The Armed Forces Institute of Pathology
Department of Veterinary Pathology

Conference Coordinator:
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WEDNESDAY SLIDE CONFERENCE 2008-2009

Conference 17

11 February 2009

Conference Moderator:

Dr. Fabio Del Piero, DVM, DACVP

CASE I – S0 10582 (AFIP 3113965)

Signalment: 20-year-old, gelding, Arabian horse (*Equus caballus*)

History: A cecal mass was submitted for histopathology.

Gross Pathology: The submitted tissue was an approximately 4x5x4 cm round, firm, nodular, dark gray, well encapsulated mass with intact surface mucosa. On cut section the mass was distinct from the mucosa, white-tan and multilobulated.

Laboratory Results: None

Histopathologic Description: The mass is a well demarcated, encapsulated, multilobulated, expansile mass located from deep tunica muscularis to the serosa compressing the adjacent tissues and muscle layers. The mass is composed of multiple large lobules separated by a thick dense fibrous connective tissue. The mass is made of a large number of spindle shaped cells arranged in interconnected and branching trabeculae separated by sinuses filled with large amount of basophilic mucinous material as well as clusters of erythrocytes **(Fig. 1-1)**. The neoplastic cells have indistinct cell borders, large amount of eosinophilic cytoplasm, one oval to elongated nucleus with rounded poles and finely granular heterochromatin and 1-2 small basophilic nucleoli. The cells have moderate anisokaryosis with rare mitotic figures per HPF (40X). There are scattered hemosiderin laden macrophages within the mass. There are multifocal areas of chronic hemorrhages with aggregations of siderophages present in the capsule.

Immunohistochemistry was performed. The submitted mass was strongly positive for vimentin and c-kit and slightly positive for NSE. It did not stain with desmin, smooth muscle actin and S100. Unfortunately, no history was submitted with this mass to know the reason for removal.

Contributor's Morphologic Diagnosis: Cecum: Gastrointestinal stromal tumor with peripheral hemorrhage and siderophages

Contributor's Comment: Equine gastrointestinal stromal tumors are unique, benign mesenchymal tumors often found in the cecum of horses. They are detected at surgery, meat inspection or necropsy and may occur in stomach, small intestine and most commonly in the

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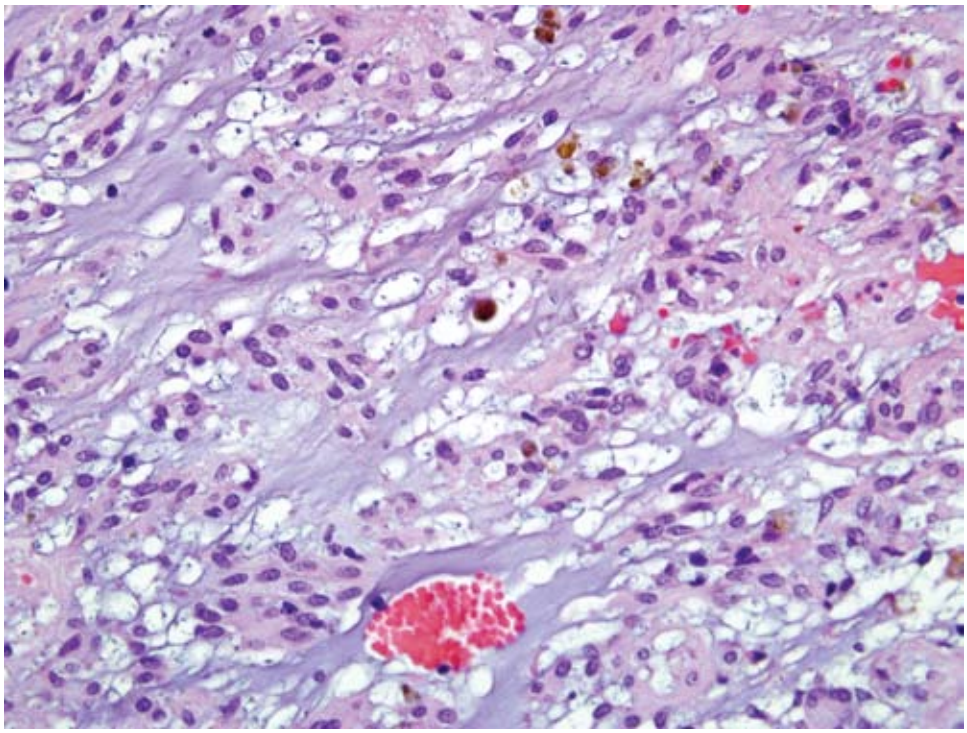

