The Armed Forces Institute of Pathology
Department of Veterinary Pathology

WEDNESDAY SLIDE CONFERENCE 2007-2008

Conference 2

12 September 2007

Moderator:

Dr. Sarah Hale, DVM, Diplomate ACVP

CASE I – PA5-60150 (AFIP 2985164).

Signalment: 2-month-old, filly, Quarter Horse, Equine, *Equus caballus*

History: The filly had a body temperature of 103F and harsh lung sounds. Ultrasound examinations showed "comet tails" on pleural surface. The animal was treated using azithromycin and banamine. The animal improved slightly and naxcel was included in the treatment. Despite the treatment, the animal was found dead in the stall. Prior to this episode, the filly was given two injections of hyperimmune plasma to prevent *Rhodococcus equi* infection.

Gross Pathology: A 2-month-old quarter horse filly was submitted for necropsy. The foal was in good body condition with adequate deposits of fat stores present. Hydration appeared adequate. The lungs were diffusely reddened, firm, and sank in formalin. There was a single 7 cm nodular area of caseation within the right cranioventral lung lobe. The tracheobronchial lymph nodes were markedly enlarged and contained a thick creamy exudate. The distal third of the trachea was hemorrhagic and contained linear streaks of ulceration/erosion. The omentum and mesentery were hemorrhagic. There was a large bilobed abscess, approximately 15 cm in diameter, within the mesentery by the ceco-colic junction. The center of the abscess was filled with pasty white necrotic material. A similar abscess was present within the mesentery adjacent to the jejunum. This abscess was adhered to the wall of the jejunum, and the overlying mucosa was focally ulcerated. There was a focal irregular area of hyperkeratosis in the nonglandular portion of the stomach next to the margo plicatus. There was an area of subcutaneous hemorrhage in the dorsal lumbar area.

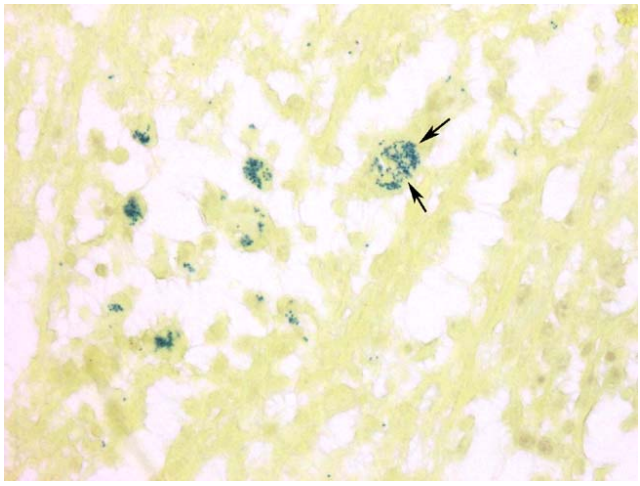
Laboratory Results:
1. Bacteriology: Lung abscess, lymph node swab yielded *Rhodococcus equi*
2. Fluorescent Antibody Tests: Negative for Adenovirus, EHV 1 & EIV
3. Virus isolation from lung: Negative

Histopathologic Description: A section of consolidated lung was examined, and is characterized by filling of alveoli with fibrin, macrophages, and occasional neutrophils. Occasional type II cell hyperplasia is present. Scattered alveoli contain multinucleated giant cells. Small numbers of plasma cells are present within some alveoli and within the thickened alveolar septae. The alveolar exudate occasionally is necrotic. Bronchioles and bronchi are generally devoid of inflammatory cells. A section of lung from the right cranioventral lung lobe contains large areas of abscessation. These foci contain sheets of degenerating neutrophils admixed with fewer

macrophages and replace normal pulmonary parenchyma. Many of the macrophages contain **intracytoplasmic bacteria consistent with *Rhodococcus*** (**fig. 1-1**). There is mild fibroplasia around these areas, and the adjacent alveoli contain multinucleated giant cells, macrophages, lymphocytes, and plasma cells. There is acute hemorrhage within the deep lamina propria of the trachea. There is diffuse congestion, and the overlying mucosa is focally ulcerated. Ulcerated foci are covered with degenerated neutrophils and fibrin, and the base is infiltrated with neutrophils, macrophages, and occasional multinucleated giant cells.

