The Armed Forces Institute of Pathology
Department of Veterinary Pathology
WEDNESDAY SLIDE CONFERENCE
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CONFERENCE 10
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The Ohio State University
Dept of Veterinary Biosciences
Columbus, OH

CASE I – 03-1893 (AFIP 2948659)

Signalment: 6-7 month gestation, female, Holstein, bovine fetus.

History: Normal pregnancy. Cow was euthanatized because of luxated proximal left femur.

Gross Pathology: None in the fetus.

Contributor's Morphologic Diagnosis: Normal plexiform cortical bone, radius and ulna.

Contributor's Comment: Normal appositional growth (widening) of cortical bone can be circumferential lamellar, simple primary osteonal, plexiform, and saltatory.[1] Laminar bone is composed of lamellae deposited circumferentially in layers parallel to the convex surface of the cortex. Primary osteonal bone consists of anastomosing vascular Haversian canals surrounded by concentric lamellae forming Haversian systems. Plexiform bone is formed by multiple relatively widely spaced lamina of periosteal woven. The spaces between these woven laminae subsequently fill in (compact) with lamellar bone. In the horse, the compaction can form osteons (saltatory formation) with the orientation of the osteon and its vessels being perpendicular to the long axis of the bone.[1] In calves, the compaction (and blood vessels) are oriented parallel with the convex surface of the bone[2] without formation of osteons. Many species exhibit multiple patterns of primary periosteal cortical bone, dependent upon regional rate of growth, pattern of vascularization, and functional requirements.[3,4]

AFIP Diagnosis: Bone, radius and ulna (per contributor): Normal fetal bone, Holstein, bovine.

Conference Comment: There are structural variations in the microscopic organization of bone tissue in different animal species depending on the growth and remodeling processes for that species. Bones undergo change in size and shape as well as remodeling of the internal architecture during normal growth and due to changes in functional stresses throughout life. These processes result in bone deposition, resorption, and remodeling, which ultimately affect the amount of bone, the amount of mineralization, and type of bone tissue present within a given bone.[1]

Fetal bone development occurs in two ways, both of which involve the replacement of primitive collagenous supporting tissue by bone. The long bones, vertebrae, pelvis, and bones of the base of the skull are formed through a continuously growing cartilage model that is progressively replaced by bone (endochondral ossification). The bones of the vault of the skull, the maxilla and most of the mandible are formed by the deposition of bone within primitive mesenchymal tissue (intramembranous ossification). Regardless of the method of ossification, initially the bone that is formed is immature, or woven bone. The developing bone is then extensively remodeled by resorption and appositional growth to form mature, or lamellar bone.[5]

Contributor: The Ohio State University, College of Veterinary Medicine, Department of Veterinary Biosciences, 1925 Coffey Rd., Columbus, Ohio http://www.vet.ohio-state.edu/docs/biosci/index.html

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CASE II – X7764-6 or X7764-7 (AFIP 2946673)

Signalment: A juvenile female Black-crowned night heron (*Nycticorax nycticorax*).

History: Found dead. This was an individual among a wild flock that nests around the bird house at the National Zoo.

Gross Pathology: Poor nutritional condition; hepatomegaly with miliary hepatitis; splenomegaly; parasitism, roundworm, ventriculus and small intestine; arthritis, tibiotarsal-metatarsal joint, chronic.

Laboratory Results:
West Nile virus PCR – negative
Liver culture – *Salmonella typhimurium*
Liver cytology – Marked, subacute inflammation with intracellular, gram-negative coccobacilli
Heart blood culture – coagulase-negative *Staphylococcus* sp.

Contributor's Morphologic Diagnosis: Bone, tibiotarsus: Osteomyelitis, granulomatous, with trabecular resorption, periosteal new bone formation, marrow cavity bone and cartilage sequestrum, and intralesional coccobacilli, Black-crowned night heron (*Nycticorax nycticorax*), Ciconiiformes.

Contributor's Comment: Salmonellosis is a worldwide disease affecting a wide range of species from humans to birds to reptiles to insects. This bacterium used to be employed in the control of rodents, but the practice was banned in the US when the public health risk was realized. Clinical presentations most commonly linked to *Salmonella* include enteritis, colitis, abortion, and septicemia. Additionally, carrier states can be established with *Salmonella* sp. in a non-clinical or subclinical host. In our experience, infection in these hosts cannot be eliminated through antibiotic administration, and may only serve to create resistant strains of the bacterium.

