The Armed Forces Institute of Pathology
Department of Veterinary Pathology

WEDNESDAY SLIDE CONFERENCE 2007-2008

Conference 11

12 December 2007

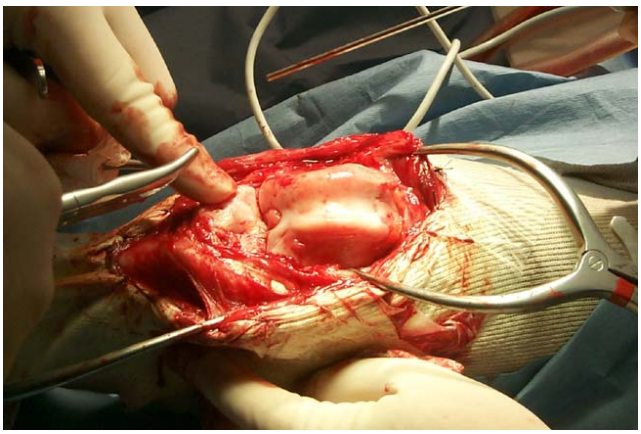
Moderator:

Dr. Steven Weisbrode, DVM, DACVP

CASE I – 306729 (AFIP 2840709).

Signalment: 1-year-old, female, boxer, canine

History: The dog presented to a referral hospital for chronic (greater than one month duration) grade II out of VI lameness on the left hind limb with a short stride and mild muscle atrophy. Physical exam revealed pain on manipulation of the left stifle, moderate left stifle thickening, and mild decreased range of motion. Radiographs revealed a fluffy proliferation in the region of the left stifle fat pad, and thickening with slight mineralization of the medial aspect of the joint. A percutaneous bone core biopsy was performed and was consistent with multifocal osteocartilaginous metaplasia. Due to the dog's age at the time of the initial biopsy (9 months), it was recommended that surgery to remove the lesional tissue be postponed until symphyseal closure occurred. The dog returned for arthrotomy 5 months later. At surgery, moderate **degenerative joint disease (fig. 1-1)** was noted with numerous osteophytes on the medial trochlear ridge. A bony mass was present that incorporated the medial joint capsule from the level of the proximal trochlea to the tibial plateau and from the medial collateral ligament to the patellar tendon. The mass was seen to be contiguous with the patellar fat pad and both the cranial medial and lateral menisci. The joint capsule was removed with the mass (excision was complete grossly).



1-1. Left stifle, boxer. The medial trochlear ridge is moderately proliferative and expanded by osteophytes. Photograph courtesy of Dr. Brian Huss, Vescone (Waltham, MA) and the Angell Memorial Animal Hospital, Pathology Department, 350 S. Huntington Ave., Boston, MA 02130

Gross Pathology: Tissues submitted for histologic evaluation consisted of a 5.5 x 3.5 x 2.0 cm and a 2.0 x 1.5 x 1.2 cm piece of hard, nodular, white-gray tissue lacking orienting anatomic features.

Laboratory Results: Aerobic culture of the joint at the time of initial core biopsy was negative.

Contributor's Morphologic Diagnosis: Synovial osteochondromatosis

Contributor's Comment: The submitted specimen consists of sections of joint capsule that include the synovial membrane and fibrous layer. There is a poorly delineated, expansile, multinodular mass comprised of broad trabeculae of woven and lamellar bone that are lined in many areas by a single layer of osteoblasts. In most areas, these trabeculae arise from foci of collagenous tissue via endochondral ossification. Hematopoietic cells and adipose tissue are present with in the intertrabecular spaces.

Synovial osteochondromatosis, or synovial chondrometaplasia, is a proliferative disorder of undifferentiated stem cells of the synovium. The proposed pathogenesis is the transformation of fibroblast-like cells under the influence of extracellular chondroid matrix material into chrondroblastic cells. It is these transformed cells that are believed to give rise to the characteristic cartilaginous nodules. These nodules may grow and project out form the synovium on delicate vascular pedicles, or as seen in this case, may form a more broad-based nodular mass. When their base is narrow, they often break off and form loose bodies within the joint. The chondrocytes of the loose body are nourished by the synovial fluid and thus will continue to form more cartilage matrix and increase in size. This will often result in degenerative joint disease due to physical damage to the adjacent joint capsule and articular cartilage. If the nodules remain attached to the synovium (as was seen in this case) they will often undergo endochondral ossification with the formation of broad trabeculae of bone. In dogs, the disease is usually seen in medium to large breeds, and has been described in the scapulohumeral, coxofemoral, talocrural, and stifle joints. There is usually no history of trauma or primary degenerative joint disease (such as osteochondritis dissecans).