1-1. Large intestine, horse. Gastrointestinal stromal tumor. Neoplastic cells are separated by myxomatous matrix with scattered hemorrhage and few hemosiderin-laden macrophages. (HE 400X)

cecum (as in this case) and colon.[1] These tumors are different from leiomyomas although they have common features. Grossly, these tumors are distinct and form exophytic multinodular masses on the serosal surface or transmurally. Histologically, they are usually composed of interlacing fascicles with multiple sinuses filled with mucinous materials stained by alcian blue. There are often hemorrhagic and may have myxomatous islands within their parenchyma.[3] Immunohistochemically, they are positive for c-kit protein (CD117), vimentin and neuron specific enolase and mildly for smooth muscle actin.[4]

AFIP Diagnosis: Cecum: Gastrointestinal stromal tumor, myxoid

Conference Comment: Gastrointestinal stromal tumors (GISTs) have been reported in numerous domestic species and are thought to arise from the interstitial cells of Cajal, the precursors to the pacemaker cells in the intestinal wall.[2] Histologically, these tumors closely resemble leiomyosarcomas, and immunohistochemistry is needed to differentiate them. GISTs are normally composed of spindle cells arranged in interlacing fascicles or in a whirling pattern. Another less common pattern is the myxoid pattern characterized by epithelioid cells arranged in trabeculae or sheets within a myxoid matrix.[1]

These tumors are a recently recognized entity in veterinary medicine, and thus criteria for malignancy have not yet been solidified. In humans, larger size and greater mitotic activity point to malignancy and a poor prognosis, but future studies will be needed to determine their behavior in domestic animals.[1]

Contributing Institution: CAHFS – UC Davis; www.cahfs.ucdavis.edu

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CASE II – 23279-08 (AFIP 3103695)

Signalment: 10-year-old female equine Thoroughbred (*Equus caballus*)

History: The mare had signs of equine protozoal myeloencephalitis (EPM) then developed pneumonia. The mare responded to EPM therapy but had continuing weight loss and cachexia despite a variety of treatments for pneumonia. The mare was euthanized due to continued loss of body condition.

Gross Pathology: Significant changes were restricted to the lungs. Bilaterally, lung lobes were diffusely firm and failed to collapse when the thoracic cavity was opened. Throughout the pulmonary parenchyma were multifocal to coalescing firm, light pink, irregularly round nodules that varied in size from 1cm to large confluent masses. The nodules were separated by normal appearing tissue. When sectioned, the masses had a firm fibrous texture and bulged slightly on cut surface.

Laboratory Results: None

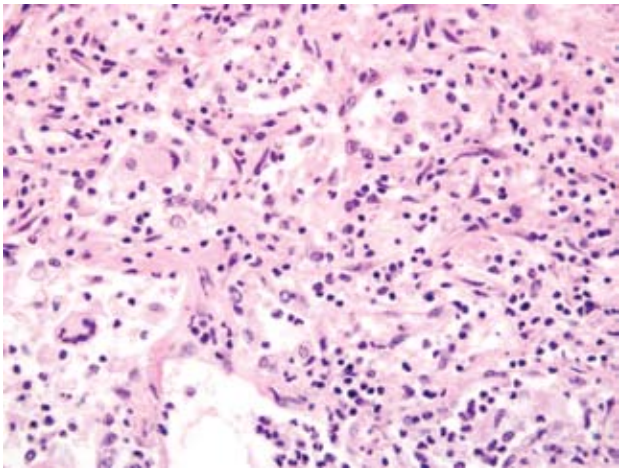
Histopathologic Description: Sections of lung are characterized by multifocal areas of pulmonary fibrosis and inflammation bordered by more normal appearing lung tissue. Alveolar septa are moderately to severely expanded by well organized mature collagen **(Fig. 2-1)**. Alveoli are often lined by plump cuboidal cells (Type 2 pneumocyte hyperplasia), and alveolar lumens contain a mixture of sloughed epithelial cells, intact and degenerate neutrophils, large foamy macrophages, and few multinucleated giant cells. Rare macrophages contain large, brightly eosinophilic, round, intranuclear inclusion bodies that peripheralize nuclear chromatin and are occasionally surrounded by a clear halo **(Fig. 2-2)**.

