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What Is Your Diagnosis?

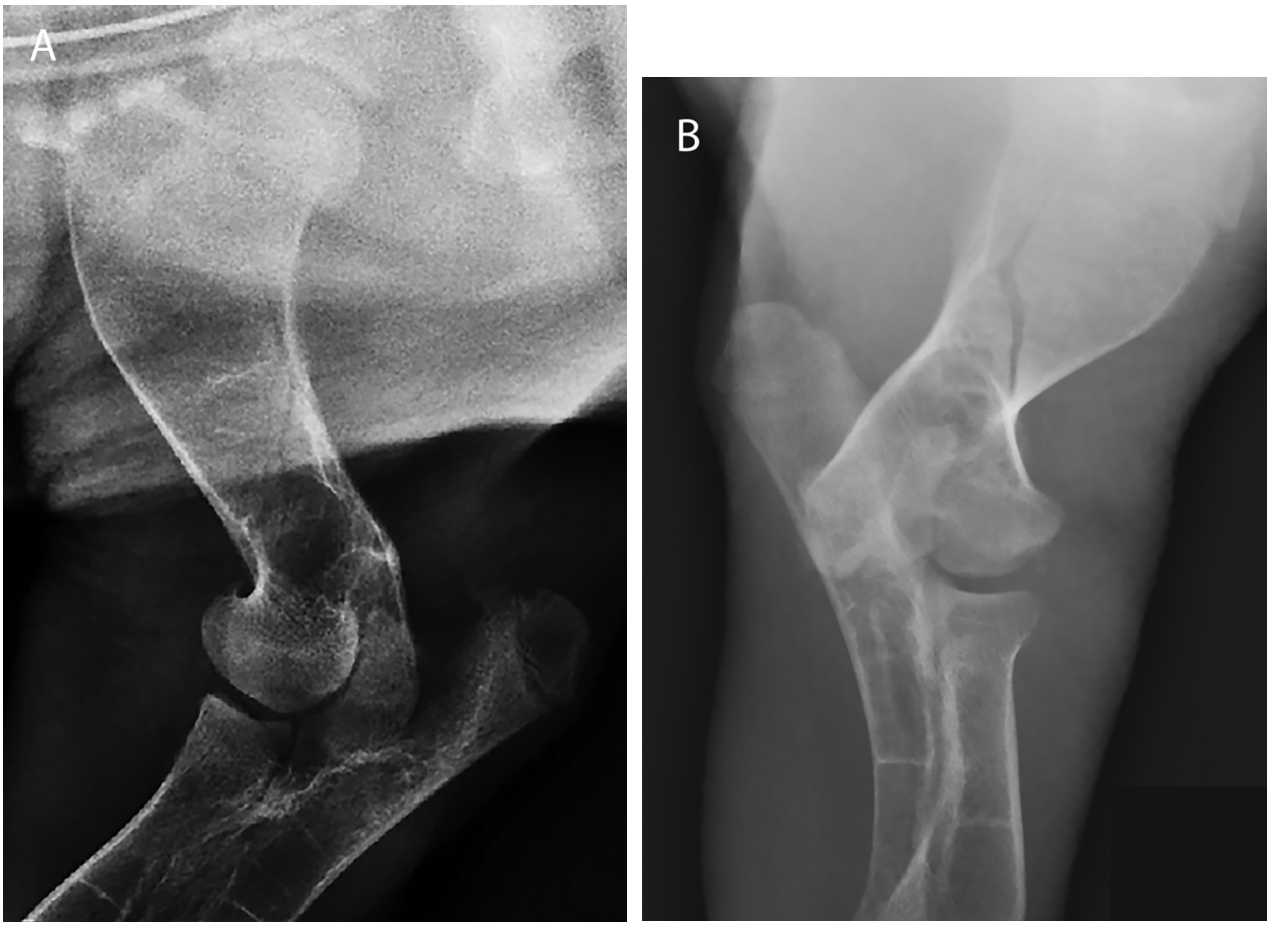

Figure 1—Lateral (A) and oblique craniocaudal (B) radiographic views of the left humerus of a 3-year-old 10.4-kg (22.9-lb) castrated male Juliana pig that was evaluated for acute left forelimb lameness after falling off a 3-foot-high porch.

