

What Is Your Diagnosis?

In cooperation with



Figure 1—Mediolateral radiographic view of the left shoulder joint of a 6-month-old Duroc gilt evaluated for a 1-month history of left forelimb lameness.

History

A 6-month-old 93-kg (205-lb) Duroc gilt was evaluated for a 1-month history of a grade 3/5 lameness of the left forelimb. Physical examination revealed moderate effusion of the left shoulder joint. Survey radiography (Figure 1) was performed to determine the cause of the lameness and effusion.

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

This report was submitted by Meredyth L. Jones, DVM, MS, DACVIM, and Robert N. Streeter, DVM, MS, DACVIM; from the Department of Veterinary Clinical Sciences, Center for Veterinary Health Sciences, Oklahoma State University, Stillwater, OK 74078. Dr. Jones' present address is the Department of Clinical Sciences, College of Veterinary Medicine, Kansas State University, Manhattan, KS 66506. Address correspondence to Dr. Jones.

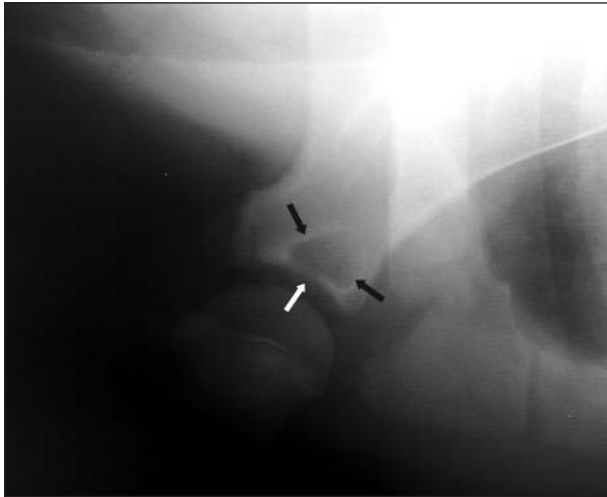


Figure 2—Detail of the mediolateral radiographic view in Figure 1. Notice the osteolytic lesion in the scapula, dorsal to the glenoid cavity (black arrows). Lysis of the subchondral plate of the glenoid cavity indicates communication with the glenoid cavity (white arrow).

Radiographic Findings and Interpretation

A smooth, well-marginated, triangular, 2.5-cm osteolytic lesion is visible in the left scapula, proximal to the glenoid cavity, along with lysis of the subchondral plate of the glenoid cavity (Figure 2). The humeral head appears normal. A caudocranial radiographic view of the left shoulder joint was also obtained, but the lesion could not be visualized. Radiography of the right shoulder joint did not reveal abnormalities. A diagnosis of subchondral bone cyst extending to the glenoid cavity was made.

Comments

Subchondral bone cysts have been reported in various species, including humans, although their cause is not fully understood. Several theories for subchondral cyst formation have been considered. A possible predecessor to subchondral cyst formation is the existence of osteochondrosis. In swine, sites of predilection of osteochondrosis, in order by decreasing frequency, are the stifle joint, elbow joint, lumbar intervertebral joints, tarsus, shoulder joint, and hip joint.¹ Swine become clinically affected by osteochondrosis between 4 and 8 months of age, with one slaughter survey reporting² a prevalence of osteochondrosis lesions to be 35.5%. In another study,³ mild to severe osteochondrosis of the

glenoid cavity was identified in 63% of culled sows, with 1.7% of the lesions being severe osteochondritis dissecans lesions. The population of the latter study included culled sows that were part of a lameness study with 80% of the sows being > 2 years old, which likely accounts for the increased rate of osteochondrosis lesions in those animals.

Radiographic reports of osteochondrosis in swine are limited, with most reports based on results of gross and histologic postmortem examinations. Studies^{1,2,4,5} in which radiography was used to diagnose osteochondrosis and subchondral bone lesions have used bones or sections of bone collected at postmortem examination. Studies^{5,6} using radiography in live animals for identification of lesions have had mixed results in animals < 100 days old. One study⁷ found computed tomography useful in the identification of osteochondrosis lesions on postmortem specimens.

As in other species, causes of osteochondrosis in swine are believed to be multifactorial. Hereditary influences, nutrition, growth rate, cartilage compression, exercise, hormones, and infectious agents have all been investigated, without definitive determination of the cause.⁸

Radiographic documentation of this lesion facilitates effective communication with swine producers regarding breeding and management decisions. Medical, surgical, or arthroscopic treatments were not pursued in the gilt of this report because of the articular involvement, large size of the lesion, and associated poor prognosis for the intended use of exhibition and breeding.

1. Reiland S. Morphology of osteochondrosis and sequelae in pigs. *Acta Radiol Suppl* 1987;358:45–90.
2. Turner GV, Collett MG, Veary CM, et al. Arthritis in slaughter pigs. *J S Afr Vet Assoc* 1991;62:107–109.
3. Jorgensen B. Osteochondrosis/osteoarthritis and claw disorders in sows, associated with leg weakness. *Acta Vet Scand* 2000;41:123–198.
4. Jorgensen B, Arnbjerg J, Aaslyng M. Pathological and radiological investigations on osteochondrosis in pigs, associated with leg weakness. *Zentralbl Veterinarmed A* 1995;42:489–504.
5. Hill MA, Hilley HD, Feeney DA, et al. Dyschondrodysplasias, including osteochondrosis, in boars between 25 and 169 days of age: radiologic changes. *Am J Vet Res* 1984;45:917–925.
6. Bittegeko SB, Arnbjerg J. Radiological aspects on the course of development of porcine epiphyseal osteochondrosis (OCD) from 42 up to 147 days of age. *Zentralbl Veterinarmed A* 1994;41:369–376.
7. Empel W, Sehested E. Qualitative, semiquantitative and quantitative diagnosis of osteochondrosis in pigs by computed tomography (CT). *Acta Agric Scand* 1986;36:186–194.
8. Hill MA. Causes of degenerative joint disease (osteoarthritis) and dyschondroplasia (osteochondrosis) in pigs. *J Am Vet Med Assoc* 1990;197:107–113.