Contributor's Morphologic Diagnosis: Severe multifocal nodular fibrosing interstitial pneumonia with intranuclear inclusion bodies, *Equus caballus*; Equine multinodular pulmonary fibrosis

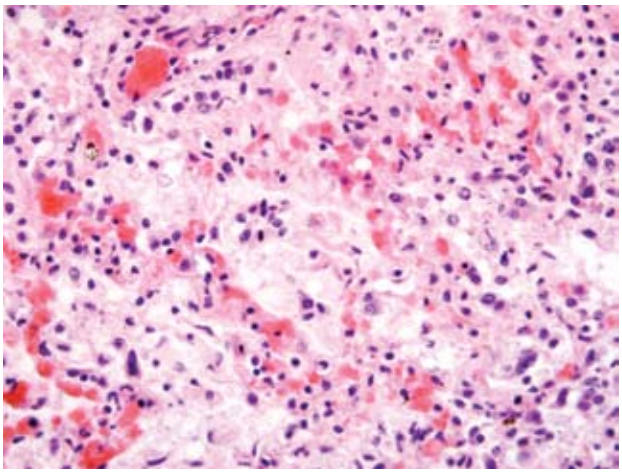
Etiology: Equine Herpesvirus-5 (EHV-5)

Contributor's Comment: Equine multinodular pulmonary fibrosis (EMPF) is a distinct entity in the category of equine chronic pulmonary disease. It is differentiated from other fibrosing inflammatory diseases, such as recurrent airway obstruction (RAO) or chronic pneumonia, by the nodular pattern of fibrosis. This disease may have been previously reported under the names "idiopathic interstitial pneumonia with fibrosis" or "idiopathic pulmonary fibrosis."

EMPF is most common in middle-aged horses. Horses with this disease present with non-specific signs of respiratory disease including cough, fever, weight loss, tachypnea, increased respiratory effort, and nasal discharge. Treatment with antibiotics may result in short-term improvement, but symptoms recur after treatment is discontinued. Pulmonary nodules can be visualized



2-1. Lung, horse. Equine multinodular pulmonary fibrosis. Abundant septal fibrosis, moderate type II pneumocyte hyperplasia, numerous lymphocytes, histiocytes, and neutrophils, with scattered multinucleated giant cells. (HE 400X)



2-1. Lung, horse. Equine multinodular pulmonary fibrosis. Abundant septal fibrosis, moderate type II pneumocyte hyperplasia, numerous lymphocytes, histiocytes, and neutrophils, with scattered multinucleated giant cells. (HE 400X)

with ultrasonography and/or radiography. Rule outs include neoplasia, fungal pneumonia, and granulomatous pneumonia, which may also result in a nodular pattern. Treatment of choice includes long-term (at least 6 weeks) administration of corticosteroids. Prognosis for the disease is considered fair to poor. Many horses are euthanized due to continued respiratory complications and poor athletic performance.

Lung tissue from this horse was positive via polymerase chain reaction (PCR) for EHV-5. EHV-5 is a DNA gammaherpes virus that has only recently been associated with this disease entity. The pathogenesis of EHV-5 in this disease has not been fully elucidated but may contribute to a "pro-fibrotic" lung environment via a Th-2 lymphocyte inflammatory response. PCR can be performed on bronchoalveolar lavage samples or lung tissue. Unlike EHV-2, another common viral cause of equine respiratory disease, EHV-5 is not isolated from healthy horses or horses with respiratory disease due to other etiologies. A study by Bell identified EHV-5 in 64% of young racehorse nasal swabs, while a study performed by Wang identified the virus in 48% of 5-9 month old Thoroughbred foals.