In humans, osteochondromatosis is uncommon and most often occurs in the stifle joint of middle aged males. The condition in humans has been classified into primary and secondary forms. The primary form is described as the spontaneous formation of intrasynovial nodules in an otherwise normal joint. It is usually confined to one joint, most commonly a larger joint (e.g. knee, hip, shoulder, elbow, and ankle). Histologically, the primary form has been described as foci of chondrometaplasia that contain chondrocytes with cellular atypia. The recurrence rate with this form is considered high. Secondary synovial osteochondromatosis is a similar condition that follows traumatic, degenerative, or inflammatory joint diseases. In this condition, detached fragments of cartilage or subchondral bone become implanted within the synovium and incite the formation of chondrometaplastic nodules. With the secondary form, there is little to no cytological atypia, and the condition usually responds favorably to surgical intervention, provided that the initial joint disease is not allowed to progress.

In the past, an attempt has been made to apply the above-mentioned classification to canine patients with osteochondromatosis, but this has proven to be difficult, as many lesions in the dog do not fit all the criteria of either classification. In the present case, there was no appreciable cellular atypia seen and there was evidence of degenerative joint disease. This would be most consistent with a classification of secondary osteochondromatosis. However, the patient had no history of trauma, and the lesion was confined to one stifle joint. This is more typical of the primary form. A lack of cellular atypia argues for the secondary form, but cellular atypia may not be seen in all primary cases. The presence of degenerative joint disease is not in of itself an adequate criterion for the secondary form, as the formation of osteochondromatous nodules can lead to degenerative changes within the adjacent synovium. Regardless of the classification scheme used, the prognosis for dogs with this condition appears to depend on the degree of degenerative joint disease noted at the time of surgery, as well as the ability to perform total synovectomy with removal of any loose bodies. Recurrence with incomplete removal of the affected synovium and/or loose bodes has been reported. Complete synovectomy may be impossible, however, and temporary relief has been reported with loose body removal alone. The dog in this report was doing well (no lameness reported) 2 months post-surgery.

Differential diagnosis should include severe degenerative joint disease, osteochondral fractures secondary to trauma, osteochondritis dissecans, and neoplasia, particularly chondrosarcoma.

AFIP Diagnosis: Joint capsule (per contributor): Osteochondral metaplasia (osteochondromatosis), diffuse, marked, Boxer (*Canis familiaris*), canine.

Conference Comment: The contributor gives a good review on a poorly characterized, rare condition in animals. We essentially agree with the contributor's diagnosis of osteochondromatosis, although we prefer the terminology of osteochondral metaplasia to describe the morphologic lesion.

Osteochondral metaplasia can occur within any synovial lined structure, such as a joint, tendon sheath, or bursa.[4]

Ectopic ossification of these structures requires a vascular supply, and the presence of detached osseous bodies (joint mice) implies a previous attachment to the synovial surface.

The underlying cause of osteochondral metaplasia in animals is not known. Due to the relatively limited responses of the joint, it can often be difficult to distinguish osteochondral metaplasia from other disease processes that result in intra-articular joint bodies such as osteochondrosis or chip fractures.[4]

There is some variability in the slides. Most slides exhibit lamellar bone formation, and only a few slides exhibiting both lamellar and chondroid bone formation. Lamellar bone is formed by endochondral ossification with a sharp line of demarcation between cartilage and bone.[5] Chondroid bone is formed directly from fibrocartilage, with blending of the cartilage and bone.

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CASE II – 07-1271 (AFIP 3066003).

