The Armed Forces Institute of Pathology
Department of Veterinary Pathology

WEDNESDAY SLIDE CONFERENCE 2007-2008

Conference 16

6 February 2008

Moderator:

Dr. Fabio Del Piero, DVM, DACVP

CASE I – 05-7666 (AFIP 3026263).

Signalment: 3.5-year-old cow

History: Animal with a suppurative osteomyelitis of the right mandible

Gross Pathology: In the right mandible, there was a hard mass 12 cm in diameter with ulceration of the adjacent gum. The mass was composed of many confluent fibrous nodules and several suppurative tracts. In the fibrous nodules, there were several cavities 1 mm to 1 cm in diameter containing variable amounts of a yellowish pus with many sulfur granules.

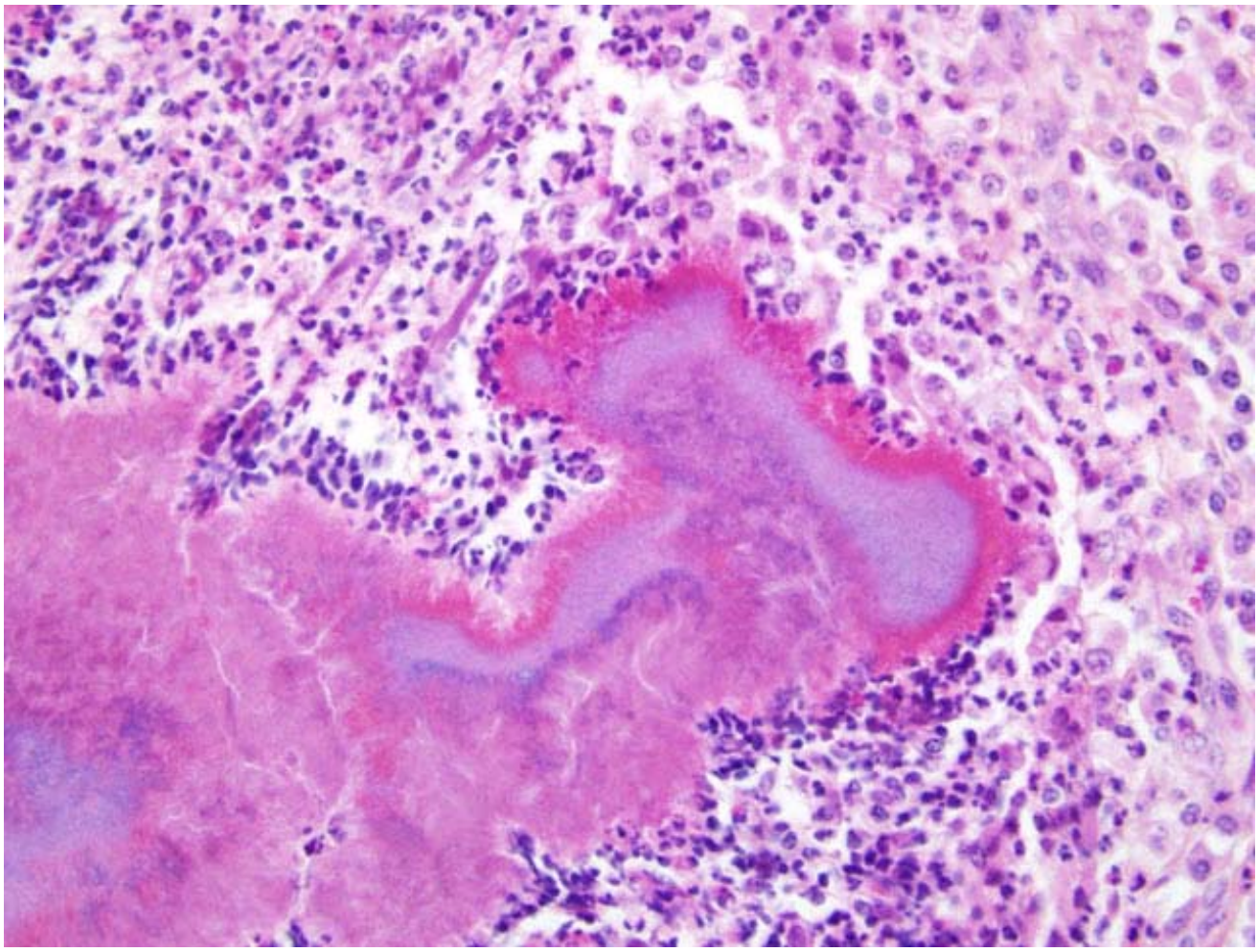
Histopathologic Description: Most of the mandibular bone is replaced by a granulation tissue infiltrated by macrophages and plasma cells. The granulation tissue is surrounding many small abscesses with granules composed of large bacterial colonies (Gram positive rod or coccoid-shaped and branching filamentous organisms) surrounded by radiating eosinophilic clubs (Splendore-Hoeppli material) **(Fig. 1-1).** There is a zone of neutrophils around the granules, surrounded by many large macrophages and plasma cells.