Intranuclear inclusion bodies are not a common feature in gammaherpes infections but may be observed in California sea lions infected with genital carcinoma caused by gammaherpes virus. Murine herpesvirus-68 is a gamma herpesvirus associated with experimental fibrotic lung disease in mice.

AFIP Diagnosis: Lung: Fibrosis, interstitial, nodular, multifocal, severe with neutrophilic and histiocytic alveolitis, type II pneumocyte hyperplasia and rare intrahistiocytic eosinophilic intranuclear inclusions

Conference Comment: There are several alpha herpesviruses that are of significance in domestic animals. Listed below are some of the major alpha herpesviruses of significant importance in veterinary medicine.

Alpha herpesvirus	Disease
Equine herpesvirus 1	Abortion, foal mortality, neurologic and respiratory disease
Equine herpesvirus 3	Coital exanthema
Equine herpesvirus 4	Rhinopneumonitis
Bovine herpesvirus 1	Infectious bovine rhinotracheitis, infectious pustular vulvovaginitis
Bovine herpesvirus 2	Bovine mammilitis, pseudo-lumpyskin disease
Porcine herpesvirus 1 (pseudorabies; Aujeszky's Disease)	Abortion in adults and generalized disease in younger animals; death in other species
Canine herpesvirus 1	Pneumonia, hepatitis, nephritis – hemorrhagic disease
Feline herpesvirus 1	Feline viral rhinotracheitis
Gallid herpesvirus 1 (chickens)	Infectious laryngotracheitis
Gallid herpesvirus 2 (chickens)	Marek's disease
Anatid herpesvirus 1	Duck plague
Cercopithecine herpesvirus 1 (B virus)	Herpes simplex-like disease in macaques; encephalitis and death in humans

Contributing Institution: University of Kentucky, Livestock Disease Diagnostic Center, Lexington, Kentucky; www.lddc.uky.edu

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3. Wong DM, Belgrave RL, Williams KJ, Del Piero F, Alcott CJ, Bolin SR, Marr CM, Nolen-Walston R, Myers RK, Wilkins PA: Multinodular pulmonary fibrosis in five horses. J Am Vet Med Assoc **15**:898-905, 2008

CASE III – 63383-02 (3103693)

Signalment: 10-month gestation placenta, Thoroughbred, (*Equus caballus*), equine

History: The mare was due to foal on January 20, 2003, but the mare foaled on December 28, 2002.

Gross Pathology: A complete placenta is submitted for examination. Along the chorionic surface of the body and non-pregnant horn are multifocal regions of denuded villi covered with a thick mucoid exudate. Adjacent villi are thickened and edematous. No other significant gross lesions are observed.

Laboratory Results:
Bacteriology
—Placenta: *Cellulosimicrobium cellulans* formerly known as *Oerskovia xanthineolytica* (numerous)
—FA testing for the detection of *Leptospira interrogans* on the placenta was negative.

Histopathologic Description: Allantochorion: Amorphous eosinophilic material with scattered aggregates of bacteria, intact and degenerate neutrophils, and cellular debris multifocally overlies the chorionic surface. The chorionic villi, lined by hypertrophic trophoblasts with mild to moderate cytoplasmic vacuoles, are multifocally blunted. Aggregates of moderate to large numbers of lymphocytes, plasma cells, and neutrophils occupy the superficial stroma adjacent to the villi and villous projections. The supporting stroma is characterized by diffuse, moderate edema, an infiltrate of low to moderate numbers of lymphocytes, neo-vascularization and moderate fibroplasia.

