

The Armed Forces Institute of Pathology  
Department of Veterinary Pathology  
WEDNESDAY SLIDE CONFERENCE  
2005-2006

CONFERENCE 20  
29 March 2006

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**CASE I – 04N123 (AFIP 2983857)**

**Signalment:** 2 year old, female, Yorkshire Terrier, *Canis familiaris*, canine

**History:** Progressive (several months duration) neurological signs including head tilt to left, hindlimb ataxia, circling to left, and not able to eat without syringe feeding. There was temporary improvement with corticosteroids.

**Gross Pathology:** Grossly, no lesions are appreciated on visual examination of the unsectioned brain. However, cross sections of the fresh brain reveal some prominence of vessels in the thalamus. After fixation, coronal sections of the brain reveal multiple small cavitations and dark discoloration of the white matter on the left frontal lobe of the cerebrum as well as extensive yellow-tan discoloration in the left side of the thalamus (malacia). No other gross lesions are noted in other organs.

**Laboratory Results:** CBC, serum chemistry, bile acids, cervical radiographs, CSF tap, and CT scan revealed no obvious problems. Serologic tests for canine distemper, Toxoplasma, Neospora were negative. MRI revealed demyelination of deep white matter of left and right frontal lobes of cerebrum, left thalamus and right rostral brainstem.

**Histopathologic Description:** The left frontal lobe of the cerebrum is characterized by multifocal to coalescing patchy areas of rarefaction of the white matter. Rarefied areas contain sporadic aggregates of spindle to stellate astrocytic cells (fibrillary astrocytosis) as well as large gemistocytic astrocytes and glial cells. Vessels in the white matter are surrounded by aggregates of lymphocytes and

plasma cells. Lesions are similar but less severe in sections from the right frontal lobe, the white matter of the cerebrum adjacent to the caudate nucleus and the cortical white matter adjacent to the hippocampus. Some thalamic lesions are similar to those described in the frontal lobe. Other lesions in the thalamus include necrosis and loss of white matter with replacement by a glial scar, characterized by a mixture of fibrillary astrocytes, gemistocytes, and abundant lymphocytes, plasma cells and glial cells. Adjacent to the glial scar there are thick perivascular cuffs of lymphocytes and plasma cells.

**Contributor's Morphologic Diagnosis:** Necrotizing leukoencephalitis, multifocal, severe with astrocytosis and glial scar (thalamus), brain, canine, Yorkshire terrier.

**Contributor's Comment:** The overall lesions and distribution within the brain (primarily the cerebral white matter and mesencephalon) along with a history of progressive CNS disease and the clinical MRI findings would be compatible with necrotizing encephalitis of Yorkshire Terriers first reported in 1993 (1). The brain of this dog was negative for canine distemper and rabies via virology. Yorkshire Terriers with this disease commonly have progressive clinical lesions associated with both cerebral and brainstem lesions including lameness, falling, circling, head pressing, depression, proprioceptive deficits, strabismus, nystagmus, cranial nerve deficits, occasional seizures, paresis and ataxia (1,2,3,4). Spinal reflexes are generally normal (1,2,4).

CBC, serum chemistry and radiographs are generally normal (1,2,3,4), and fluorescent antibody testing for distemper is negative (1). CSF tap usually reveals pleocytosis of monocytic and lymphocytic cells along with increased protein (1,5). MRI and CT scan often reveal asymmetric lesions in the white matter of the cerebrum, diencephalon and mesencephalon consistent with malacic to cavitated lesions, and variable asymmetric ventriculomegaly with no mass effect (2,3,4,5).

Gross lesions include variably dilated lateral ventricles, often unilateral multifocal grayish discoloration of cerebral white matter and brainstem, variable atrophy of the cerebral hemispheres, malacia/cavitation often involving thalamus and/or cerebral white matter, and rare dilatation of third ventricle (1,2,3,4,5).