Contributor's Morphologic Diagnosis: Chronic pyogranulomatous mandibular osteomyelitis, with large colonies of Gram-positive filamentous organisms.

Contributor's Comment: This pyogranulomatous mandibular osteomyelitis with the presence of colonies of Gram-positive branching filamentous organisms forming sulfur granules is a good example of mandibular actinomycosis in cattle caused by *Actinomyces Bovis*.[1,2] The osteomyelitis would result from an extension of the infection of the gums or peridontium by the bacteria, following injury by foreign bodies or as a complication of periodontitis.[1] The excessive periosteal proliferation and the granulation tissue induced by the chronic inflammatory process, can cause a marked enlargement of the affected mandible (lumpy jaw).

AFIP Diagnosis: Bone; skeletal muscle; fibrous connective tissue, right mandible (per contributor): Pyogranulomas, multifocal to coalescing, with Splendore-Hoeppli material and colonies of Gram-positive filamentous bacteria, cow (*Bos taurus*), bovine.

Conference Comment: Actinomycetes are Gram-positive, non-acid-fast, branching filamentous rods. They are facultative anaerobes and normal inhabitants of the oral mucous membranes, tooth surfaces, and gastrointestinal tract.[1,2] Actinomycosis, or lumpy jaw, is primarily a disease of cattle, although it has been reported in



1-1. Mandible, bovine. Large bacterial colonies admixed with brightly eosinophilic Splendore-Hoeppli material. (H&E 400X).

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horses, pigs, deer, sheep and dogs.[2] Infections usually are restricted to the bone of the mandible resulting in a chronic suppurative and fibrosing osteomyelitis, although infections have been reported to involve the maxilla, regional lymph nodes or tongue.[2,3]

Infection usually occurs secondary to trauma with subsequent extension into the periosteum.[2] The normal architecture of the mandible is progressively destroyed inciting an extensive proliferative periosteal reaction.[2] The purulent exudate may contain necrotic trabecular bone (bone sand), or soft yellow granules containing mats of tangled, filamentous bacteria and Splendore-Hoeppli material (sulfur granules).[2]

Residents at AFIP utilize the mnemonic "YACS" to develop a differential diagnosis when large colonies of bacteria are present in hematoxylin and eosin stained sections.

YACS stands for:
Y *Yersinia sp.*
A *Actinomyces sp., Actinobacillus sp. Arcanobacter sp.*
C *Corynebacterium sp.*
S *Staphylococcus sp., Streptococcus sp.*

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CASE II – 06L-0264 (AFIP 3028612).