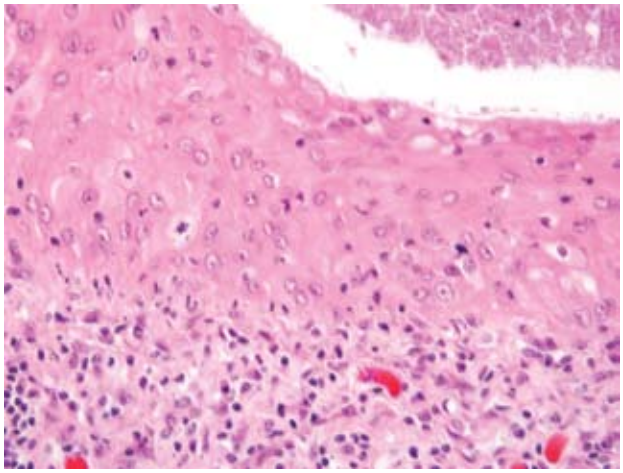
Contributor's Morphologic Diagnosis: Severe multifocal necrotizing exudative placentitis; *Equus caballus*

Contributor's Comment: *Cellulosimicrobium cellulans* is a gram positive, branching, motile, oxidase negative, catalase positive, non-acid fast-bacillus. When grown on agar, the colonies have a characteristic yellow color.[3] The bacteria are found in soil and have been associated with middle and late term abortions, as well as premature births in horses.[1] Lesions are typically but not always seen in the both the fetus and placenta. Fetal lesions include firm, expanded lungs, and mildly enlarged friable livers with occasional pale foci along the capsular surface that extend into the parenchyma.[1] Lesions observed in the allantochorion include a brown mucoid exudate overlying well demarcated areas of denuded villi along the chorion. In previous reports, portions of the chorion affected include areas adjacent to the cervical star, along the body, or encompassing the horns. Microscopically, the chorionic surface is covered by an eosinophilic exudate and had blunted villi and marked inflammation of both the villi and supporting stroma. Often seen with the placentitis is a pyogranulomatous pneumonia characterized by multinucleated giant cells, macrophages, and neutrophils occupying the lumens of alveoli, bronchi, and bronchioles.[1]

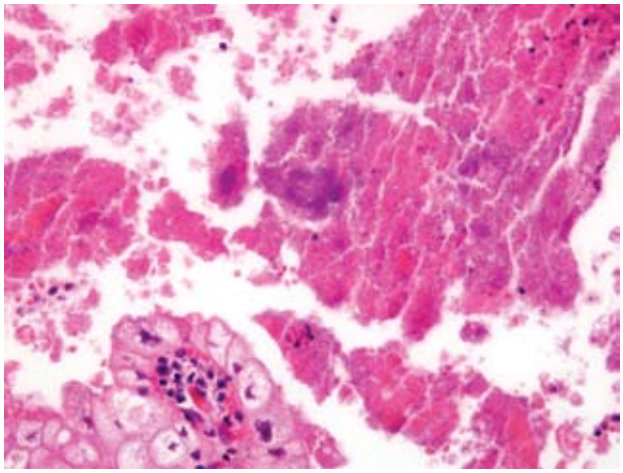
Given the presence of an exudative placentitis, the distribution of the placental lesions, and the characteristics of *Cellulosimicrobium cellulans*, there are similarities between the bacteria described here and placentitis resultant of a *Crossiella equi* infection. Pyogranulomatous pneumonia, as seen with *Cellulosimicrobium cellulans*, does not accompany nocardioform placentitis.

Common bacterial agents resulting in placentitis and abortion in the horse include *Leptospira* spp, nocardioform, *Escherichia coli*, *Streptococcus zooepidemicus*, *Pseudomonas aeruginosa*, *Streptococcus equisimilis*, *Enterobacter agglomerans*, *Klebsiella pneumoniae*, and alpha- hemolytic *Streptococcus*.[2]

AFIP Diagnosis: Placenta, allantochorion: Placentitis, necrotizing, subacute, diffuse, moderate, with squamous metaplasia **(Fig. 3-1)**, fibrin, edema, and large colonies of coccobacilli **(Fig. 3-2).**



3-1. Chorioallantois, horse. Necrosuppurative bacterial chorionitis. Squamous metaplasia of the allantoic epithelium. (HE 400X)



3-2. Chorioallantois, horse. Necrosuppurative bacterial corionitis. Multifocally within necrotic chorionic villi, there are large colonies of 0.5 x 1 micron basophilic coccobacilli. (HE 400X)

Conference Comment: *Cellulosimircobium cellulans*, formerly named *Oerskovia xanthineolytica*, is rarely found in humans and is generally an opportunistic pathogen in immunocompromised hosts.[1] This organism is being recognized more frequently as a cause of equine abortion.