Contributor's Morphologic Diagnosis: 1. Severe diffuse histiocytic bronchointerstitial pneumonia with focal abscessation with intracellular bacteria
2. Acute ulcerative tracheitis with hemorrhage and pyogranulomatous inflammation

Contributor's Comment: The pulmonary lesions present in the caudoventral portion of the lungs were typical in distribution and gross and microscopic appearance to lesions caused by *Rhodococcus equi*. The mesenteric lymph node involvement is also common in this disease. The diffuse inflammatory changes present in the rest of the lung actually predominated in this case, and have not been typically associated with this disease. These changes have been described in a group of foals with *Rhodococcus* infection.[3] An underlying viral etiology was suspected in these foals, but attempts to demonstrate a viral component were generally unrewarding. It may be that the bacteria are inducing a hypersensitivity type response in areas of the lung not colonized by bacteria. The reason this response occurs in particular groups of animals is unknown.



1-1 Lung, Quarter horse. Intrahistiocytic gram positive bacteria, consistent with Rhodococcus (arrows).

Twenty-three foals, between 1 and 7 months old, with signs of acute respiratory distress, were examined at the Veterinary Medical Teaching Hospital (VMTH), University of California, Davis, between 1984 and 1989. Characteristic features included sudden onset of severe respiratory distress and tachypnea, cyanosis unresponsive to nasal oxygen, pyrexia, hypoxemia, hypercapneic respiratory acidosis, poor response to treatment, and histopathologic lesions of bronchiolitis and bronchointerstitial pneumonia. Seven of the 23 foals were normal before the onset of respiratory distress, 3 foals were found dead, and 13 foals were being treated for respiratory tract infections at the time of presentation. Laboratory data obtained for 13 horses showed increased plasma fibrinogen concentration (630.7 +/- 193 mg/dL), leukocytosis (18,607 +/- 7,784/microL), and neutrophilia (13,737 +/- 8,211/microL). Thoracic radiographs showed a diffuse increase in interstitial and bronchointerstitial pulmonary opacity and, in 5 foals, an alveolar pulmonary pattern of increased density was also seen. In 3 foals, heavy interstitial infiltration proceeded to a coalescing nodular radiographic appearance. Microbiological culture of tracheobronchial aspirates (TBA) from 9 foals yielded bacterial growth, but no one bacterial species was consistently isolated. Microbiological culture of postmortem specimens of the lung from 6 foals yielded growth of bacteria that included *Escherichia coli*, *Enterobacter spp.*, *Proteus mirabilis, Klebsiella pneumoniae, Rhodococcus equi,* or beta-hemolytic *Streptococcus spp.* Tracheobronchial aspirates from 4 foals and lung samples collected from a further 4 foals at necropsy yielded no bacterial growth. Cultures were not taken from two foals premortem or postmortem. Virologic examination of TBA, lung tissue, or pooled organ tissue from 12 foals was negative. Viral culture of TBA from 1 foal showed cytopathic effects and positive immunofluorescence for equine herpes virus type II (EHV-II). In addition to the 3 foals that were found dead, 11 foals died or were euthanatized. Pathologic lesions were limited to the lungs in 50% of the foals; the remainder also had bowel lesions suggestive of hypoxic injury. The predominant histopathologic pulmonary lesions included bronchiolitis, bronchiolar and alveolar epithelial hyperplasia, and necrosis. Many bronchioles were filled with mucoid and fibrinocellular exudate. The peribronchiolar interstitium and adjacent alveolar spaces were also infiltrated with inflammatory cells and contained proteinaceous edema fluid. Type II cell hyperplasia and hyaline membrane formation were observed in the majority of foals and in 2 foals alveolar multinucleate giant cells were also present.[3] Later, another foal from the same farm was submitted for necropsy. The second foal had similar gross and histopathologic lesions indicating an endemic infection.[2]

AFIP Diagnosis: 1. Lung: Pneumonia, interstitial, necrotizing, histiocytic, lymphoplasmacytic, and neutrophilic, diffuse, marked, with fibrin and hyaline membranes, Quarter horse (*Equus caballus*).
2. Lung: Pneumonia, pyogranulomatous, focally extensive, severe, with intrahistiocytic coccobacilli.

Conference Comment: *Rhodococcus equi* is a facultative, intracellular, Gram-positive bacteria that is present in soil and feces and is often enzootic on farms.[4] Two classic forms of the disease are suppurative to pyogranulomatous bronchopneumonia and ulcerative enterocolitis. Approximately half of the foals affected with the respiratory form have concurrent intestinal lesions. Intestinal lesions without the respiratory form is not common.[4] The lymph nodes, joints, bones, genital tract, and other organs may also be involved.[4] There are sporadic reports in other species, including cattle, goats, pigs, dogs, cats, and immunocompromised humans.