Signalment: 2- 3 years, female, pony, equine, *Equus caballus*

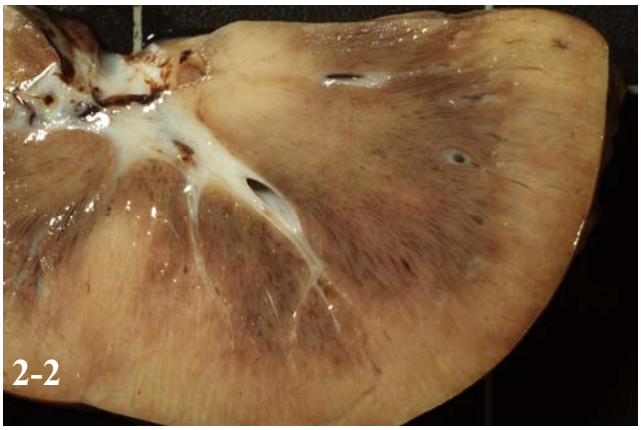
History: The case was part of an investigation in an animal cruelty case. The pony was cachectic and deteriorated. Due to poor body condition, severe elevated levels of urea and creatinine (azotaemia), the horse was euthanased. Multiple formalin-fixed fragments of right and left kidney were submitted for histopathology.

Gross Pathology: Kidney moderately firm, with prominent, pale beige glomerula, mild diffuse hyperaemia, moderate diffuse cortical and medullary interstitial fibrosis and focally indistinct cortico-medullary transition zone **(Fig. 2-1, 2-2).**

Histopathologic Description: The renal architecture is preserved. Within the epithelium of proximal and distal tubular epithelia of the cortical and medullary zone, numerous intracytoplasmic apicomplexan coccidian trophozoites and sporoblasts are seen. Few tubular epithelia are desquamated. Multifocal, mild to moderate tubular epithelial necrosis, intratubular, Von Kossa stain-positive mineralisation (calcification), and Von Kossa stain-negative, birefringent crystals (oxalate) are seen. Multifocally, distal tubules contain moderate to large amounts of cellular debris (desquamated, necrotic epithelia?) and show mild dilation. There is focal mild glomerular fibrosis with glomerular synechia formation and multifocal mild to focally moderate interstitial fibrosis. A focal mild interstitial infiltration by lymphocytes, plasma cells and small numbers of eosinophils is seen.

Contributor's Morphologic Diagnosis: Glomerular and interstitial fibrosis, tubulonecrosis with (dystrophic) mineralisation, oxalate crystal formation, mixed cellular interstitial nephritis; diffuse, moderate (end-stage renal disease); with intracellular sporogenic and gametogenic stages of apicomplexan coccidian parasites, consistent with *Klossiella equi*, (sporozoa, apicomplexa, coccidia) infection, kidney, horse, *E. caballus*.

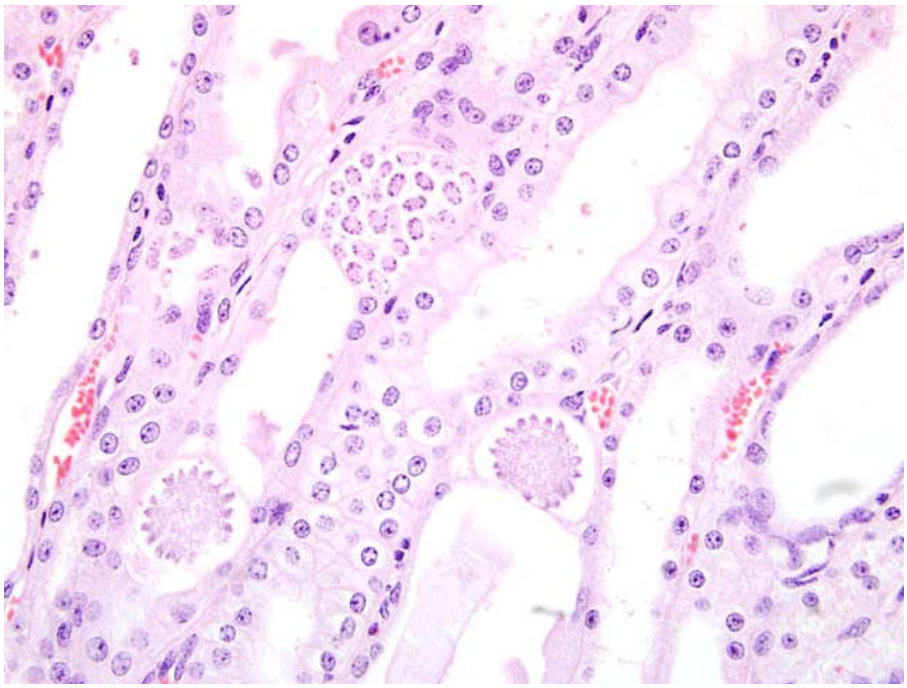
Contributor's Comment: The parasitic genus *Klossiella* belongs to the subphylum sporozoa which is characterised by intracellular life-cycle and an apical complex at some point during its development. The trophozoites have no cilia or flagella. The reproduction



