# Latin Comparative Pathology Group

## The Latin Subdivision of the CL Davis Foundation

# Diagnostic Exercise

**Case #: 25 Month: September Year: 2012**

**Answer sheet**

**Contributor:** Fabio Del Piero, DVM, PhD, DACVP, Professor of Pathology, Department of Pathobiological Sciences, School of Veterinary Medicine, Louisiana State University, Baton Rouge, 70803.

**Signalment:** Pig, cross breed, red, 2-year-old, male, recently castrated.

**Clinical History:** The clinical signs were mostly attributed to a left hind limb disorder. The affected leg appeared shorter than the right leg. Sudden hind limb lameness was observed and pain at the hip was detected. There was also slowness to rise, and walking on tiptoes with short steps. Eventually the patient showed marked reluctance to rise and was euthanized.

**Necropsy Findings:** Left femoral head epiphyseal detachment and moderate to severe displacement was observed. In addition there was scrotal fibrosing, suppurative dermatitis and tunical vaginalitis.

**Fixation:** 10% Buffered formalin.

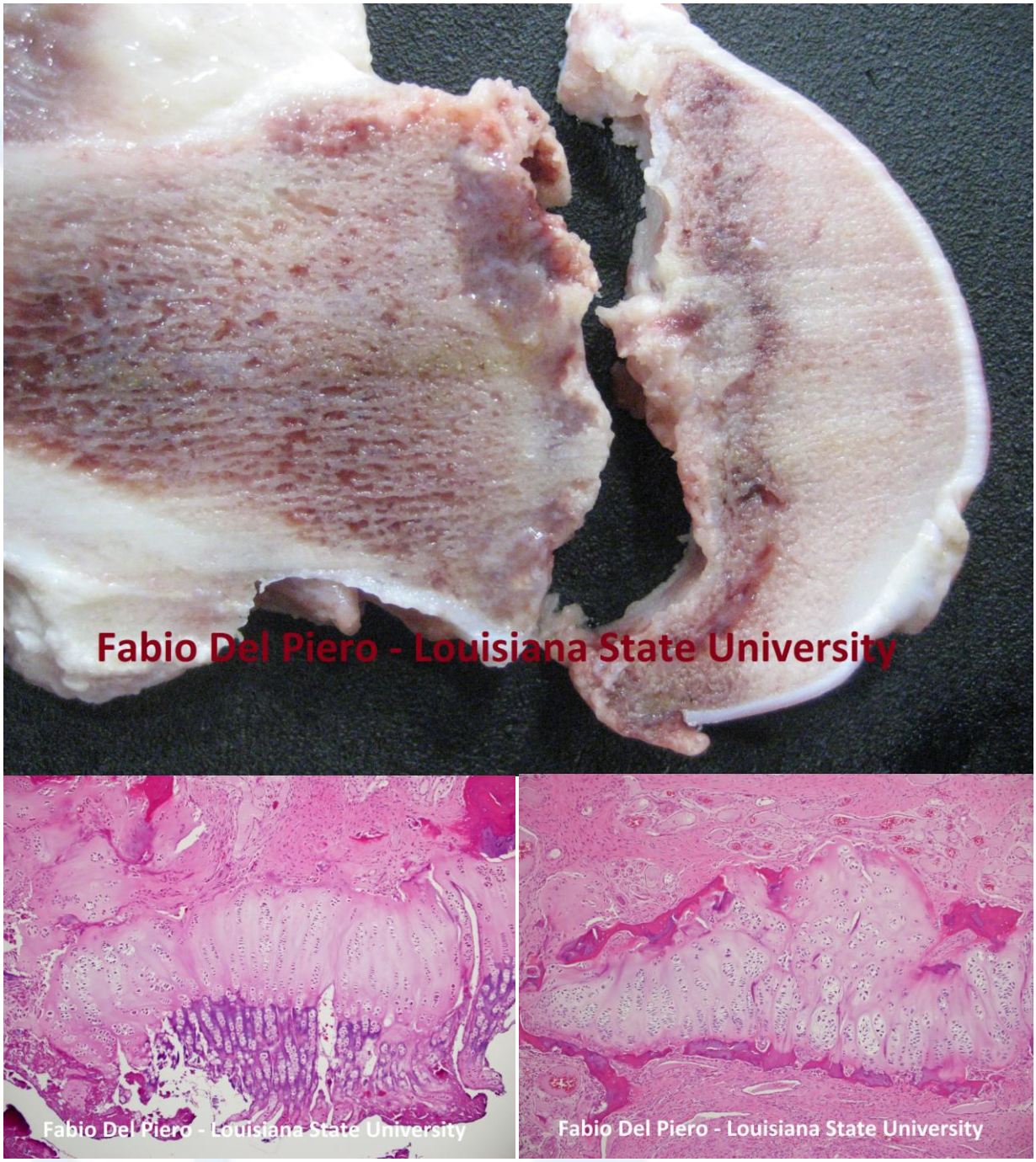


**Figure # 1:** *Left femoral head epiphyseal detachment and moderate to severe displacement (longitudinal mid section).*

**Histologic Description**

*Femur head*. The lesions are physeal and there are no significant changes involving the articular cartilage and the subchondral subarticular spongiosa. There is unusually prominent and irregular retention of physeal cartilage and separation in the middle of the hyperplastic zone with digitiform fringes on the ventral detachment side. This cartilage present longitudinal interruptions and contain branching eosinophilic streaks. Areas of eosinophilic cartilage (degeneration) have some small chondrocytes with hyperchromatic nuclei. Multifocally areas of hyaline cartilage alternate with bony trabeculae and areas of eosinophilic cartilage. There is oblique to irregular arrangement of chondrocytic columns in zones of proliferation and maturation. Clustering of chondrocytes (irregular chondrones) are frequently observed. These chondrocytes are smaller, and round to polygonal, with scant cytoplasm most closely resembling the mitotic pairs of the reserve zone. Loosening of the cartilage matrix is also present. There is osteonecrosis with hypereosinophilia and loss of osteocyte nuclei, and osteoclastic bone resorption. Multifocal to coalescing intraphyseal fibroplasia extends toward the metaphysis and toward the diaphysis (the latter not in the slide). These areas are irregularly vascularized and contain foci of osteoclastic aggregation associated with small fragments of necrotic bone or short necrotic lamellae. Some osteoclast are very large and with numerous nuclei. There is multifocal histiocytic and plasmacytic infiltration. Additionally there is mild to moderate multifocal hemorrhage.