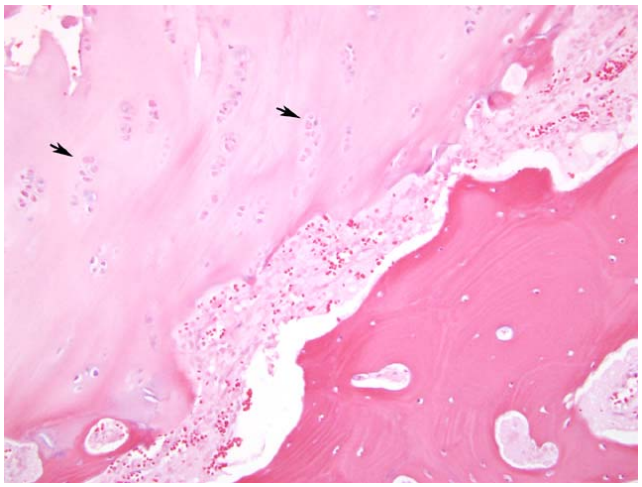
Signalment: Two-year-old, male castrate, Main Coon cat.

History: Femoral neck fracture. Femoral head osteotomy performed. Radiographically the femoral neck appeared less dense and slightly lytic?

Histopathologic Description: The femoral head has a normal shape and the articular cartilage is microscopically normal. There is variable lack of differential staining in marrow, bone lining cells and osteocytes interpreted to be artifacts of decalcification. The marrow is mostly fatty with limited hematopoiesis and areas of acute hemorrhage. At the deep specimen margin there is bone debris within the marrow spaces that is presumed to reflect method of surgical removal since no sawing was done to the specimen once received. There are shredded fragments of hyaline cartilage present on the deep margin in what would be the location of the growth plate. Compared with a normal active growth plate, the cartilage is hypocellular and chondrocytes are present in **irregular groups (fig. 2-1)** rather than columns.

Contributor's Morphologic Diagnosis: Femoral head: Dysplasia and fracture of physis

Contributor's Comment: This particular case of atraumatic fracture of the femoral capital epiphysis (in a cat over one year of age) was selected to submit to the Wednesday Slide Conference because, in our experience, it is typical of the appearance of such specimens. While the most important lesions are in the growth plate, little growth plate is present on many of these specimens received for histopathologic evaluation. Only fragments of the physis remain on the femoral head and these fragments have variable hypocellularity and disorganization. The remainder of the femoral head has no significant lesions. Subtle marrow or bone lining-cell changes are not possible to detect in this specimen due to over-decalcification. Most important in considering the diagnosis of physeal dysplasia at this site in the cat is the PRESENCE of a growth plate relative to the age of the cat. The microscopic appearance of the plate might actually be non-specific and reflect that although the plate has not closed, it is not contributing to longitudinal growth. On average, the growth plate at the femoral neck in cats closes at 40 weeks.[10] Castration delays this by about 6 weeks but this delay is NOT associated with increased length of the bone (reported for the radius).[11] Therefore, although it is open longer, the growth plate is not significantly adding to longitudinal growth. The age of presentation of cats with physeal fractures appears to have changed from earlier to more recent literature. Reports of physeal fractures of the femoral neck in cats in 1993 and 1996 have no cat older than 12 months of age affected.[3,9] Publications in 2001[2], 2002[6], 2004[5] and 2006[8] report femoral capital physeal fractures mostly in cats older than 12 months (one paper restricted itself to cats over 12 months of age)[6] and one cat in these reports was 4 years



2-1. Femoral head, Maine Coon cat. Multifocally, surrounded by abundant cartilaginous matrix are few irregular clusters of chondrocytes (arrows). (H&E 200X)

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old and still had several open physes.[8] Most of these cats are castrated over-weight males and have other growth plates open well beyond the age expected for normal closure. One study reported the contralateral physis open in 13 of 18 cats with slipped physes of the femoral head.[6] Many of these cases of fracture of the physis of the femoral head in cats older than 12 months appear not to be associated with trauma. This is similar to the condition in the pig for the physis of the femoral head which is considered a form of osteochondrosis (epiphysiolysis).[4] In the pig however, the lesions develop mostly between ages 6-18 months. At 18 months, skeletal maturity is reported to be reached.[4]

The classification of this lesion as a dysplasia in cats, appears to be appropriate but the disorganization and hypocellarity seem more likely to be secondary to failure to properly close than a primary chondrodysplasia of the growth plate. The conclusion that the growth plates WERE normal during growth is supported by the fact that the cats appear to have reached normal skeletal growth with normal appearing skeletons within normal time. Since the signals for longitudinal growth have apparently appropriately ceased in these cats but the signals for closure have either not been recognized or sent, it is understandable that the chondrocytes remaining in the non-functional growth plate would not have their normal arrangement and density. Likely the persistence of the plate and not its abnormal arrangement and density of chondrocytes is predisposing it to slip with minimal trauma in these heavy fully grown cats.