2-1. Kidney, equine. Moderate diffuse cortical and medullary interstitial fibrosis and focally indistinct cortico-medullary transition zone.
2-2. Kidney, equine. Higher magnification.

Gross photographs courtesy of Department of Veterinary Pathology, Faculty of Veterinary Science, University of Liverpool, Crown Street, Liverpool L6 9 7ZJ, United Kingdom
http://pcwww.liv.ac.uk/vets

involves both asexual (schizogony) and sexual (gametogony) phases. Following gametogony a zygote is formed which divides to produce spores (sporogony). Klossiellidae can be found in the kidneys of equids, mice, guinea pigs, bats, opossums, and snakes.[2,7] Renal infections with *K. equi* are often clinically inapparent. Mild infections, usually self-limiting, are followed by a predetermined cycle of development, with the production of infective stages that are shed in the host urine, and are thought to be ingested by another host. There are few reports on *K. equi*-related nephritis in severely infected equids.[1,3] Both sporogenic and gametogenic stages of *Klossiella equi* were identified in the kidneys. All stages developed in individual tubular epithelia cells. Schizonts were seen mostly in the proximal convoluted tubules, but also free within the lumen of a tubule. Macrogametocytes and microgametocytes were present in syzygy in the loop of Henle and collecting ducts. Sporont and budding sporont stages were also seen in the loop of Henle. All stages of development of sporoblasts were observed protruding into the tubular lumen. Sporocysts were identified rupturing out of the sporoblast membrane into the lumen of tubules. Renal tubules were greatly dilated and contained cellular debris. The tubulonecrosis and desquamation of tubular epithelia in this case most likely can be ascribed to the infection by *K. equi*. Whether the additionally described chronic renal alterations are due to the parasitic infection or are a separate underlying pathomechanism, cannot be stated.



2-3. Kidney, equine. Klossiella equi sporonts with radiating sporoblasts and mature sporocyst containing sporoblasts. (H&E 400X).

AFIP Diagnosis: 1. Kidney, tubules: Degeneration and necrosis, multifocal, moderate, with cellular casts and protozoa **(Fig. 2- 3),** etiology consistent with *Klossiella equi*, pony (*Equus caballus*), equine.
2. Kidney: Nephritis, interstitial, lymphoplasmacytic, multifocal, moderate, with intratubular crystals.

Conference Comment: The contributor gives an excellent description of *Klossiella equi*. *Klossiella equi* is the only known coccidian parasite of the equine urinary tract, with various stages of development located in the kidney.[3] The life cycle is not currently known, although infection is presumed to occur via ingestion of infective sporocysts that were shed in the urine.[6] It is also believed that one schizont generation develops in the glomerular endothelium and another in the proximal tubular epithelium, with sporogony occurring in the epithelium of the thick limb of Henle's loop.[4] Infection is thought to be an incidental finding although it has been associated with nephrosis and nephritis in immune-compromised individuals.[1,6] We agree with the contributor that it cannot be determined if the interstitial nephritis and crystals are the result of the *K. equi* infection.

Ultrastructurally, developing sporoblasts are encased by a bilaminated cell membrane composed of an overlying thin granular layer, and an underlying dense inner layer.[1]

We thank Dr. C. H. Gardiner, PhD, veterinary parasitology consultant to the AFIP, for his review of this case.

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CASE III – R06-120 (AFIP 3027084).

Signalment: Two-month-old, Duroc-Hampshire crossbred, barrow, hog (*Sus scrofa domestica*)

History: The animal presented and culled for necropsy was from a farrow to finish swine herd in central Taiwan. Piglets were born healthy and began developing skin lesions and weight loss 6-7 days prior to necropsy. Four piglets from a batch of 60 piglets in the nursery pen were affected. Two piglets were found dead. Recent management changes included new farrowing and nursery houses and pens for batch production.