Rhodococcus equi appears to be easily killed by neutrophils but not macrophages. Upon entry through either inhalation or ingestion the bacteria are phagocytosed by either alveolar or intestinal macrophages. Several proposed virulence factors encoded by plasmids allow survival within macrophages. Vap A, Vap B, and Vap C, as well as glycolipids, capsular polysaccharides, and "equi factors" (cholesterol oxidase and choline phosphohydrolase) contribute to the virulence of certain *Rhodococcus equi* strains.[1] They prevent lysosomal fusion and/or result in premature lysosomal degranulation, survival of the bacteria, and death of the macrophage.[4]

Diffuse interstitial pneumonia is not a classic lesion of *Rhodococcus equi* pneumonia and is likely due to a separate disease process. The findings of necrotizing interstitial pneumonia with hyaline membrane formation are suggestive of the acute phase of diffuse alveolar damage (DAD). DAD results from diffuse injury to type I pneumocytes with subsequent hyaline membranes formation, type II pneumocyte proliferation and interstitial fibrosis.[3] These histologic lesions are non-specific, and identification of an etiologic agent is often difficult. Causes of DAD include, but are not limited to, thermal injury, toxic gases, septicemia, ingested toxins (paraquat, kerosene, *Brassica*, and perilla mint), endotoxemia, acute hypersensitivity reactions, ventilator-induced injury, and chronic left heart failure.[3]

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http://www.vet.uga.edu/vpp/index.html

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CASE II – 4029-07 (AFIP 3065685).

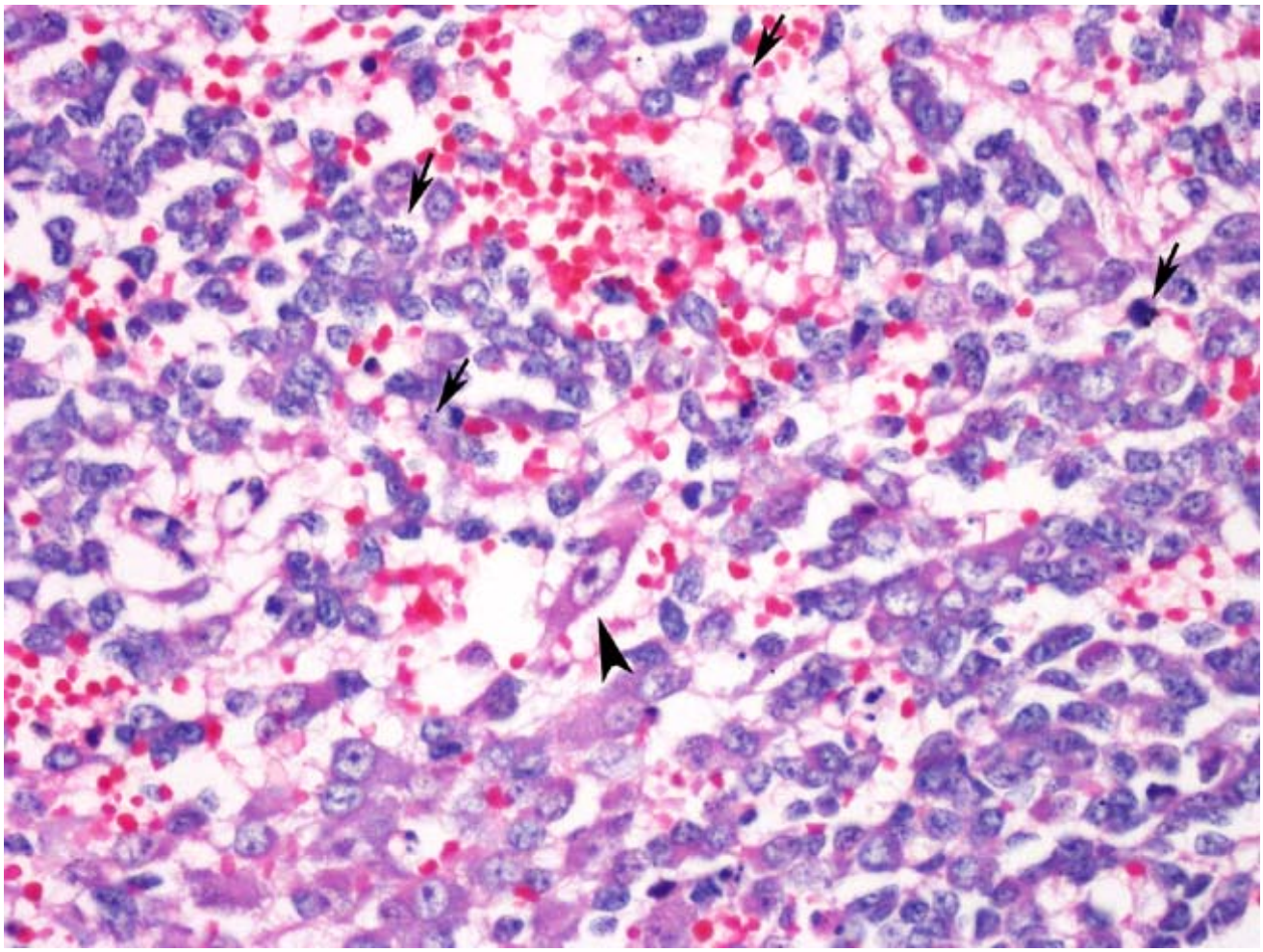
Signalment: 13-month-old, neutered male mixed breed dog, *Canis familiaris*