In birds, salmonellosis is usually fatal in the animal's first 2-3 weeks of life. Older birds may survive the infection to become carriers of the pathogen. The two most common *Salmonella* diseases in domestic fowl are Pullorum Disease and Fowl Typhoid, caused by *S. pullorum* and *S. gallinarum* respectively. In either disease, young birds may have splenomegaly, hepatomegaly, and caseous material within the yolk sac. Joints, especially the hock, may become enlarged and filled with viscous, yellow fluid. A common gross lesion in older, female birds is misshapen, firm, and discolored ovarian follicles. Pericarditis and hydropericardium may also be seen. Microscopically, inflammation is initially heterophilic and lymphocytic, and progresses to necrotizing and granulomatous lesions in target organs.

Wild waterfowl are less susceptible to *Salmonella*-induced disease. Pullorum Disease and Fowl Typhoid are only rarely reported in waterfowl. The most common *Salmonella* strain isolated from wild waterfowl is *S. typhimurium*, the strain isolated from this heron. In this case, infection was of utmost concern, as potential shedders of the organism live in and around the wetlands exhibit and defecate in close proximity to collection animals and the public.

AFIP Diagnoses: 1. Bone, tibiotarsus (per contributor): Osteomyelitis, granulomatous and heterophilic, multifocal, marked, with medullary bone and cartilage sequestrum, trabecular resorption, periosteal new bone formation, and colonies of coccobacilli, black-crowned night heron (*Nycticorax nycticorax*), avian. 2. Tendons, leg: Tenosynovitis, chronic-active, proliferative, multifocal, minimal to moderate.

Conference Comment: There are two species of *Salmonella*: *S. enterica*, which is very common and comprised of over 2000 serotypes; and *S. bongori*, comprised of 10 serotypes, all of which are rare. The Kauffmann-White classification system for serotypes is based on differences among somatic (O), capsular (Vi), and flagellar (H) antigens. Each serotype is named by where it was first isolated or by the clinical syndrome it produces in a particular host. In conventional terminology, the serotypes are treated as species. Based on host specificity, serotypes can be divided into two groups: those that are highly adapted to a specific host species, and those that affect a wide range of species. Most serotypes fall into the latter category, although there can be marked differences in virulence of a serotype in various hosts.[3]

Some common and important animal diseases caused by *Salmonella* sp. include the following:[4,5]

Species	*Salmonella* sp.	Disease/Lesions
Porcine	*S. choleraesuis*	Septemia (piglets) (cyanosis of the skin, turkey-egg kidney); necroulcerative enterocolitis (button ulcers); hepatic paratyphoid nodules
	S. typhimurium	Enterocolitis; rectal stricture
	S. typhisuis	Ulcerative enterocolitis; caseous tonsillitis and lymphadenitis
Equine	*S. typhimurium*	Septemia (foals); enterocolitis (older horses)
	S. abortus-equi	Abortions (6-9 months of gestation); orchitis

Bovine	*S. dublin*	Fibrinous cholecystitis
	S. typhimurium	Septemia (calves); fibrinonecrotic enteritis and necrosis of the Peyer's patches
Ovine	*S. abortus-ovis*	Abortion
	S. typhimurium	Septemia (lambs); fibrinonecrotic enteritis
	S. dublin	Fibrinous cholecystitis
	S. enteritidis	Enterocolitis
Avian	*S. typhimurium*	Septemia
	S. pullorum	Pullorum disease: necrotizing typhilitis and necrotic foci in the liver, lung, myocardium, gizzard (chicks); oophoritis (adults)
	S. gallinarum	Fowl-typhoid: catarrhal enteritis and necrotic foci in the liver, myocardium, intestine, pancreas
Carnivores	*S. dublin*	Rare; associated with septemia in puppies
	S. typhimurium	Rare; gastroenteritis in immunosuppressed kittens
Lab animals	*S. typhimurium*	Uncommon; mice, gerbils, hamsters, rabbits, guinea pigs; non-human primates
	S. enteritidis	Uncommon; rats, hamsters, rabbits, guinea pigs; non-human primates

Contributor: Smithsonian National Zoological Park, Department of Pathology, 3001 Connecticut Ave, NW, Washington, DC
http://nationalzoo.si.edu/default.cfm

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CASE III – N2004-0345 (AFIP 2942032)

Signalment: 10 day-old, male, boat-billed heron (*Cochlearius cochlearius*).