[The contralateral femoral epiphysis had a 1 mm thick, undulant and sometimes irregular cartilaginous component with areas of abnormal chondrocyte clustering and oblique chondrocyte columns. No eosinophilic streaks, necrosis and areas of detachment were observed].

**Morphologic diagnosis**

- Proximal femoral physeal endochondral ossification defect with cartilage retention, necrosis, epiphysiolysis and femoral head detachment
- Microfractures of cartilage and bone, multifocal, chronic

**Diagnosis**

Osteochondrosis with epiphysiolysis and femoral head detachment

**Discussion**

This is a case of leg weakness caused by epiphysiolysis which is a form of osteochondrosis. Traumas and consequent microfractures significantly contributed to the retention of a thick and irregular epiphyseal cartilage and to fibrous tissue formation. Osteomyelitis could have predisposed by necrosis of bone and cartilage or could have been present before (neonatal infection). "*Leg weakness*" is a locomotor disability of pigs unassociated with infectious arthritis. It is a combination of noninfectious arthropathy and osteopathy, and is a significant cause of mandatory culling in pig herds. Causes are defects of conformation, osteochondrosis (including epiphysiolysis), arthrosis, lumbar intervertebral disk degeneration, and spondylosis. The clinical syndrome varies from lameness to difficulty in rising to

recumbency. Characteristic signs are carrying of a hind leg, sitting on the haunches for long periods, and shuffling gait.

*Osteochondrosis* is defined as a *focal disturbance of enchondral ossification* and is regarded as having a multifactorial etiology, with no single factor accounting for all aspects of the disease. The most commonly cited etiologic factors are heredity, rapid growth, anatomic conformation, trauma, and dietary imbalances; however, only heredity and anatomic conformation are well supported by the scientific literature. The way in which the disease is initiated has been debated. Although formation of a fragile cartilage, failure of chondrocyte differentiation, subchondral bone necrosis, and failure of blood supply to the growth cartilage all have been proposed as the initial step in the pathogenesis, some literature supports failure of blood supply to growth cartilage as being the most likely. Based on all available evidence, the primary lesion of articular osteochondrosis could be defined as *focal ischemic necrosis of growth cartilage initiated by necrosis of cartilage canal blood vessels*. Because the necrotic cartilage does not undergo mineralization or vascular penetration, *focal failure of enchondral ossification* occurs when the ossification front approaches the lesion. Osteochondrosis can be subclassified as *latens* (lesion confined to epiphyseal cartilage), *manifesta* (lesion accompanied by delay in endochondral ossification), and *dissecans* (cleft formation through articular cartilage).

*Epiphysiolysis* is a form of osteochondrosis (OCD) and is the separation of an epiphysis from metaphyseal bone. It is a *traumatic lesion predisposed by a defect in growth cartilage of the physis*. It may develop either from and extended *eosinophilic streak (areas of matrix degeneration)* or from areas of necrosis in the growth of cartilage, rather than from foci of metaphyseal dysplasia. These irregular eosinophilic streaks, associated with areas of disorganized growth plate architecture, occur in osteochondrosis. These may reflect either vestiges of cartilage canals or infraction lines occurring as a sequel to growth plate trauma. Similar eosinophilic streaks are normally present in the growth plate of young animals but are usually parallel to cartilage columns. In osteochondrosis the eosinophilic streaks are often stellate and may subdivide the physeal cartilage into disorganized sometimes degenerate lobules. The common sites for epiphysiolysis are femoral head, ischiatic tuberosity of females and lumbar vertebrae. The distal epiphysis of the ulna and anconeal process can also be involved, although strictly speaking the lesion involving the latter should not be called epiphysiolysis. In pigs unlike in dogs the anconeal process does not develop from a separate ossification center, so apophysiolysis is a more appropriate term. Separation may be complete and is often the case with the ischiatic tuberosity or partial as occur in the head of the femur which sometimes remains attached at its lateral margin. Separation probably occurs when the process of endochondral ossification reaches or approaches the cartilage defect. The resulting fracture may extend in a jagged crack through primary and secondary spongiosa. It may be that the traumatic forces applied to the epiphyseal cartilage at the site of empty spaces near atrophic blood vessels or at eosinophilic streaks cause further separation and epiphyseal lysis.

In summary the pathogenesis of physeal osteochondrosis is still poorly elucidated; however, failure of blood supply, either from the epiphyseal or metaphyseal side of the plate, may be involved.

**Ref:** Ytrehus B, Carlson CS, Ekman S. Etiology and pathogenesis of osteochondrosis. Veterinary Pathology 2007 44(4): 429-48. Review.

Please send your comments/questions to the whole LCPG list by hitting “reply to all”.

A final document containing this material with answers and a brief discussion will be posted on the C. L. Davis website by the end of the current month (http://www.cldavis.org/lcpg_english.html).