History: This dog was presented for lethargy and progressive emaciation of several weeks duration. Fluid therapy, antibiotics, antifungals, and anti-inflammatory treatments were unsuccessful. The animal became recumbent and painful and was euthanized.

Gross Pathology: The animal is severely emaciated, evidenced by lack of internal body fat and severe muscle wasting. There is a very large, firm, gray-white and mottled red mass in the mediastinum measuring 30x9x15cm that compresses the lungs caudoventrally. The mass surrounds the esophagus, trachea, and great vessels but does not appear to compress their lumens. The spleen is meaty and markedly enlarged (30x10x3cm). The liver contains numerous gray-white solid and sometimes fluid-filled vesicles scattered diffusely.

Laboratory Results: Complete blood count and chemistry profile were fairly unremarkable with the following abnormalities:

CBC results from one week prior to euthanasia include



2-1 Spleen and liver, dog. Multifocally, the neuroblastoma contains cells which resemble ganglion cells (arrowhead and lower left). The small neoplastic cells consistent with neuroblasts have a high mitotic rate (arrows). (H&E 400X)

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the following abnormalities (normal values with reference range in brackets)

Hematocrit	17.5%	[37.0 – 55.0]
Hemoglobin	6.2 g/dL	[12.0 – 18.0]
Platelets	118 x10^9/L	[175 – 500]

Serum chemistry profile taken at the same time was normal except for a slightly low creatinine (0.4 mg/dL [0.5 – 1.8]).
AGID testing for aspergillosis, blastomycosis, coccidiodomycosis, and histoplasmosis was negative.
Blood parasite analysis was negative.
Reticulocyte counts were 8.1% one week prior to euthanasia.
Bacterial culture of the liver taken at necropsy was negative.

Histopathologic Description: Sections of the tumor mass, spleen, and liver are submitted.

Sections of the tumor mass consist of sheets of small, polygonal to stellate cells with scant cytoplasm and dense, central nuclei within a fibrovascular stroma. Islands of large, polygonal cells resembling neurons are scattered irregularly within lobules. Similar foci are seen in the liver, but the spleen is filled primarily with the small cells.

The small cells observed in all sections are consistent with **neuroblasts (fig. 2-1),** whereas the larger neuron-like cells resemble ganglion cells. The histologic features are consistent with ganglioneuroblastoma, a rare tumor arising from the sympathetic ganglia. This one was malignant and sent metastases to the liver and spleen. Metastases were not found in the lung.

Contributor's Morphologic Diagnosis: Ganglioneuroblastoma with hepatic and splenic metastasis

Contributor's Comment: Neuroblastomas are rare tumors that originate from the sympathetic nervous system ganglia and have been reported in a number of domestic animal species, including dogs, cats, pigs, horses, and cattle.[2] Ganglioneuroblastoma is differentiated from the more primitive neuroblastoma histologically by the presence of a mixed population of cells including large ganglion-like cells, small neuroblastic cells, and Schwannian stroma in varying proportions. Ganglioneuroblastoma has been reported as solitary lesions in the canine olfactory epithelium[5], brain[4], oral mucosa[6], and thorax[7].

Clinical signs will vary depending on the location of the tumor. In all of the previously cited cases, clinical signs were limited to the effects of the space occupying mass on the local tissue. This dog was presented for nonspecific lethargy, pain, and recumbency that was most likely due to severe compression of the heart and lungs. The cause of the anemia and thrombocytopenia in this case was not identified.

Immunohistochemical markers used in previous studies[4-7] demonstrate consistent staining of ganglion-like cells with neurofilament protein (NFP), but variable patterns of staining with markers such as S 100, synaptophysin, GFAP, and NSE. Case reports are few therefore a pattern of immunoreactivity is not clearly established for the dog. Studies of human neuroblastic tumors[8] report variable staining for these and other markers.