History: This animal was the first offspring produced from a group of boat-billed herons, and presented after falling from the nest. On physical exam, all long bones were very flexible. Radiographs revealed multiple fractures of the long bones with very poor bone density. Due to a poor prognosis, humane euthanasia was elected.

Gross Pathology: Gross findings confirmed moderate to severe flexibility of all bones, with multiple folding fractures. Additionally, there was a 0.5 cm diameter focus of subdural hemorrhage within the right frontal region of the cerebrum (consistent with trauma associated with the recent fall).

Laboratory Results:
Serum calcium: 6.0 MG/DL
Serum phosphorus: 10.5 MG/DL
Serum 25-hydroxy Vitamin D: 36 nmol/L*
*Laboratory comment: Reference values for this test are not defined for this species in our laboratory. However, this concentration is similar to values seen in chickens, turkeys, and conures.
Serum Vitamin A: Retinol = 610 ng/ml**
**Laboratory comment: We do not have serum vitamin A reference ranges for boat-billed herons. For comparison, in adult mammals, adequate serum vitamin A (retinol) concentrations range between 175 to 500 ng/ml.

Contributor's Morphologic Diagnoses: Long bone: Elongation and thickening of the zone of hypertrophic cartilage, with excess osteoid (unmineralized matrix), fibroplasia, and folding fracture (rickets).
Parathyroid (not submitted): Hyperplasia, diffuse, moderate.

Contributor's Comment: The gross and histologic findings within the long bones of this bird are consistent with rickets. Rickets is caused by the failure of mineralization of newly deposited osteoid and may be caused by deficiencies of vitamin D, calcium, or phosphorus, as well as excess calcium or phosphorus. Grossly, affected birds have swollen joints, soft bones, and flared metaphyses. Curved deformities and folding fractures may occur.[1]

Much research has been done on the different histologic lesions associated with rickets depending on the specific nutrient imbalances present. With calcium deficiency, there is disorganization and thickening of cartilage within the zone of

proliferation, with poor physeal vascularization. Only a small zone of hypertrophic cartilage is present in this type of rickets. Calcification of the hypertrophic cartilage is reduced, and there is little vascular invasion of this cartilage. At the base of the cartilage, few calcified bone trabeculae are present, with a loss of the normal longitudinal arrangement.[1,2] The defect of matrix calcification appears to be due to lack of continuation of mineral deposition, rather than failure of its initiation. In one study, electron micrographic studies revealed that failure of progression of matrix deposition is likely due to the presence of hypertrophic cells (responsible for the initiation of matrix deposition).[3] The bone marrow is often replaced by fibrous tissue, and osteoclasts are abundant. The defect in calcium-deficiency rickets is thought to be impaired hypertrophy of chondrocytes, rather than increased cell replication. In calcium-deficiency rickets, the parathyroid glands are often hypertrophied.

Conversely, with phosphorous deficiency, the zone of proliferation is unchanged, but the hypertrophic zone is elongated and thickened, and there is defective mineralization of the hypertrophic cartilage cells resulting in long columns of cartilage, surrounded by wide unmineralized osteoid seams extending into the primary spongiosa. There is normal invasion by metaphyseal blood vessels. Osteoclasts are reduced, osteoblasts are increased, and amounts of osteoid are limited.[1,2] This condition is thought to be due to decreased resorption of hypertrophic cartilage by chondroclasts, rather than an increased proliferation of chondrocytes.[4] Birds with phosphorus-deficiency rickets often have atrophy of the parathyroids.[2]

Vitamin D-deficiency rickets results in lengthening and disorganization of the proliferating zone and variable lengthening and dysplasia of the mineralizing zone. The primary spongiosa is short, thick cartilage columns.[1] As in calcium-deficiency rickets, there is often parathyroid gland hyperplasia.[2]

It is interesting to note that the morphologic appearance of rickets in this case is most consistent with decreased phosphorous. However, the presence of parathyroid hyperplasia and relatively low serum calcium with high serum phosphorous is suggestive of disease due to hypocalcemia. It should be stressed that the previously-described histologic patterns are general guidelines only, and may vary according to deficiency. Other causes of rickets, such as magnesium toxicity, excess vitamin A or fat in the diet, mycotoxins, and enteritis have also been reported, and may cause modifications in the typical morphology.[2]

AFIP Diagnoses: 1. Long bone: Failure of endochondral ossification and retained cartilaginous cores, with increased osteoid seams (rickets), boat-billed heron (*Cochlearius cochlearius*), avian.
2. Long bone: Fracture with callus formation.

