What Is Your Diagnosis?

**History**

An 8-year-old castrated male Labrador Retriever was evaluated because of right thoracic limb lameness of 2 weeks' duration. The lameness may have been precipitated by jumping on a curb during a routine walk. Treatment with an NSAID for 2 weeks by the referring veterinarian improved but did not completely alleviate the lameness. On physical examination, the dog had a body condition score of 7 of 9 and a grade 1 of 4 lameness in the right thoracic limb. There were mild signs of pain on palpation of the craniomedial portion of the right shoulder joint, but no joint effusion was noted. The patient had a history of hip dysplasia and tarsal osteoarthritis. Orthogonal radiographic views of the right shoulder joint were obtained (Figure 1).

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

---

This report was submitted by Thomas Monaco, DVM, and Pamela Schwartz, DVM, DACVS; from Veterinary Surgical Centers, 140 Park St SE, Vienna, VA 22180 (Monaco); and the Department of Surgery, Animal Medical Center, 510 E 62nd St, New York, NY 10065 (Schwartz). Address correspondence to Dr. Monaco (tmonaco@veterinarysurgicalcenters.com).
The signalment of the dog of this report and the shape and location of the fragment aided in ruling out the possible differential diagnoses. Osteochondritis dissecans, which is due to a defect in endochondral ossification, is usually seen in young large-breed rapidly growing animals at 4 to 10 months of age. The characteristic radiographic signs in the shoulder joint include flattening or concavity of the caudal humeral head with subchondral bone sclerosis. Concurrent osteochondritis dissecans lesions can include a mineralized cartilage flap occasionally seen within the subchondral defect or within the joint space. None of these signs were seen in the dog of the present report. An old fracture of the caudal aspect of the glenoid cavity with remodeled edges is also a differential diagnosis for incomplete fusion of the ACGOC. However, glenoid fractures of this type are rare and the lack of trauma in the history made this less likely.

A synovial osteochondroma is more difficult to rule out. These lesions are usually multiple, well-defined intra-articular mineralized opacities thought to arise from metaplasia of islands of cartilage produced by the synovial membrane. They can remain attached to the synovium or can break off and become a movable fragment within a joint (ie, a joint mouse). As with ACGOC, synovial osteochondromas can cause severe lameness that can be alleviated with surgical removal.

In the dog of the present report, the shape and location of the fragment made the diagnosis of incomplete fusion of the ACGOC most likely. The fragment conforms almost perfectly to the borders of the caudal aspect of the glenoid cavity, and the secondary osteophyte formation may be due to local inflammation from its instability. Because an ACGOC can be found in clinically normal dogs, it is important to rule out other contributing causes of lameness. Other diagnostic tests, such as joint fluid analysis, positive contrast arthrography, magnetic resonance imaging, or arthroscopy, can be used to evaluate other contributing causes of lameness aside from incomplete fusion of the ACGOC.

This patient was treated with medical management consisting of strict exercise restriction for 2 weeks and continued administration of an NSAID. Further diagnostic testing or surgical treatment would be considered if the lameness progressed or failed to respond to medical treatment. The owners had not reported any increased lameness since diagnosis, and no further treatment was instituted.