Gross Pathology: The piglet was in fair body condition. The integument of the piglet had multiple 1 to 3cm diameter, slightly raised, crusted skin lesions affecting all parts of the body. Lesions had dark-brown friable surfaces. There were multiple, black, raised, smooth, shiny nodules disseminated throughout the thoracic viscera, affecting the lungs and myocardium. Some dark, melanin like lesions were noted on the liver. All other organs were normal.

Laboratory Results: Specimens of skin, lung and liver were submitted for aerobic bacterial culture. No bacterial organism was cultured from these organs.

Histopathologic Description: Microscopically, the skin mass is well demarcated and nonencapsulated. The intact epidermis of the skin section exhibits mild epidermal hyperkeratosis while a large coalescing zone of superficial epidermal necrosis with inflammatory infiltration of neutrophils and histiocytes is observed in the affected skin. The skin mass is focally expanding the subcutaneous fat, compressing the underlying subcutaneous tissue and elevating the overlying dermis. The mass is composed of closely packeted large, polygonal to spindle-shaped cells arranged in sheets and short bundles contained within a scant intervening fibrous stroma. Most tumor cells (melanocytes) contain variable amount of brown to black intracytoplasmic pigment. The pigmentation varies from fine dusting to large quantities of granular to coarse material. Some of the spindle cells are less pigmented. Nuclei vary considerably in size; many nuclei are large, round to oval. Most nuclei contain one or rarely two large and round nucleoli. Cells along the superficial margins of the dermis abut on and occasionally surround the bulbs of hair follicles. The morphology of the tumor cells (melanocytes) is applicable to the metastatic focus in myocardium. Some myocardial tissue is destroyed and replaced by the growing metastases.

Contributor's Morphologic Diagnoses:
Skin: Melanoma and moderate subacute necrotizing epidermatitis, Duroc-Hampshire cross, swine.
Myocardium: Melanoma, metastatic.

Contributor's Comment: Melanomas have been reported in a variety of domestic and wild animals.[4,7,8] It is a devastating disease frequently encountered with both veterinary and human medicine. The Sinclair miniature and Duroc breeds have a genetic predisposition for melanomas; in addition, the Sinclair miniature pig has served as a model for spontaneous cutaneous melanoma in humans. Melanomas occur as congenital lesions and sporadically in all ages of Duroc-Jersey, Hormel, Sinclair and their crossbreeds, whereas these tumors are rare in other swine breeds. Regression of such tumors are common and in some breeds may occur *in utero* and at various times after birth.[1] Other tumors arising from the skin may look clinically very similar to melanoma. These include melanocytoma, dermal hemangioma and hemangiosarcoma as well as pigmented lesions of the epidermis and adnexa.[2,4]

Specific immunohistochemistry (IHC) to identify melanocytic tumors of swine is needed. In a recent study, normal and neoplastic porcine melanocytes were vimentin positive, cyto keratin negative, S-100 positive and alpha-1-antitrypsin (AIAT) negative, similar to the immunophenocyte reported for human normal and neo-

plastic melanocytes.[6]

AFIP Diagnosis: 1. Haired skin and subcutis: Melanoma, Duroc-Hampshire crossbreed (*Sus scrofa domestica*), swine.
2. Heart: Melanoma, metastatic.

Conference Comment: The contributor provides a complete, concise description of melanomas in pigs. In dogs, 56% of melanomas develop in the oral cavity.[3,5] It is also the second most common subungual neoplasm.[3,7] Known as the "great imitator", melanoma may appear with or without melanin granules; in an interwoven, whorled, or nested pattern; with round, polygonal, and /or spindled cells; or any combination of these types.[3,7] Malignancy of canine cutaneous melanocytic neoplasms is often determined by number of mitoses (>3/10 HPF).[3,7] Melanocytic neoplasms involving the oral cavity, subungual region, and mucocutaneous junctions are almost always malignant.[7] In feline cutaneous melanocytic neoplasms, extensive nuclear atypia, high mitotic activity, and an epithelioid cell type are suggestive of malignancy.[7] When numbers and size of melanin granules obscure the mitotic rate, an H&E stained slide pre-treated with bleach can aid in evaluation. In this Wednesday Slide Conference case, the neoplasm has a mitotic rate of 1 per HPF, with some fields containing up to 3 mitotic figures.