AFIP Diagnosis: Femoral head: Dysplasia and fracture of physis, Maine Coon (*felis domesticus*), feline.

Conference Comment: Feline physeal dysplasia is characterized by the observation of irregular clusters of chondrocytes that are separated by abundant matrix on both the epiphyseal and the metaphyseal side of the physeal cartilage cleavage site.[1,2] This is in contrast to a traumatic fracture, in which the chondrocytes retain their linear arrangement on both sides of the fracture site.[2]

Although the underlying cause of feline physeal dysplasia is not known, it has been associated with various factors including genetics, nutrition, obesity, endocrine imbalances, and other factors.[1] Due to causing a delay in physeal closure, neutering has been previously considered associated with feline physeal dysplasia[6], although in more recent literature this observation has been challenged.[8] It is not known if the association with obesity is due to the increased stresses placed on the physis from the additional weight, causing failure under conditions of minimal trauma, or if there is an underlying endocrine abnormality that results in both obesity as well as a weakened physis.[2]

Epiphysiolysis in pigs is a manifestation of osteochondrosis, which has many clinical manifestations. The growth plate in affected pigs is usually characterized by a focal failure of endochondral ossification in which retained cartilage extends into the metaphysis.[2,12] The chondrocytes of the cartilage core usually maintain their normal alignment. This differs from feline physeal dysplasia where the entire physis is usually affected, and the physis consists of irregular clusters of chondrocytes that have lost their normal alignment.[2]

The Salter-Harris classification system has been used to classify fractures of the growth plate in animals. In this system, fractures are divided into five types based on their location **(table 2-1).**

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http://vet.osu.edu/biosciences.htm

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Table 2-1. Salter-Harris classification system[13]

Type 1	Fracture through the physis without involvement of the epiphysis or metaphysis
Type 2	Fracture involving the metaphysis and extending into the physis
Type 3	Fracture involving the epiphysis and extending into the physis
Type 4	Fracture involving the epiphysis and metaphysis going through the physis
Type 5	Compressive fracture of the physis, crushing the growth plate

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CASE III – 335-07 (AFIP 3074807).

Signalment: Five (four male, one female) young guinea pigs, *Cavia porcellus*

History: Five young guinea pigs from a pet shop developed fever, lethargy, joint swelling and reluctance to move over a period of a few weeks. Their diet had been a pelleted food formulated by the shop owner. Comparable signs had been observed in other guinea pigs in the colony in the preceding summer but in the intervening period all guinea pigs had appeared healthy.

Gross Pathology: Four male and one female guinea pigs were necropsied. All had subcutaneous and intramuscular hemorrhage involving the proximal hindlimbs and hemarthrosis of the stifle joints. In some animals, there was also hemorrhage into the elbow, carpal and tarsal joints. All other organs were grossly normal.

Histopathologic Description: There was both loose and organizing fibrin within the stifle joint and the synovium contained acute hemorrhage and edema, dark brown pigment consistent with hemosiderin, and moderate fibrosis. There was prominent synovial hyperplasia. The periosteum was lifted from the bone by edema and hemorrhage and there was hemosiderin present in the edematous tissue. The perimysium and fascial tissues also contain hemorrhage and severe **edema (fig. 3-1)** and a moderate degree of fibrosis. There was hemorrhage separating myofibers, and degeneration of myofibers.

There were no identifiable osteoclasts on the periosteal surface of the diaphysis. The endosteal surface hosted an adequate population of osteoblasts. There was replacement of the terminal plate and cancellous bone of the epiphysis with loose connective tissue containing residual osteoclasts. The growth plate cartilage was disorganized and lacked normal chondrocyte columns. In the primary **spongiosa (fig. 3-2)**, there was hemorrhage and necrosis,

and predominance of spicules that were solely cartilage with no bony transformation. Active resorption of mineralized tissue was occurring and there were fragments of normal spongiosa present, surrounded by loose connective tissue, probably remnants of trabecular fractures. In the tibial metaphysis, there was a well developed scorbutic lattice.

