic cranial cruciate ligament rupture or caudal cruciate ligament rupture, tibial tuberosity avulsion, Salter-Harris type fracture, or injury to other soft tissue structures of the stifle joint, such as the menisci. Radiographs of the left stifle joint were obtained (Figure 1).

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

Figure 1—Lateral (A) and caudocranial (B) radiographic views of the left stifle joint of an 18-week-old neutered female Mastiff evaluated because of traumatic left hind limb lameness of 2 weeks’ duration.
Radiographic analysis revealed severe intracapsular soft tissue swelling consistent with effusion in the left stifle joint and normal positioning of the tibial tuberosity growth plate and patella (Figure 2). Remaining differential diagnoses included cranial cruciate ligament rupture, caudal cruciate ligament rupture, and meniscal injury.

T1-weighted fat-saturated proton density and heavily T2-weighted spoiled gradient-echo (with multiple echoes) MRI sequences of the left stifle joint were obtained with a 1.5-T MRI unit and extremity coil (Figure 3). Severe effusion was evident in the left stifle joint. The cranial cruciate ligament had a normal hypointense signal, was of normal size, and appeared intact. The caudal cruciate ligament also produced a normal hypointense signal at its attachment to the popliteal notch of the tibia. However, the caudal cruciate ligament had a hyperintense T2 signal and was enlarged from its origin at the femoral intercondylar fossa to the level of the caudal tibial condyle. The medial femoral condyle also had a focal area of increased signal intensity in T2-weighted images, suggesting bone edema or hemorrhage in this region. On the basis of the MRI findings, a caudal cruciate ligament tear was the primary differential diagnosis. It was not determined whether a midbody ligament tear or avulsion fracture of the caudal cruciate ligament was present.

Computed tomography was performed to further evaluate the medial intercondylar fossa. An 8-slice CT scanner was used to obtain images of the left stifle joint, which were reformatted in sagittal and dorsal planes. Computed tomography revealed fragmentation of the medial femoral condyle, confirming a final diagnosis of caudal cruciate avulsion fracture at its origin (Figure 3). The stifle joint was again examined with the patient under anesthesia, and a caudal drawer sign was elicited. A cranial drawer sign and tibial thrust were not present.

Treatment and Outcome

Given that the avulsion was minimally displaced, the patient was treated conservatively with 6 weeks of strict rest and judicious use of the anti-inflammatory drug carprofen (2 mg/kg [0.9 mg/lb], PO, q 12 h). Although surgical options for stabilizing caudal cruciate avulsion fractures are available, it was decided that conservative management in a skeletally immature patient was best. In human medicine, pediatric and adolescent patients with posterior cruciate ligament tears and nondisplaced avulsion fractures are often treated conservatively and have good clinical outcomes. The dog regained excellent use of the affected limb with gradual reintroduction of exercise after the 6 weeks of strict rest.
Isolated caudal cruciate ligament injury is rare and is often not considered in cases of stifle joint instability. In one study, 7 of 14 dogs with caudal cruciate rupture were misdiagnosed as having cranial cruciate ligament rupture, and in 9 of the 14 affected dogs, caudal cruciate rupture was not listed as a differential diagnosis. The diagnosis of caudal cruciate ligament rupture was confirmed in 12 of these dogs only after surgical exploration of the stifle joint.

The use of various diagnostic imaging modalities in the dog of the present report was fundamental to achieving the final diagnosis without surgical exploration. Radiography was useful in eliminating osseous damage such as Salter-Harris type fractures or long bone fractures. It also confirmed stifle joint effusion, narrowing the region of injury to the joint space. Magnetic resonance imaging was useful in identifying the soft tissue structures of the stifle joint that could not be identified on radiographs. Magnetic resonance imaging confirmed normal integrity and signal of the patella tendon, collateral ligaments, and cranial cruciate ligament. Magnetic resonance imaging identified T2-hyperintensity, poor definition, and increased size of the caudal cruciate ligament and a focus of T2-hyperintensity in the medial femoral condyle, consistent with areas of edema and injury. Computed tomography offered improved spatial resolution and definition of bone in comparison to MRI. The CT scan best demonstrated the multiple avulsion fragments of the lateral femoral condyle.

Isolated caudal cruciate ligament injury is a rare condition. It is difficult to diagnose on physical examination alone. The drawer tests are poor indicators for determining the cause of stifle joint instability. For the dog of the present report, the combined use of radiography, MRI, and CT was useful in diagnosing caudal cruciate ligament rupture and the location of this injury along the ligament.