AFIP Diagnosis: Spleen; liver; and mediastinum (per contributor): Neuroblastoma with multifocal poorly differentiated ganglion cells, dog (*Canis familiaris*).

Conference Comment: Neuroblastic tumors include neuroblastoma, ganglioneuroma, and ganglioneuroblastoma. Neuroblastomas may occur in both the PNS and CNS. They are most commonly located in the adrenal medulla or in the sympathetic ganglia. Neuroblastomas in the PNS are derived from neuroectodermal cells of the neural crest and show varying degrees of differentiation toward postmitotic neuroblasts. Ganglioneuromas arise from primitive neuroepithelial cells but further differentiate towards neoplastic neurons. Those tumors exhibiting histologic features of both well-differentiated neurons and neuroblastic cells are called ganglioneuroblastomas.[2] Ganglioneuroblastomas are thought to originate from the cranial and spinal ganglia or sympathetic ganglia of the autonomic nervous system. They consist of ganglion cells, Schwann cells, and nerve fibers in variable levels of differentiation.[2]

In this case, the predominant cell type is neuroblastic. There are scattered areas containing poorly differentiated ganglion cells that lack Nissl substance. Following the conference the case was reviewed in consultation with pathologists in the AFIP Department of Soft Tissue Pathology. Their diagnosis, based on the human classification of the International Neuroblastoma Pathology Committee (INPC), was neuroblastoma (Schwannian stroma-poor), differentiating subtype, with ganglion cells. The designation "Schwannian stroma-poor" indicates neuroblastic cells forming groups and nests without or with limited Schwannian proliferation. In the "differentiating subtype," Schwannian stromal development containing mature and maturing ganglion cells comprise less than 50% of the neoplasm. We made our diagnosis based on the predominance of neuroblastic cells and absence of mature ganglion cells.

Contributor: Arkansas Livestock & Poultry Commission, #1 Natural Resources Drive, Little Rock AR 72205 www.arlpc.org

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CASE III – 5983-02 (AFIP 2841678).

Signalment: A 4-year-old Tennessee Walking horse (*Equus caballus*) gelding

History: The horse was examined in late March for severe lethargy that rapidly progressed to recumbency later that same day. The animal was euthanized late that evening based on the poor prognosis. The horse had been purchased the previous day and transported to the farm in Tennessee from Kentucky. A killed tetanus Eastern / Western encephalitis flu vaccine was administered approximately 5 days before the purchase. The owner was unaware of any previous vaccinations having been given to the horse.

Gross Pathology: There were no significant gross findings.

Laboratory Results: Rabies virus examination was negative utilizing fluorescent antibody methods. Eastern Equine Encephalitis virus was isolated in mice and cell culture from the brain. The sample was also negative for West Nile virus and positive for Eastern Equine Encephalitis viral RNA utilizing reverse transcriptase polymerase chain reaction testing.

Contributor's Morphologic Diagnosis: Brain: Meningoencephalitis, suppurative,
subacute, severe, Tennessee Walking horse, equine

Contributor's Comment: Multiple sections of brain from varying sites were submitted and feature a widespread meningoencephalitis with extensive perivascular cuffing consisting of neutrophils and mononuclear cells. Multiple supurative foci were also relatively common within portions of gray matter with scattered neuronal degeneration and necrosis being evident. Intense inflammatory foci are sometimes associated with necrosis of neuropil. A few neutrophils and mononuclear cells are present within pia-arachnoid spaces.

Eastern equine encephalitis is an alphavirus in the togavirus family that causes encephalitis in both humans and horses. The reservoir host is wild birds, where virus replicates to sufficiently high titers to facilitate vector transmission of the disease. Mosquitoes serve as the biological vector for Eastern equine encephalitis. In contrast to birds, horses and humans are "dead-end" hosts since a sufficient viremia to allow transmission does not occur. Infected horses often present with fever, anorexia, and lethargy that ultimately progresses to a range of neurological signs that include paresis, seizures, paralysis, and death. Mortality due to Eastern equine encephalitis is quite high, often approaching 90%.

Eastern equine encephalitis is sporadically seen in Tennessee, primarily in western portion of the state during the months of August and September. The horse in this case was euthanized in late March due to the infection, and defies a simple explanation since the biological vector would not yet be available. Iatrogenic transmission has been suspected in another recent case of EEE and administration of a "killed" vaccine several days prior to onset of clinical signs warrants consideration in this case. Additionally, the rapid clinical progression and the severity of inflammation seen in the brain could reflect introduction of a much larger inoculum than would be seen in association with normal vector-borne disease.

AFIP Diagnosis: Brain: Meningoencephalitis, necrotizing, neutrophilic, lymphoplasmacytic, and histiocytic, diffuse, moderate, Tennessee Walking horse (*Equus caballus*).