History

A 3-year-old 10.4-kg (22.9-lb) castrated male Juliana pig (also known as a Miniature Painted Pig) was referred for a non–weight-bearing lameness of the left forelimb that developed after falling off a porch of a height of approximately 3 feet. Following the fall, the patient was lame and water intake was decreased, but there were no changes in the patient's behavior or appetite, according to the owner. The pig's diet consisted of oats, fruits, and vegetables. Physical examination revealed left forelimb non–weight-bearing lameness, but the pig was otherwise apparently healthy. While the pig was under general anesthesia, radiographs of its left humerus **(Figure 1)** and left elbow joint were obtained.

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

This report was submitted by Leah M. Moody, BSc, and Erin Brinkman, DVM; from the Department of Clinical Sciences, College of Veterinary Medicine, Mississippi State University, Mississippi State, MS 39762.

Address correspondence to Dr. Brinkman (brinkman@cvm.msstate.edu).

Diagnostic Imaging Findings and Interpretation

A closed, simple, incomplete, long oblique mid-diaphyseal fracture that extends from the caudoproximal to craniodistal aspect of the humerus is evident **(Figure 2)**. There is no displacement or angulation of the fracture. Mild, smooth periosteal proliferation with a long zone of transition is present at the mid-diaphysis of the caudal aspect of the humerus. Cortices of the humerus are thin, and the overall opacity of the bone is decreased. Radiography of the left elbow joint (images not shown) also revealed thin cortices and decreased opacity of the radius and ulna.

Differential diagnoses for the incomplete fractures of the humerus included traumatic and pathological fractures. Cortical thinning and decreased overall opacity of bone can result from metabolic disease or disuse osteopenia.[1] Disuse osteopenia was not considered likely in this pig because changes in bone from disuse do not become radiographically evident until 4 to 6 weeks following injury or immobilization.[2] Given these imaging findings, dietary history, and history of only mild trauma, the presumptive diagnosis was pathological fractures due to metabolic disease.

Because of the decreased bone mineralization and thin cortices, surgical fixation carried a substantial risk of exacerbating the fractures and was not performed. Alternatively, the patient was prescribed strict cage rest for 4 to 6 weeks to allow the fractures to heal by second intention. Carprofen (4 mg/kg [1.8 mg/lb], PO, q 24 h) and tramadol (4 mg/kg, PO, q 12 h) were prescribed to manage pain, and it was recommended that a balanced commercial pig feed be added to the diet.

Seven weeks later, the patient was readmitted to the hospital for left forelimb lameness and swelling of the left carpometacarpal region after jumping from its owner's arms. Ultrasonography of the left metacarpal region was performed **(Figure 3)** to identify the cause of the soft tissue swelling and evaluate the extent of the injury. Discontinuities of the cortices of the third and fourth metacarpal bones were discovered at the mid-diaphyses, and a small amount of anechoic fluid was present superficial to the bones.

Radiographs of the left metacarpal region were obtained to further evaluate the suspected fractures **(Figure 4)**. Radiography revealed a closed, simple, short oblique, complete fracture of the mid-diaphysis of the third metacarpal bone with dorsal and medial displacement. A closed, simple, short oblique, incomplete fracture of the mid-diaphysis of the fourth meta-