The secondary spongiosa were normal.

Contributor's Morphologic Diagnoses: 1. Dysplasia of the proximal tibial growth plate and primary spongiosa with trabecular fractures, hemorrhage, necrosis, failure of ossification and development of a scorbutic lattice

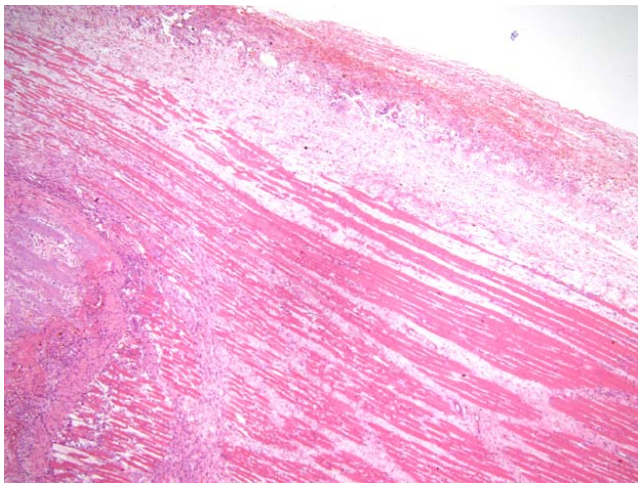
2. Chronic active hemarthrosis of the stifle joint, with severe chronic active intra-periosteal, intramuscular and periarticular edema and hemorrhage

Etiological diagnosis: Chronic vitamin C deficiency (scurvy)

Contributor's Comment: Vitamin C deficiency, known as scurvy, is an ancient disease showing a modern recurrence. It is traditionally associated with sailors and long sea voyages, and was responsible for more deaths at sea in the 15th to 18th centuries than all other causes combined, accounting for up to 80% of the occupants of a ship on a long journey. It was not until the 20th century that a link was made between lack of fresh vegetables in the diet and the onset of "land scurvy".

Vitamin C (ascorbic acid, ascorbate) is water soluble and degraded by heat, ultraviolet radiation or free radical oxidation. Synthesis is widespread in nature, including micro-organisms and fungi. In those vertebrates which synthesize the vitamin, the site(s) of production is in the liver and/or kidneys. Fruit eating bats, red vented bulbul birds, guinea pigs, human and non human primates, most fish and insects lack gulonolactone oxidase to catalyze the last step of the synthesis, and require dietary intake as there is no storage within the body. Absorption occurs in the ileum via active transport.

The earliest signs of vitamin C deficiency are generally non specific, such as weakness, anorexia and weight loss. Guinea pigs fed on severely scorbutic diets will voluntarily decrease their intake and begin to lose weight after 2 weeks. After 3 weeks, serum $25OHD_3$, calcium and albumin levels are significantly reduced, bone mineral density and bone content are significantly lower than normal, and bone volume is reduced in long bones, with fewer and thinner trabeculae and a thinner growth plate. There is also bone loss with osteonecrosis, osteopenia and cortical thinning with periosteal proliferation.



3-1. Bone (tibia and femur), guinea pig. Multifocally, the periosteum, adjacent muscle and fascial tissues are expanded and separated by hemorrhage, edema and fibrosis. (H&E 40X)

The first histological signs in bone are flattening of osteoblasts and failure to lay down matrix. A lattice of vascularized, calcified cartilage is formed in the metaphysis and is not replaced by bone as it increases in thickness; vitamin C is required for the differentiation of osteoblasts from progenitors. Being relatively unresistant to mechanical forces, this "scorbutic lattice" develops numerous microfractures. Blood vessels of all bone regions dilate, with those of the metaphysis being particularly prominent. Active growth zones are severely hyperemic and microhemorrhages are common. Intercellular junctions between endothelial cells are wider in scorbutic vessels than those of normal animals.