Conference Comment: The contributor gives an excellent review of the eastern equine encephalitis (EEE) virus. Other members of the Togaviridae family include Alphaviruses such as western equine encephalomyelitis (WEE), Venezuelan equine encephalomyelitis (VEE), Highlands J, and Semliki forest viruses, and Flaviviruses including Cache Valley virus, St. Louis encephalitis, and Japanese B encephalitis viruses.[3]

EEE, WEE, and VEE are caused by related but distinct alphaviruses. EEE and VEE are lethal in approximately 90% of cases, whereas WEE is less virulent with approximately 40% mortality in the horse. In endemically infected areas, EEE and WEE are maintained by a wild bird-mosquito (reservoir-vector) cycle, particularly in swampy or tropical areas. Avian reservoirs maintain sufficient viremia to permit infection of mosquitoes. The infection of domestic animals and humans occurs with the movement of virus from swampy areas carried by reservoirs, vectors, or both. *Culiseta* and *Culex* sp. of mosquitoes are most important in maintaining endemic

infections.

Contributor: C. C. Kord Animal Disease Laboratory, Tennessee Department of Agriculture, Nashville, TN 37204

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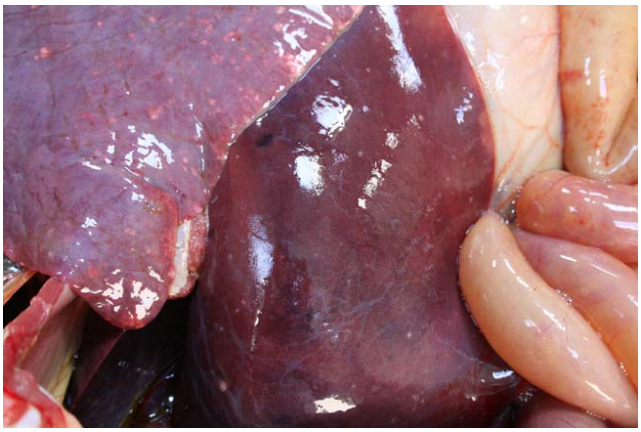


4-1 Liver and lung, horse. Random pinpoint foci of necrosis. Photograph courtesy of the University of Pennsylvania, School of Veterinary Medicine Laboratory of Pathology & Toxicology, Philadelphia, PA 19104-787 http://www.vet.upenn.edu/departments/pathobiology/pathology/

CASE IV - N0713427A (AFIP 3065826).

Signalment: Two-day-old, male Thoroughbred, *Equus caballus*, equine.