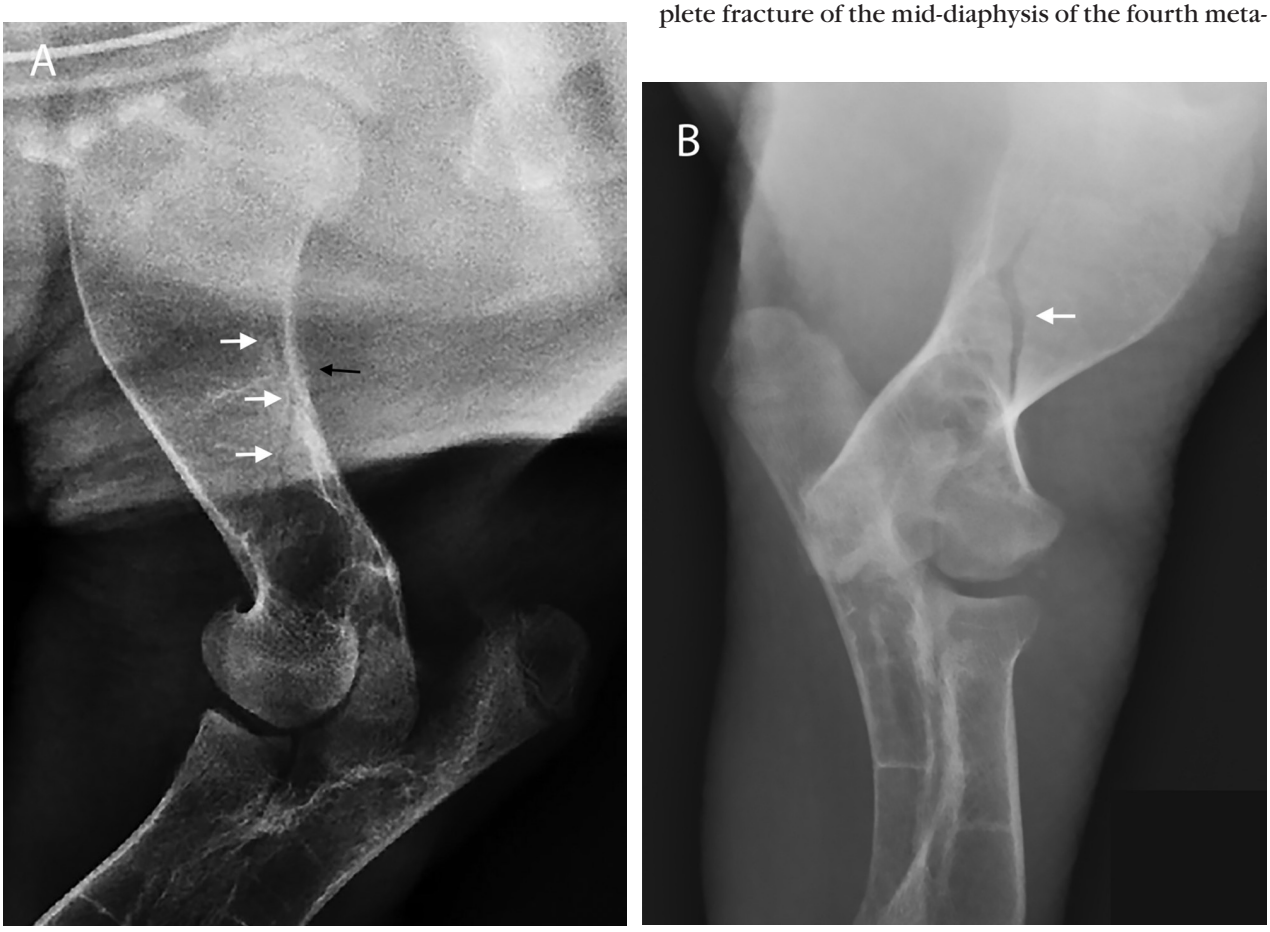

Figure 2—Same radiographic images as in Figure 1. A closed, simple, incomplete, long oblique fracture of the left humeral mid-diaphysis is evident (white arrows). Mild smooth periosteal new bone is present along the caudal aspect of the humeral diaphysis (black arrow). Humeral, radial, and ulnar cortices are thin, and there is an overall decrease in opacity of the bone.

carpal bone was also evident. There was decreased opacity of the radius, ulna, carpal bones, and phalanges. Mild extracapsular soft tissue swelling was present surrounding the fracture sites.

The complete and incomplete fractures, cortical thinning, and decreased overall opacity of the third and fourth metacarpal bones further supported the initial diagnosis of metabolic disease.