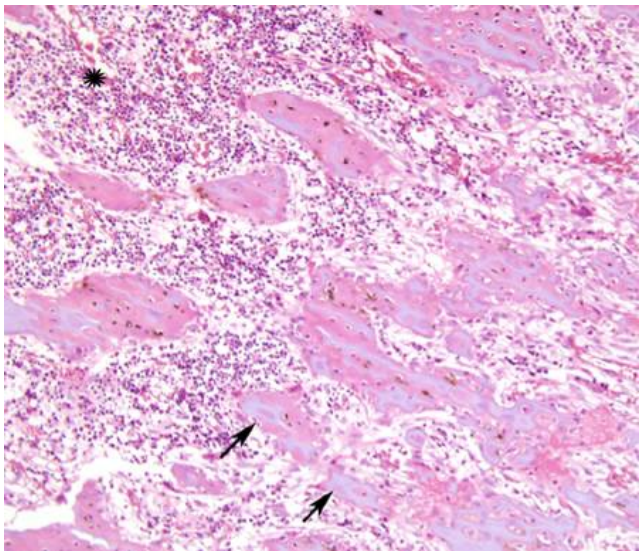
With time, hematopoietic tissue of the marrow is replaced by immature collagen-poor mesenchyme. In moderately advanced disease, vascularity of the bone is not a prominent feature but vascular fragility is increased. Chondrocyte columns of the growth plate become distorted and shortened as the disease progresses, the number of chondrocytes decreases and the growth plate becomes thin and uneven. Proliferation of spindle cells between the fibrous periosteum and the cortical bone surface thickens the periosteum of long bones, and metaphyseal infarction may occur.

Arthralgia and myalgia develop; eventually bleeding occurs into the joints due to damage to synovial vessels and microfracture of bone. Hemorrhage into stifle joints is among the most obvious signs of scurvy in guinea pigs.

There may also be non-hemorrhagic joint effusions. Hair loss and corkscrew hairs are reported; Vitamin C is important in the disulfide bonding of hair. Also reported are follicular hyperkeratosis, and pigmented ichthyosis, fibrosis of dental pulp, diarrhea and reproductive failure. Wound healing is delayed and incomplete. Conjunctival and intraocular bleeding are also common, and there is disruption of corneal epithelial and stromal organization with stromal vascularization in later stages.

Laboratory findings are non specific. Anemia is due less to bleeding than to the concomitant iron and folate deficiencies. Foods high in Vitamin C are also sources of folate and the deficiencies often coexist. Furthermore, Vitamin C increases the absorption of nonheme iron by reducing ferric iron in the stomach and enhances the amount of iron stored in ferritin. Guinea pigs with lowered levels of vitamin C but normal growth (phase 1 of scurvy) may have serum iron levels decreased to 50% of normal, and by the time clinical scurvy (phase 2) is evident, levels may be as low as 10 – 15% of normal. Intravascular hemolysis may also lower red cell counts. Leukopenia and hypoalbuminemia, a marker of malnutrition, are also common.

Response to treatment is rapid with most clinical signs reversed within a week of onset of adequate intake. In severely scorbutic guinea pigs, complete restoration of normal trabecular structure takes about 20 to 25 days.



3-2. Bone (tibia and femur), guinea pig. Multifocally within the primary spongiosa, there is retention of cartilaginous cores with a lack of ossification (arrows). Additionally, the marrow cavity is expanded by necrotic debris and hemorrhage (star). (H&E 100X)

Vitamin C is a major antioxidant, in co-operation with vitamin E and glutathione, and glutathione adminstration can delay the onset of clinical scurvy in guinea pigs. Persistant deficiency leads to hepatocyte apoptosis through endoplasmic reticulum stress as a result of its participation in oxidative protein folding. Oxidative injury in the absence of adequate vitamin has also been linked to motor neuron disease, demyelination of pyramidal tracts and consequent muscular atrophy.

Vitamin C is also an electron donor. It interacts with proline oxidase and lysine oxidase in the hydroxylation of procollagen, in two hydroxylation steps in the production of carnitine, which promotes transport of long chain fatty acids into mitochondria and assists the flux of substrates into the TCA cycle, and with β-monoxygenase in the conversion of dopamine to noradrenaline.