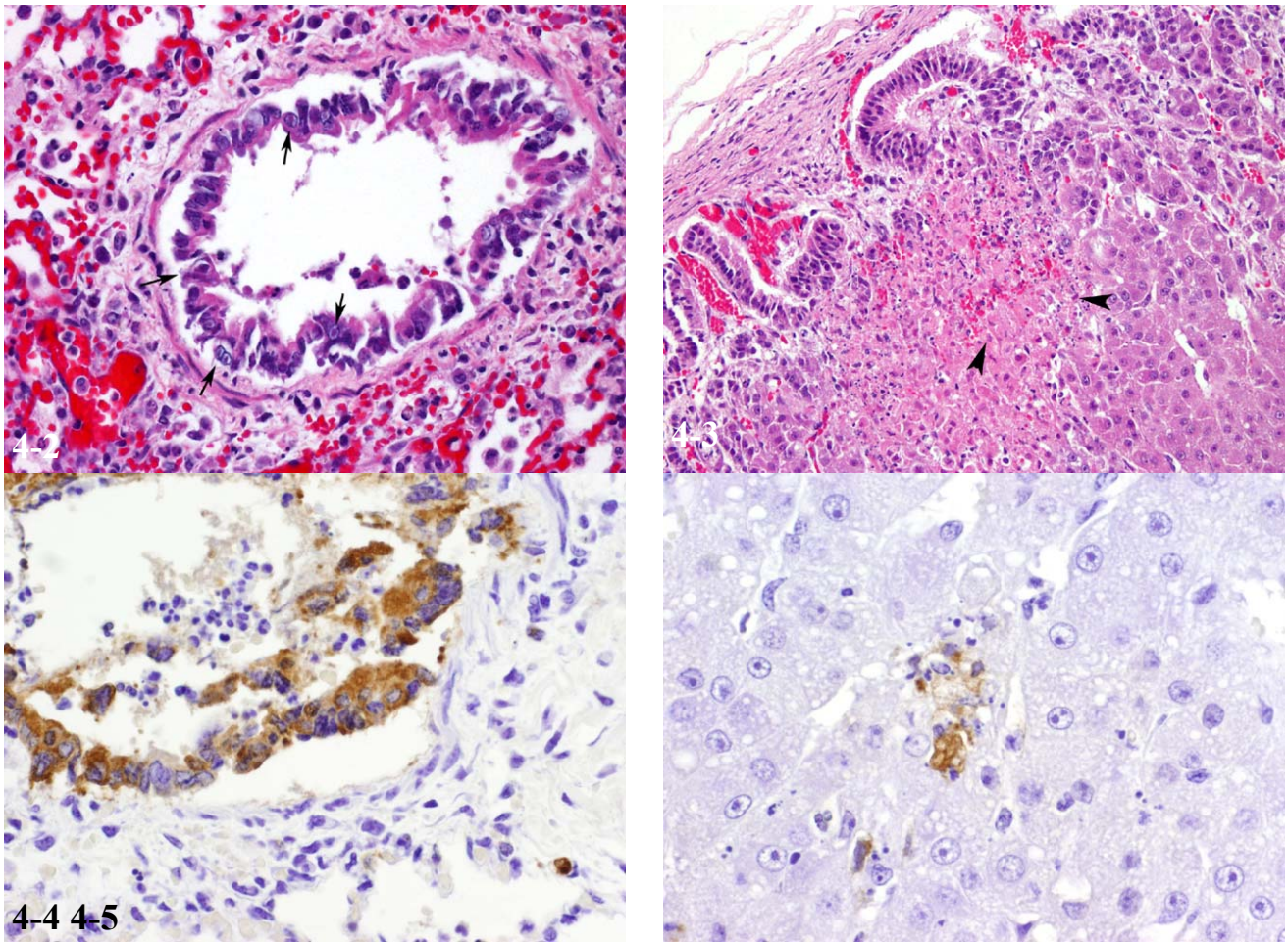
History: The colt presented at 9 hours of age with a history of premature placental separation at birth. Severe respiratory disease developed whilst the colt was hospitalized and worsened despite mechanical ventilation.

Gross Pathology: The lungs are heavy and edematous, and mottled red purple. The most cranioventral portions have numerous air filled pockets under the pleura. Scattered on the pleural surface and also on cut section are multiple white, less than 3mm diameter. The pericardium is expanded by edema. The capsular surface and parenchyma of the liver contain randomly disseminated pinpoint to less that 3mm diameter white-gray **foci** (**fig. 4-1**). The cortex of the adrenal glands contains scattered hemorrhagic foci.

Laboratory Results: Virus isolation performed on lung, liver, kidney, spleen and thymus was positive for EHV-1 and negative for EVA. FA performed for EHV-1 antigens on lung and liver was also positive, and EVA FA was negative. Aerobic culture of the lung produced no growth. Leptospira was not detected in the lung, liver, spleen and kidney by FA.

Histopathologic Description: Within the lung, there is extensive necrosis of the respiratory epithelium, predominantly affecting bronchioles but also bronchi and terminal airways. Sloughed cells admixed with necrotic debris and inflammatory cells accumulate within the lumens. Both necrotic cells and viable epithelium contain eosinophilic nuclear inclusion bodies that peripheralize the chromatin (**Cowdry type A**) (**fig. 4-2**), and there is formation of epithelial syncytia. Extending into alveoli (which are often necrotic) are accumulations of fibrin, neutrophils and macrophages. Type II pneumocytes are hyperplastic. Interlobular septa are edematous and contain an infiltrate of macrophages and neutrophils.

Scattered randomly within the adrenal cortex are areas of congestion, hemorrhage and **necrosis** (**fig. 4-3**). Immedi-



4-2 Lung, horse. Many nuclei of the bronchiolar epithelium contain an eosinophilic inclusion body which is surrounded by a clear halo and peripheralizes the chromatin (arrows). (H&E 600X)
Adrenal cortex, horse. Necrosis and hemorrhage (arrowhead). (H&E 200X)
4-3 Adrenal cortex, horse. Necrosis and hemorrhage (arrowhead). (H&E 200X)
4-4 Lung, horse. Pulmonary epithelial cells are immunohistochemically positive for equine herpesvirus type 1 antigen. Photomicrograph courtesy of the University of Pennsylvania, School of Veterinary Medicine Laboratory of Pathology & Toxicology, Philadelphia, PA 19104-787 http://www.vet.upenn.edu/departments/pathobiology/pathology/
4-5 Adrenal gland, horse. Leukocytes within the adrenal gland are immunohistochemically positive for equine herpesvirus type 1 antigen. Photomicrograph courtesy of the University of Pennsylvania, School of Veterinary Medicine Laboratory of Pathology & Toxicology, Philadelphia, PA 19104-787 http://www.vet.upenn.edu/departments/pathobiology/pathology/