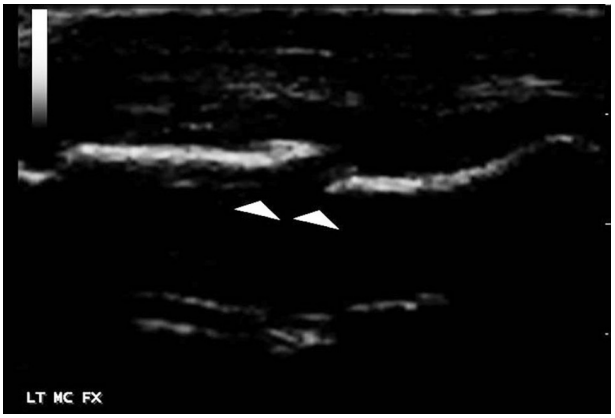

Figure 3—Ultrasonographic image of the left third metacarpal bone of the pig in Figure 1. Notice the discontinuity of the cortex at the mid-diaphysis (arrowheads) and a small amount of anechoic fluid superficial to the bone. Image was obtained with a 7.5- to 12-MHz linear array transducer.

Treatment and Outcome

The patient was prescribed additional cage rest. Increasing the dietary calcium-to-phosphorus ratio to 3:1 by supplementing with over-the-counter calcium carbonate (288 mg/kg [130.9 mg/lb], PO, q 24 h) was recommended to treat the imbalance. Recheck radiography was recommended following 6 months of treatment to assess fracture healing and bone den-

Figure 4—Lateral (A) and dorsopalmar (B) radiographic views of the left metacarpal region of the pig in Figure 1. In both views, a closed, simple, short oblique, complete fracture of the mid-diaphysis of the third metacarpal bone is evident as well as a closed, simple, short oblique, incomplete fracture of the mid-diaphysis of the fourth metacarpal bone (arrows). There is decreased overall opacity of the radius, ulna, carpal bones, and phalanges.

sity; however, this was not performed. Ten months following the initial diagnosis, the patient had greatly improved, and there was no persisting lameness, gait abnormality, or angular limb deformity.

Comments

Considering the history of mild trauma, diet, and radiographic findings, a diagnosis of nutritional secondary hyperparathyroidism with multiple pathological fractures was made. Nutritional secondary hyperparathyroidism is a generalized metabolic bone disease that is characterized by osteopenia and can be caused by excess phosphorus or inadequate calcium or vitamin D in the diet. This condition is no longer common because balanced commercial pet foods are now widely available; however, it can be seen in pets eating natural, homemade, or all-meat diets, especially if the animal is still growing.[3]

Clinical signs of nutritional secondary hyperparathyroidism can range from no signs of illness to acute signs of orthopedic pain and pathological fractures. Signs of hypocalcemia, such as muscle fasciculations, seizures, and arrhythmias, are possible in severe cases. A serum assay may reveal an increased parathyroid hormone concentration, which is a definitive diagnosis of nutritional secondary hyperparathyroidism.[4] One study[5] showed that measuring the calcium and phosphorus concentrations in urine can be useful in evaluating dietary insufficiency or excess of these minerals in growing pigs.

Radiographic findings of nutritional secondary hyperparathyroidism include generalized decrease in bone opacity, cortical thinning, and greenstick or folding fractures.[3,6] Because each of these findings was present in the pig of the present report and a serum parathyroid hormone assay was not performed, radiographic imaging was paramount in making the diagnosis. The patient's improvement with treatment and diet change further supported the diagnosis. Additionally, advanced imaging, such as CT or dual energy x-ray absorptiometry, can be used to measure bone mineral density to facilitate the diagnosis or assess the treatment of nutritional secondary hyperparathyroidism.[3]

Treatment of this condition includes supportive care and surgical repair of pathological fractures, if indicated. The incorporation of a well-balanced diet that is appropriate for the growth stage of the animal is essential.[4] Once the dietary imbalance of calcium and phosphorus is corrected, the reversal of bony changes is relatively rapid.[6]

Acknowledgments

No third-party funding or support was received in connection with the writing or publication of this manuscript.

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