Conference Comment: As mentioned by the contributor, rickets in avian species may be the result of a deficiency in vitamin D, calcium, or phosphorus as well as an excess of calcium or phosphorus. Although there may be histological differences in rachitic bones depending on the cause, the common denominator is the failure of mineralization of newly deposited osteoid (failure of endochondral ossification), which results in bone deformities and fractures.[1]

In domestic animals, rickets is most commonly caused by a deficiency in vitamin D or phosphorus. However, it may also result from chronic renal disease or chronic fluorosis. Histologically, the lesions in domestic animals are similar to those seen in birds. There is abnormal endochondral ossification characterized by failure of mineralization of the growth plate cartilage and osteoid. However, there is also disorganization of chondrocytes within the zone of hypertrophy, which may or may not be present in birds, depending on the etiology. It is unclear if the disorganization of chondrocytes in vitamin D-deficiency rickets is due to a primary affect of vitamin D metabolites or a mechanical consequence of the failure of endochondral ossification.[5]

Contributor: Wildlife Conservation Society, Department of Pathology, 2300 Southern Blvd., Bronx, NY
http://wcs.org/

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CASE IV – AFIP 04 Case 1 (AFIP 2936425)

Signalment: Approximately 8-10 weeks of age, intact males, DBA/1LacJ mice (*Mus musculus*) from Jackson Laboratories.

History: Mouse with collagen-induced arthritis as a model for rheumatoid arthritis was used to investigate the therapeutic effect of a drug under development.

Gross Pathology: Swelling and redness of the fore and hindpaw joints with visible phalangeal distortion.

Contributor's Morphologic Diagnosis: Front and rear paw joints (with associated soft tissues): Polyarthritis, suppurative, severe, chronic with pannus formation, articular cartilage degeneration, periosteal bone remodeling and surrounding tissue involvement (bursitis, tendinitis and myositis), DBA/1LacJ mice (*Mus musculus*), rodent.

Contributor's Comment: The tissue section on the slide can be either the front or rear paw of the mouse. Microscopically, all joints (not present in all submitted slides) of the paws are severely disrupted with replacement of the synovium and capsule by severe fibrosis and marked infiltrations of neutrophils and a small number of macrophages and lymphocytes. Across the synovial cavity bridging the synovial capsule, a pannus was usually noted and consisted of a fibrous structure usually poorly vascularized, containing basophilic debris, and infiltrates of neutrophils and a few macrophages. The pannus formation is present with a partial to complete erosion and/or degeneration of the articular cartilage extending in the subchondral bone and occasionally in the underlying bone marrow containing mixed inflammatory cells and slight fibrosis. Along the metaphyseal and diaphyseal bone, a marked periosteal reaction is noted and consists of a marked thickening of the periosteum by a mild infiltration of mostly neutrophils, fibrous connective tissue, bone remodeling (cartilage metaplasia, osteoid matrix and/or woven bone formation with intense osteoclastic activities), and/or sometimes marked osteoblastic hyperplasia (i.e., cells with a large vesicular nucleus and abundant pale cytoplasm). Furthermore, in the surrounding tissues, a marked chronic multifocal suppurative bursitis, tendinitis and myositis are noted.

This submission is a severe case (i.e., grade 4) of arthritis induced by collagen [collagen-induced arthritis (CIA)] in mice as a model for rheumatoid arthritis (RA). The severity of the arthritis was scored on a scale of 0 to 4 by histological assessment as noted in Wooley and Wooley et al.[1,2] CIA is induced in MHC-susceptible mice immunized with bovine type II collagen emulsified in complete Freund's adjuvant. Typically the mice are boosted 3 weeks after immunization with bovine type II collagen in incomplete Freund's adjuvant. The resultant

response is dependent on T cells, B cells, and cytokines. About 4 weeks after immunization, clinical signs of disease develop in the paws. Active inflammation of the paws remains for about 2-3 weeks and usually results in joint destruction and deformity.