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ately adjacent to the cytoclastic debris are cells that contain eosinophilic nuclear inclusion bodies that peripheralize the chromatin.
Equine herpesvirus 1 (EHV-1) antigen was detected by immunohistochemisty within the nucleus and cytoplasm of several epithelial cells and leukocytes in the **lung** (**fig. 4-4**) and **adrenal gland** (**fig. 4-5**). Appropriate positive and negative controls were used and examined and worked accordingly.

Contributor's Morphologic Diagnosis: Lung: Bronchointerstitial pneumonia, necrotizing, acute, diffuse, severe with eosinophilic nuclear inclusion bodies and epithelial syncytia.
Adrenal gland: Adrenalitis, necrotizing, acute, multifocal, moderate with eosinophilic nuclear inclusion bodies.

Contributor's Comment: Equine herpes virus 1 is an alphaherpes virus, responsible for causing abortion, peri-

natal foal mortality, respiratory disease and neurologic disease in horses.[7] Due to its direct effect on breeding and performance, and also through interference with horse movement, EHV-1 is of major economic and welfare importance in horse related industries throughout the world.[3] Pregnant mares exposed to infection abort three weeks to four months after exposure to infection. Abortion occurs anytime after five months gestation, but more commonly from nine months to term. Foals may be born alive, as in this case, but death occurs within a few days.[4,11]

The pathogenesis of EHV-1 abortion is not fully elucidated.[11] Virus is translocated from the maternal circulation to the uterus and placenta. Uterine lesions consist of vasculitis in the small arterioles of the endometrium.[11] In some cases, abortion can occur without fetal lesions or virus spread to the fetus, presumably from widespread virus-related thrombosis and infarction leading to premature placental separation and expulsion of the fetus.[14] Placental lesions in these cases consist of chorionic necrosis and fibrinoid vascular necrosis of chorionic blood vessels with fibrin thrombi.[13,14] EHV-1 has been detected in endometrial and chorionic endothelial cells in experimental and spontaneous cases of abortion by ISH and immunohistochemistry.[12,13,15]

More commonly virus spreads to the fetus. In addition to placental endothelial cells, DNA ISH also has identified EHV-1 in necrotic debris associated within infarcted microcotyledons, debris within endometrial glands and also trophoblasts, suggesting trophoblast infection results from diffusion of virus from sites of endometrial infarction and also from emptying of debris from infected glands directly onto the surface of trophoblasts.[12]

EHV-1 infection of the fetus results in well described and documented lesions. Grossly, the aborted fetus is usually fresh with subcutaneous edema and petechiae of the mucous membranes. The lungs are edematous and the trachea may contain a fibrinous cast. The liver contains miliary white foci of necrosis. The spleen may contain prominent lymphoid follicles.[2,9] Histologic lesions consist of necrosis and eosinophilic intranuclear inclusion bodies in parenchymal organs, especially the liver and adrenal glands, with minimal inflammatory cell infiltrate, lymphocytolysis in the thymus and bronchointerstitial pneumonia.[2,6] Syncytia formation in EHV-1 infection, as seen in this case, is rarely described. Previous reports include syncytia in the lungs of aborted fetuses[6] and in experimental neurologic disease.[3]

AFIP Diagnosis: 1. Lung: Pneumonia, bronchointerstitial, necrotizing, acute, multifocal, moderate, with fibrin, edema, syncytia, and eosinophilic intranuclear inclusion bodies, Thoroughbred (*Equus caballus*).
2. Adrenal gland, cortex: Necrosis, multifocal, with rare eosinophilic intranuclear inclusion bodies.

Conference Comment: The contributor includes an excellent review of EHV-1 associated abortions. EHV-1 is transmitted primarily through the respiratory system. Following an initial replication in the upper respiratory mucosal epithelium, the virus is transmitted throughout the body via mononuclear cells, primarily T-lymphocytes. Horses are latently infected for life.

There are three types of Equine Alphaherpes viruses:

EHV-1: Equine viral abortion, myeloencephalopathy, respiratory disease
EHV-3: Equine coital exanthema
EHV-4: Rhinopneumonitis virus

EHV-1 and EHV-4 both can cause abortion, although it occurs more often with EHV-1. EHV-1 and EHV-4 both can cause respiratory disease, although it is more common with EHV-4.

Slide variation includes some slides with syncytia in the adrenal cortex.

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