The bulk of the conference discussion centered on the outbreak of abortions in Kentucky in the spring of 2001 and 2002 dubbed Mare Reproductive Loss Syndrome (MRLS). MRLS caused large numbers of abortions reaching epidemic proportions in Kentucky.[4] The economic loss alone has been estimated at almost $500 million.

Mares with MRLS abort late in gestation or at term and the fetuses are still enclosed within the placenta and in good condition. Mares show no overt clinical illness prior to abortion. In the fetus, gross lesions include hemorrhages in the chorion, amnion, and amniotic segment of the umbilical cord, pleura, and heart, hypema, and uninflated lungs. Additionally, the amniotic cord is often dull gray and thickened. Histologically, macrophages and neutrophils are seen in ulcerated areas on the surface of the amniotic cord in association with bacteria. The allantochorion also has similar lesions. Funisitis, amnionitis, pneumonia, fetal bacteremia, and chorionitis are also evident microscopically.

The most commonly implicated cause for MRLS is the eastern tent caterpillar (ETC). Feeding of the eastern tent caterpillar to pigs has resulted in abortions in one study. Similar studies in horses have demonstrated that the feeding of the exoskeleton of the eastern tent caterpillar causes abortion in mares. One of the most current hypotheses is that a portion of the caterpillar cuticle is responsible for its abortifacient effects.[4]

Contributing Institution: University of Kentucky; Livestock Disease Diagnostic Center; Lexington, Kentucky; 40512; www.lddc.uky.edu

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CASE IV – S07-514.1 (AFIP 3103232)

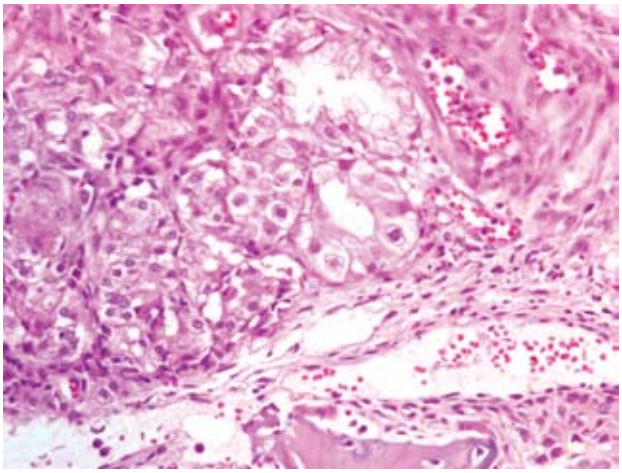
Signalment: Suckling pig, about 4 weeks of age, female, porcine, breed unspecified (*Sus domestica*)

History: Reduced weight gain within the herd. Suckling pigs show heavy breathing and die unexpectedly.

Gross Pathology: Multiple tongue erosions, yellowish secretion from the nose, yellowish plaques on nasal turbinates.

Laboratory Results: *Corynebacterium* spp. were isolated from the nasal turbinate.

Histopathologic Description: Nasal turbinate: Diffusely, the subepithelial connective tissue is expanded and infiltrated by numerous lymphocytes, plasma cells, and fewer macrophages. Epithelial cells of the nasal mucous glands and the respiratory epithelium are multifocally slightly enlarged (cytomegaly) with abundant, foamy, eosinophilic cytoplasm and a single 20-30 mm, basophilic, irregular, smudgy and granular, intranuclear inclusion body **(Fig. 4-1)** that fills and distends the karyomegalic nucleus. Multifocally the mucous glands are ectactic, lined by attenuated epithelium, and contain variable amounts of eosinophilic cellular and karyorrhectic debris (necrosis) with rare neutrophils. The respiratory epithelium is multifocally lost and replaced by a moderate amount of karyorrhectic and pyknotic cell debris (necrosis). In the nasal passage, there is an exudate composed of erythrocytes, sloughed epithelial cells, and some slightly eosinophilic material (mucous).