In response to joint injury, hypertrophy and hyperplasia of synoviocytes of the synovial membrane, and villous and/or pannus formation are observed. Pannus is an intraarticular fibrovascular structure with inflammatory cell infiltrates that arises from the synovial membrane and spreads over neighboring cartilage subsequent to chronic infectious nonsuppurative synovitis and/or immune-mediated diseases such as RA. With time, as observed in this case, opposing cartilaginous surfaces are united by fibrous tissue resulting in fibrous ankylosis of the joint. The pannus can act as a physical barrier between the synovial fluid and the cartilage preventing chondrocyte nutrition. The pannus macrophages with the proteolytic enzyme-producing neutrophils and the collagenase-producing fibroblasts enhance the cartilage degeneration that commonly extends into adjacent subchondral bone.[3]

CIA in the mouse and adjuvant-induced arthritis (AIA) in the rat are two models used to mimic the clinical manifestations of RA due to similarities in the development of synovial and cartilage lesions. RA is a chronic, erosive polyarthritic disease observed in both humans and dogs, but rarely observed in the latter. The cause of RA is not yet fully understood, but may involve both immune mediated processes i.e., humoral and cell-mediated immunity. IgG or IgM classes known as rheumatoid factors are produced in response to an unknown stimulus. The factors that might be involved are alterations in the stearic configuration of IgG, persistent bacterial cell wall components that cross-react with normal proteoglycans, anticollagen antibodies, and/or defective suppressor T cell activity. Immune complexes formed are ingested by neutrophils that release lysosomal enzymes (e.g., IL-1 promotes secretion of prostaglandins, nitric oxide, and neutral proteases inhibiting proteoglycan synthesis), which are responsible for the inflammatory reaction and destroy intraarticular structures.[3,4] The loss of proteoglycans from cartilage results in alterations in the hydraulic permeability of the cartilage, thus interfering with joint lubrication and leading to further mechanically-induced injury to the cartilage. TNF-α has similar effects to IL-1: increasing concentrations of agents that will decrease matrix synthesis and increase matrix destruction.[3,4] Furthermore, matrix metalloproteinases (gelatinases, collagenases) activated by products of degenerating or reactive chondrocytes and inflammatory cells result in digestion of the matrix.[3]

AFIP Diagnosis: Paw, bones and joints: Polyarthritis and osteomyelitis, chronic-active, diffuse, severe, with articular cartilage erosion, subchondral pannus, cortical resorption, periosteal fibroplasia, reactive bone formation, and extensive soft tissue inflammation, DBA/1LacJ mouse, murine.

Conference Comment: The contributor provides a thorough overview of collagen-induced arthritis (CIA) and its use as an animal model for human rheumatoid arthritis (RA). There is significant variation in slides due to the necessary use of multiple animals and multiple paws. Therefore, as mentioned by the contributor, not all aspects of the described lesions are present on all slides.

Arthritis in the dog is often classified as erosive or nonerosive. Rheumatoid arthritis in the dog is a chronic, erosive polyarthritis that resembles RA in humans. The cause is unknown, but the process is immune-mediated, and as described by the contributor, involves both humoral and cell-mediated immunity, as well as inflammatory mediators and fibroblasts. In dogs, RA is characterized by progressive lameness due to involvement of the peripheral joints of the limbs. Grossly, the lesions consist of marked villous hypertrophy of the synovial membrane, erosion of the articular cartilage, pannus formation, periarticular osteophytes, and occasionally fibrous ankylosis of affected joints.[3]

Nonerosive arthritis occurs in dogs with systemic lupus erythematosus (SLE), or chronic disease processes such as pyometra or otitis externa. In dogs with SLE, in addition to arthritis, these animals often present with anemia, thrombocytopenia, polymyositis, or glomerulonephritis. Immune complexes (type III hypersensitivity) can localize in the synovium and lead to synovitis. In contrast to erosive arthritis, in joints with nonerosive arthritis there is usually minimal villous hypertrophy, no pannus formation, no articular cartilage destruction, and the exudate in the synovial fluid is neutrophilic.[3]

Contributor: Wyeth Research, Department of Pathology, Chazy, New York

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