The interactions leading to scurvy are not entirely understood. Collagen related signs were thought to be due to failure of hydroxylation of pro-collagen proline and lysine with consequent failure of cross-linking, leading to fibril instability. However, starved animals with vitamin C supplementation show the same reduction in collagen production as scorbutic animals as a result of inhibition of insulin-like growth factor (IGF) by binding protein (IGFBP) induction, and deficiency of collagen type IV and elastin leads to defects in blood vessels with consequent hemorrhage in both scorbutic and vitamin supplemented starved animals. Similar effects of IGFBP on collagen are seen in bone, but the decreases in alkaline phosphatase activity are independent of the fasting effect. In cartilage, type II collagen production drops in the early stages of deficiency, but stabilizes to around 50% of normal.

The poor wound healing of scorbutic animals, however, is not due to inhibition of proline hydroxylation or induction of IGFBPs. Vitamin C is known to promote wound healing following irradiation through stimulation of collagen synthesis and deposition, and increased fibroblast density and tissue vascularity. Poor wound healing in scurvy may therefore be a consequence of failure of interstitial procollagen gene expression and blood vessel formation.

AFIP Diagnosis: Bone, tibia and femur: Osteochondrodysplasia, scorbutic, with lack of normal primary spongiosa, osteopenia, microfractures, and subperiosteal hemorrhage, guinea pig (*Cavia poricellus*), rodent.

Conference Comment: The contributor gives an excellent overview of Vitamin C/Ascorbic acid deficiency in

general and in the guinea pig in particular. Ascorbic acid is an important antioxidant and reducing agent. It is required for the hydroxylation of proline and lysine, a process that is essential in the formation of collagen.[5] Functional failure of these enzymes results in formation of collagen fibrils that are not cross-linked and lack tensile strength, leading to blood vessel fragility and poor wound healing.[5] Lesions associated with scurvy, such as subperiosteal, subcutaneous, intramuscular and gingival hemorrhages, reflect this defect in collagen synthesis.

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CASE IV - H06-0308C (AFIP 3025649).

Signalment: 2-day-old, Landrace piglet, male, *Sus scrofa*, porcine

History: At a piggery with 500 sows a farm worker noted that there were one or two piglets in rare litters over the past few weeks that had bilateral thickened forelimbs. The piglets had a stilted forelimb gait. The sows were multiparous and had not had litters previously with this condition. The piglets did not survive more than a few days after birth.

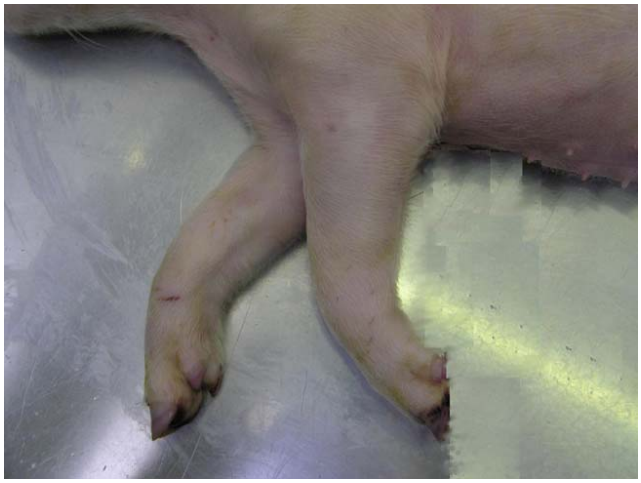
Gross Pathologic Findings: One piglet was submitted for post mortem examination. The forelimbs were bilaterally **thickened (fig. 4-1)**. On dissection the radius and ulna of both limbs were thickened uniformly through the diaphysis. There were no other significant post mortem findings.

Histopathologic Description: Ulna and Radius, diaphysis: There is a cross section of radius, ulna and attached fibrous tissue and skeletal muscle. The cortex of both bones is distended by interconnecting trabeculae of woven bone radiating from the periosteum at right angles to the existing cortical lamellar bone. Within the interstices of the new bone is myxodematous tissue without haemopoietic cells. The periosteum is irregularly expanded by polygonal to plump spindle cells set in eosinophilic fibrillar stroma extending from mature uniform fibrous stroma. The central cavity of the bone consists of multiple aggregates of haemopoietic cells set between trabeculae of bone. Within the connective tissue surrounding the bones there is separation of the adventitia of small arterioles from the attached collagen by proteinaceous material consistent with oedema.