4-1. Nasal turbinates, pig. Inclusion body rhinitis. Amphophilic to eosinophilic intranuclear inclusion bodies within mucosal epithelium and glandular epithelium of the nasal turbinates. (HE 400X)

There is a large focally extensive necrotic area (not included in every slide), characterized by a large amount of eosinophilic karyorrhectic and pyknotic cell debris, and bordered by an abundant amount of small and medium caliber vessels surrounded by reactive fibroblasts and collagen (granulation tissue).

Contributor's Morphologic Diagnosis: Nasal turbinate: Rhinitis, lymphoplasmacytic and necrotizing, subacute, diffuse, marked, with glandular epithelial karymegaly due to intranuclear inclusion bodies

Contributor's Comment: Inclusion body rhinitis – porcine cytomegalovirus (PCMV)
PCMV infections are ubiquitous and occur throughout the world, but clinical disease is much less frequent. Inclusion body rhinitis is typically an acute to subacute disease of 3-5-week-old suckling piglets. Piglets exhibit fever, sneezing, catarrhal nasal exudate, shivering, and occasional dyspnoe. Morbidity is high and mortalitiy is low unless secondary bacterial infections develop. Systemic cytomegalovirus infections usually infect piglets less than 3 weeks of age. These piglets may be found dead without premonitory signs, or exhibit sneezing, lethargy, anorexia, subcutaneous edema of the jaw and tarsal joints, and dyspnea. Infection of naïve pregnant sows induces mild lethargy, anorexia, and delivery of stillborn or weak piglets. Inclusion body rhinitis is caused by PCMV, a beta-herpesvirus. Piglets commonly shed the virus soon after weaning at 3 weeks of age, suggesting that infection is usually acquired by contact with nasal secretions of infected cohorts. Other pigs, particularly those that develop generalized disease, are probably infected from the sow in the neonatal period. The virus replicates in nasal submucosal and lacrimal glands. Viremia develops at 5-14 days after infection depending on the age of the pig and leads to infection of epithelial cells in renal tubules, liver, duodenum, and elsewhere. Pulmonary alveolar and splenic macrophages may be additional sites of viral replication. Virus is shed in nasal and ocular secretions, in the urine, and in vaginal secretions of sows.[3]

Infection can be diagnosed by the presence of characteristic large, basophilic, intranuclear inclusion bodies in cytomegalic cells of the nasal glandular epithelium. Such inclusion bodies and mononuclear cellular infiltrations can also be detected in the tubular epithelia of the kidneys, as well as the epithelia of the salivary and tear glands. Other, less frequent, lesions include interstitial pneumonia and lymphocytic perivasculitis in the brain. Predisposing factors of the infection are not fully known. A low level of immunity within the herd, e.g., in newly established

Herpesviridae		
Alphaherpesvirinae: focal lesions in skin and mucosa of resp. and genital tract; abortion; neonates: necrosis in multiple organs, latency in nerves	Equine herpesvirus 1: Equine herpesviral abortion, rhinopneumonitis, neurologic disease	horse
	Equine herpesvirus 3: Equine coital exanthema	horse
	Equine herpesvirus 4: Equine rhinopneumonitis	horse
	BHV-1: infectious bovine rhinotrcheitis/infectious pustular vulvovaginitis/infectious balanoposthitis	cattle
	BHV-2: bovine mammilitis virus (bovine herpes mammilitis)	cattle
	BHV-5: bovine herpesvirus encephalitis	cattle
	SHV-1: Aujezky´s disease, Pseudorabies	pig>others
	Canine herpesvirus 1:	dog
	Feline herpesvirus 1: upper respiratory tract disease (rhinotracheitis) and conjunctivitis (ulcers)	cats
	Feline herpesvirus 1: feline herpesvirus ulcerative dermatitis	cats
	Gallid herpesvirus-1: Infectious laryngotracheitis (ILT)	chicken
	Gallid herpesvirus-2: Marek´s disease	chicken
	Psittacine herpesvirus: Pacheco's disease	psittacines
	Anatid herpesvirus-1: Duck plaque/Duck virus enteritis	ducks, geese, swan
	Simplexvirus: HSV-1, HSV-2, HBV, BHV-2	
	Herpesvirus simplex, type 1/type 2	human & nonhuman primates
	Herpesvirus simiae/Herpes B/Cercopithecine HV	rhesus macaques
	Simian varicella virus	macaques, AGM, Patas monkeys
Betaherpesvirinae: no cell lysis, karyomegaly, latency in secretory glands, lymphoreticular organs, kidney	HHV-5, HHV-6, MCMV-1	humans