Melanocytes are dendritic cells that are derived form neuroectodermal melanoblasts, and are normally found within the basal layer of the epidermis.[7] Neoplastic transformations have been linked to various molecular changes such as mutation in the *INK4a* and *INK4b* and *Waf-1* genes resulting in malfunction of two tumor suppressor proteins (retinoblastoma protein and p53), proto-oncogene mutation to oncogene, altered expression of epithelial cadherin and CD44 adhesion molecules, and upregulation of angiogenic and other growth factors.[7] A recent study by van Kempen *et al.* has linked the increased expression of Type I collagen to the angiogenic switch that facilitates the progression of microinvasive to deeply invasive tumors in a porcine cutaneous melanoma model.[9]

Malignant melanomas in canines and humans may show chondroid or osseous metaplasia.[3,5] Oyamada *et al.* shows that the cartilaginous matrix transitions from the myxoid matrix produced by de differentiated neoplastic melanocytes.[5] Since the osseous matrix is not associated with either the cartilaginous matrix or the myxomatous matrix, it is theorized that the osteoid matrixes are formed form dense collagenous connective tissue that is also produced by the dedifferentiated neoplastic melanocytes.[5]

Melanomas are common in gray or white horses.[7] More than 90% of these tumors are benign at initial presentation, but approximately two-thirds are thought to progress to malignancy.[7] German Shepherd Dogs and Boxers are more prone to develop oral melanoma.[7] Sinclair miniature and Duroc breeds of swine have a genetic predisposition to developing melanomas.[7] Melanomas have also been reported in cats, cattle, sheep, and alpaca.[7]

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CASE IV - 04-0843 (AFIP 2985667).

Signalment: Adult, 55 lb. male pygmy goat, *Capra hircus*

History: The pygmy goat presented with ulcerated, oozing, pustular lesions on the face and muzzle prior to euthanasia.

Gross Pathologic Findings: Numerous confluent ulcerative, scabby, verrucous and proliferative oozing lesions are present on the muzzle, commissures of the lips, surrounding the eyes, left lateral tongue and the dental pad. A circular ulcer is also present on the left cheek below the left eye. Creamy white exudate drains from some of the larger lesions. The thorax contains creamy tan pus and the pleural surfaces are lined with thick exudate forming adhesions to the thoracic wall and diaphragm. The lungs are consolidated ventrally with multifocal variably sized abscesses containing thick tan pus.

Laboratory Results:
Aerobic bacterial cultures of the muzzle yielded heavy growths of *Arcanobacterium pyogenes* and moderate growths of *Pseudomonas aeruginosa* and *Staphylococcus intermedius*.
Aerobic bacterial cultures of the lungs yielded heavy growths of *Pasteurella treha losi* and *Arcanobacterium pyogenes*.
Electron microscopy: Tissues from muzzle and lips yielded Parapoxvirus (179 X 300nm)

Histopathologic Description: The lesions consist of locally extensive papillary projections of acanthotic, hyperkeratotic epidermis and extensive dermal (or submucosal) inflammatory infiltrate of neutrophils, histiocytes, and lymphocytes with occasional epidermal or dermal pustules and microabscesses. Numerous small capillaries course throughout the dermis. Occasional ballooning vacuolation of keratinocytes with rare eosinophilic intracytoplasmic inclusions are seen. The epidermis is covered with thick serocellular crusts containing degenerating cells and small clusters of bacteria. Deep anastomosing rete pegs extend into the dermis (or submucosa).