Contributor's Morphologic Diagnosis: Ulna and radius: Hyperostosis

Contributor's Comment: Hyperostosis is a rare recessive autosomal disease seen in newborn piglets.[5] Most commonly these piglets die due to malnutrition, starvation or cardiac insufficiency.[5] The condition has been reported in Landrace and Duroc pigs.[1,3] Generally hy-

4-1. Landrace piglet. The forelimbs are diffusely thickened. Photograph courtesy of the Department of Veterinary Biology and Biomedical Sciences, School of Veterinary and Biomedical Sciences, Murdoch University, South St, Murdoch WA 6150, Australia



perostosis affects the forelimbs, most commonly the radius and ulna.[3]

The radioulnar region is thickened and may be twice normal diameter. The overlying skin is hyperaemic. At necropsy there is enlargement of the soft periosseous tissues mostly at the cranial surface of the limbs from proximal end of the radius down to and surrounding the metacarpal bone distally. Fibrous tissue may invade and partially fuse with muscles.[3]

Microscopically there are radiating osseous spicules associated with marked hyperplasia of periosteal osteoblasts. They are numerous, large and can appear as syncytium. Normal cortical bone is lamellar and forms concentric osseous plates consistent with rapid periosteal apposition. Osseous trabeculae of woven bone are orientated radially in relation to the medullary cavity.[3]

The pathogenesis of congenital hyperostosis is not known, however there are two suggested mechanisms reported in the piglet. These include 1) disruption of the growth of bone at the ossification groove of Ranvier or 2) local circulatory abnormality.

Dalton et al[3] proposed that radioulnar hyperostosis may be the result of an initial lesion situated at the anchor site of the periosteum to the epiphysis at the level of the perichondrial ossification groove of Ranvier. This true separation could be the cause of the fine supernumerary trabeculae of woven bone. The groove of Ranvier is an ossification groove (a component of the perichondrial ring supporting the zone of provisional calcification) that supplies chondrocytes to the physis for diametric growth of the foetal bone and also fibrous attach of the periosteum to the epiphysis.

A second study of piglets with hyperostosis, conducted by Roels et al[4] demonstrated distinct circular constrictions in the proximal antebrachial region of the median artery, in conjunction with the consistent finding of oedema within the connective tissue surrounding the thickened bone suggesting hypertension. In affected piglets the initial segment of the median artery (ie proximal antebrachial region) showed distinct circular constrictions (not seen in controls) suggesting acute hypertension. There was extensive smooth muscle fibre proliferation, intimal fibrosis and fibrinoid necrosis of tunica media and less narrowing of lumen in small arteries and arterioles of upper dermis.

AFIP Diagnosis: Bone, radius and ulna: Hyperostosis, periosteal, circumferential, severe, Landrace (*Sus scrofa*), porcine.

Conference Comment: Although not proven, hyperostosis is presumed to be an autosomal recessive inherited disease that has been described in Landrace swine.[1,3,5] The characteristic histology lesion associated with hyperostosis is proliferation of subperiosteal, radiating trabeculae of woven bone extending from the surface of apparently normal cortical bone, covered by a thickened periosteum.[5]

A similar hyperostotic condition has been reported in a single West Highland White Terrier dog, in which new bone formation involved the pelvis, scapulae, humeri, ulnae, femora, radii, and tibiae.[5]

The deeper, older portion of the examined cortex is formed of woven bone. This type of bone is formed in areas in which a support structure needs to be put in place quickly, such as in the developing fetus or at sites of fracture repair, inflammation, or neoplasia. It consists of collagen fibers within the bone matrix that are arranged in a haphazard interwoven fashion.[5] These haphazard arrangements are usually replaced with the more structurally sound lamellar bone during skeletal maturation.[5] In young rapidly growing animals, especially ruminants, a different type of lamellar bone is deposited along the surfaces of long bones. This type of bone is called laminar bone and consists of lamin ar arrays rather than the Haversian system seen more commonly in lamellar bone.[5]

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