	Porcine herpesvirus 2: porcine cytomegalovirus disease/Inclusion body rhinitis	porcine
	Cytomegalovirus	humans + nonhuman primates
Gammaherpesvirinae: primates: lymphoproliferative disease, latency in lymphoid tissue	EHV-2	
	EHV-5	
	BHV-4: bovine herpes mammary pustular dermatitis	cattle
	OHV-2/AHV-1: malignant catarrhal fever	various ruminants
	Epstein-Barr virus (lymphocryptovirus-gamma 1)	primates
	Kaposi-sarcoma-associated herpesvirus/human herpesvirus-8 (KSHV/HHV8) (Rhadinovirus-gamma 2)	primates
Deltaherpesvirinae	Anatid herpesvirus-1: duck plague	
	SHV-2: Eischlusskörperchenkrankheit	pig
	Karpfenpocken	fish
Uncharacterized viruses	Koi-herpesvirus (KHV, carp nephritis and gill necrosis virus, CNGV, Cyprinid-Herpesvirus-3, CyHv-3)	fish

herds, and immunosuppressive effects may play a role. Seroconversion due to PCMV is probably much more frequent than clinical disease.[1] In our case, sows in the herd had problems with insufficient lactation.

AFIP Diagnosis: 1. Nasal turbinates: Rhinitis, necro-ulcerative, subacute, diffuse, moderate, with glandular epithelial eosinophilic intranuclear inclusions
2. Haired skin and bone: Necrosis, focally extensive, with granulation tissue, fibrosis, osteonecrosis and osteolysis

Conference Comment: The contributor provides a comprehensive summary of this condition. One additional rule-out considered by attendees was atrophic rhinitis; however, changes in the nasal turbinate cartilage and bone would be an expected finding, as well the absence of inclusion bodies. For comparison purposes, a brief discussion of atrophic rhinitis is included here. Atrophic rhinitis is also a common disease in pigs worldwide. Atrophic rhinitis can be split into nonprogressive atrophic rhinitis (NPAR), caused by *Bordetella bronchiseptica*, or progressive atrophic rhinitis (PAR), caused by toxigenic *Pasteurella multocida* as a single agent or in combination with *Bordetella bronchiseptica*. Both of these disease cause hypoplasia of the nasal turbinates that clinically manifest as frequent sneezing by affected swine. Progression of either of these diseases can lead to distortion of the snout. Nasal hemorrhage is commonly seen in PAR; NPAR nasal hemorrhage is uncommon.[2]

Both *Bordetella bronchiseptica* and *Pasteurella multocida* produce their own toxins, and the severity of disease is dependent on the amount of toxin absorbed. One major difference between these two organisms is the age of affected pigs. PAR can affect pigs older than 3 months of age, whereas NPAR normally only affects pigs up to 6 weeks of age.[2]

Gross lesions in PAR are restricted to the nasal cavity and adjacent bone with the ventral scrolls of the nasal turbinates

most often suffering the worst lesions. Histologically, the pathognomonic lesion of PAR is replacement of the bony plates of the ventral conchae with fibrous tissue. Metaplasia of adjacent respiratory epithelium is also common.[2]

Contributing Institution: Institute of Veterinary Pathology of the University of Zurich, CH-8057 Zurich, Switzerland, www.vetpathology.unizh.ch

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