The University of Georgia

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Case #: A15-3749

Presenter: Susan Fogelson, DVM, MS (fogelsb@uga.edu)
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Signalment: African crowned crane

History: Presented for necropsy was an approximately 5-week-old African crowned crane with a reported history of being a "poor doer" with an inability to walk well. The bird had a poor appetite and a suspected broken leg for 1 week in duration. Radiographs suggested incomplete development of the leg bones.

Gross Findings and Ante-mortem Diagnostic Information: The crane was in good nutritional status with a BCS of 3/5 and there was moderate postmortem autolysis. The left hind leg had a significant focal deviation of the proximal tibiotarsal bone with mobility at this focus. A large dark red, blood clot expanded the subcutaneous tissues and muscle extending from the inguinal region medially to the mid tibiotarsus. Surrounded by this area of hemorrhage, the proximal tibiotarsal bone had a closed, simple, oblique, complete fracture that protruded through the muscle and was displaced caudally. At the level of the fracture the bone was also cranially bowed. The proximal tarsometatarsus of both legs were moderately flared.

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Histopathology: The physeal cartilage is markedly thickened and disorganized, up to 5 times normal. The zone of proliferation is expanded up to 3 mm wide, and there is retention of the zone of hypertrophy as thick tongues of cartilage that extend into the diaphysis and fill the medullary cavity. Bony trabeculae are sparse and thin (osteopenia). Bony and cartilage trabeculae are often fractured and surrounded by hemorrhage, fibrin, and edema admixed with moderate numbers of heterophils. Medullary spaces contain small amounts of bone marrow, plump fibroblasts and collagen (myelofibrosis), and osteoclasts, often within Howship's lacunae. Cortical bone is markedly thinned, discontinuous, and essentially parallel from the metaphysis to the cut border (loss of the cutback zone). The periosteum is diffusely thickened by fibrous connective tissue. The epiphysis is diffusely cartilaginous and lacks mineralization.

Morphologic Diagnoses:
Tibiotarsus: Failure of endochondral ossification, with failure of chondroclasis, persistent metaphyseal and diaphyseal tongues of hypertrophied chondrocytes, myelofibrosis, and periosteal fibrosis

Comment: Gross and histologic evaluation of the tibiosarsal bones of this crane are consistent with rickets. Rickets is a disease that affects young, rapidly growing animals prior to epiphyseal closure and is characterized by abnormal endochondral ossification leading to defective bone formation, deformities, and fractures. In avian species, a nutritional etiology is suspected such as seen with insufficient dietary calcium, vitamin D_3, or phosphorus and excess calcium or phosphorous[1,2]. Defective bone formation predisposes these animals to curving of long bones and folding fractures. The pathogenesis of this disease includes failure of mineralization of cartilage at the growth plate followed by failure of hypertrophic chondrocytes to degenerate. There is failure of capillary invasion from the metaphysis and lack of osteoprogenitors and osteoblasts along with persistence of hypertrophic chondrocytes at sites of endochondral ossification that ultimately produce thick cartilage trabeculae[3]. In addition, trabecular bone fails to mineralize and osteoclasts cannot bind so there is reduced bone and cartilage removal in cutback zones, impaired bone remodeling, and thickened, irregular metaphyseal trabeculae covered in thin seams of un-mineralized osteoid[3]. Due to the decreased stability microfractures and infractions are common. A definitive underlying cause was not determined in this case.

References:
1. Dinev, I. (2012). Clinical and morphological investigations on the incidence of forms of rickets and their association with other pathological states in broiler chickens. *Research in veterinary science*, *92*(2), 273-277.
2. Schmidt, R. E., Reavill, D. R., & Phalen, D. N. (2008). *Pathology of pet and aviary birds*. John Wiley & Sons. 155-156
3. Zachary, J. F., & McGavin, M. D. (2013). *Pathologic basis of veterinary disease*. Elsevier Health Sciences. 948.