Contributor's Morphologic Diagnosis:
Muzzle: Lymphocytic, neutrophilic, histiocytic, pustular and proliferative dermatitis and stomatitis with papillomatous epidermal hyperplasia, acanthosis, hyperkeratosis and occasional eosinophilic intracytoplasmic inclusions in keratinocytes
Lungs: Severe fibrinosuppurative bacterial bronchopneumonia (not included)

Etiologic Diagnosis: Parapoxvirus

Contributor's Comment: Contagious pustular dermatitis (contagious ecthyma, sore mouth, orf) is an infectious dermatitis of sheep and goats with worldwide distribution, caused by *Parapoxvirus*. The genus *Parapoxvirus* is a member of the *Poxviridae* family, and includes orf virus, bovine papular stomatitis virus, and pseudocowpox virus. It is an ovoid, enveloped, double stranded, DNA virus. Transmission is into skin abrasions through aerosols, direct contact, or through mechanical transmission via arthropods.[6] Lesions typically develop on commissures of lips and buccal cavity, but also develop on feet, teats (from nursing an affected kid), and genitals. Lambs and kids are at greatest risk because they are immunologically naïve, and the colostrum from a previously infected animal does not provide protection. Due to its tropism for epithelial cells, *Parapoxvirus* will cause epidermal hyperplasia, producing papular lesions usually within 7 days. Papular lesions progress to vesicles, pustules, and then crusty scabs. In a 2002 study of 16 persistently infected goat kids, lymph node enlargement, premature thymic involution, and a number of secondary bacterial infections were present. It is suggested in this study that individual susceptibility factors of the host, such as breed, genetic susceptibility and immune defects, are contributing factors in orf virus persistence and progression.[1] Infections typically last 3-4 weeks, depending on the severity of systemic disease. Cell mediated immunity is of high importance in recovery from infection. Antibiotics are recommended to prevent secondary complications such as cellulitis, mastitis, aspiration pneumonia, and necrotizing stomatitis. Animals that do recover have transient to solid immunity. Mortality rates in lambs is reported to be 15%.[2] Transmission between sheep and goats can occur, but is uncommon. *Parapoxvirus* may also be transmitted to humans causing similar pustular lesions, commonly on the forearm, hands and face.[3]

Diagnosis of *Parapoxvirus* is based on the recognition of characteristic lesions and lesion distribution. Microscopically, eosinophilic intracytoplasmic inclusion bodies are visible, along with vacuolation and swelling of keratinocytes. The virus particles can also be photographed with an electron microscope. The virus can survive in the environment for months in the scab material shed from affected animals. Virulent, live virus vaccines do exist but are only recommended for use in persistently

infected herds.[4] Contagious pustular dermatitis is of economic significance because lambs and kids become reluctant to eat or suckle, causing weight loss and reduced growth rates. Differential diagnosis for contagious pustular dermatitis should include Foot and Mouth disease, Rinderpest, and Bluetongue.

AFIP Diagnosis: 1. Mucocutaneous junction, lip: Cheilitis, proliferative and necrotizing, focally extensive, severe, with intracytoplasmic eosinophilic inclusion bodies, pygmy goat (*Capra hircus*), caprine.
2. Haired skin, lip: Abscess, focal, with foreign material and fungal hyphae.

Conference Comment: Members of the parapoxvirus genus include orf virus, papular stomatitis virus, pseudocowpoxvirus, parapoxvirus of red deer in New Zealand, and squirrel parapoxvirus.[5] Other species that have been tentatively included include auzduk disease virus, chamois contagious ecthyma virus and seal parapoxvirus.[5] Seal parapoxvirus is the preferred term used rather than 'sealpox virus' to distinguish it from the orthopoxviruses that cause similar clinical diseases.[7]

Characteristic ultrastructural features of parapoxvirus include 250nm X150nm particles, with an oval- to dumbbell-shaped core surrounded by a membrane, lateral bodies, and a surface membrane.[5]

Histopathologic lesions of contagious ecthyma are typical of other poxviral lesions except they usually have a very brief vesicle stage, a prominent ulcer and crust stage, and inclusion bodies present for only a brief period of time during the vesicular stage.[2]

There is variation in sections. Some sections have a focal ulcer with bacterial colonies and neutrophilic mural folliculitis with fungal arthrospores, both likely secondary to the ulcerative lesions induced by the orf virus.

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