Histopathologic lesions primarily have an asymmetric cerebral white matter and brainstem (mesencephalon) distribution pattern. Lesions include necrosis and cavitation with astrocytosis, gemistocytes, fibrillary astrocytes, perivascular cuffing of lymphocytes at the periphery of necrotic areas, variable hemorrhage, some macrophages, microgliosis, neovascularization, gitter cells, sclerosis, swollen axons, and astroglial scarring (1,2,3,4,5). Occasional involvement of the dorsal funiculus of the cervical spinal cord is also seen (3). No evidence of viral, bacterial,

or protozoal diseases is detected histologically or by culture or immunohistochemical stains.

Necrotizing encephalitis of Yorkshire Terriers can be differentiated from distemper by the lack of inclusion bodies and negative virology testing and immunohistochemical stains (1,2,4,5). Another differential is granulomatous meningoencephalitis which has a prominent perivascular nodular histiocytic infiltration that is lacking in the Yorkshire Terrier encephalitis (3). Also, necrotizing encephalitis of Yorkshire Terriers can be differentiated from necrotizing encephalitis of Pugs and Maltese because those diseases have a cerebral distribution with seizures as the predominant sign and lack brainstem involvement/signs (1,3).

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**AFIP Diagnosis:** Brain, cerebrum: Leukoencephalitis, necrotizing, nonsuppurative, multifocal, chronic, severe, with gemistocytic astrogliosis, Yorkshire Terrier, canine.

**Conference Comment:** The contributor provides a thorough review of necrotizing encephalitis of Yorkshire Terriers.

Attendees described the distribution of the lesions as primarily within the white matter. Discussion focused on the profound astrocytic response to injury and the presence of reactive astrocytes with gemistocytic morphology.

The common astroglial response to CNS injury is manifest by the development of visible cytoplasm within the cell. Reactive astrocytes have a central nucleus and multiple, long, slender processes best demonstrated by glial fibrillary acidic protein (GFAP) staining. Positive immunohistochemical staining for GFAP is evident within 1-3 days and will remain prominent for approximately a month. Gemistocytes are broad and polygonal with a peripherally located nucleus, abundant, smooth, eosinophilic cytoplasm and small vacuoles sometimes located along the margins. (6) In this case, the extremely large numbers of gemistocytes suggest chronicity, and in combination with white matter sclerosis, form a glial scar.

Without the benefit of laboratory or virology testing results, attendees considered the chronic form of canine distemper the top differential. Readers are encouraged to review case 2, conference 4, 1992-1993 for a case of canine morbilliviral encephalitis (canine distemper) in a German Shepherd Dog.

As mentioned by the contributor, Pugs and Maltese dogs suffer from their own form of necrotizing encephalitis; however, their lesions vary in distribution pattern and predominantly affect gray rather than white matter. Readers are encouraged

to review case 1, conference 15, 1995-1996 for a case of necrotizing meningoencephalitis in a 5-year-old Maltese terrier.

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**CASE II – WN05/1091 (AFIP 2996889)**

**Signalment:** 8-week- old, Large White, *Sus scrofa*, pig.

**History:** An intensive piggery included five sheds into which pigs were weaned at three weeks of age, and where they remained as a batch for a further six weeks until sold. On 4 April 2005, the water supply to one of these sheds was turned off at 7 am. This shed contained 432 pigs, in 12 pens of 36 pigs each. This batch of pigs had been in the shed for 5 weeks. The temperature within the shed was usually 31°C, and the pigs were observed to be very thirsty when their water was restored at 2 pm that afternoon.

The following day, four pigs were dead and 20 were sick, lying about, depressed and head shaking. Two live pigs were submitted to the laboratory. Pig 1 was recumbent, with head trembling and frothy fluid around the mouth. When placed on its feet, it stood immobile with its head down or pressing against nearby objects.

Occasionally it had a seizure, with its head thrown back and legs paddling. Pig 2 was recumbent and comatose, with the margins of its ears dark blue and cold. Both animals were euthanized by barbiturate overdose and necropsied at 4 pm.

**Gross Pathology:** Pig 1: No gross lesions were detected.

Pig 2: The jejunum was congested and distended with watery contents.

**Histopathologic Description:** Pig 1: Forebrain: In the dorsolateral cerebral cortex, adjacent to the sulci and, in some cases, also adjacent to the gyri, there is laminar (predominantly middle laminar) necrosis. Mildest changes are of vacuolation (edema) of the neuropil with capillary endothelial hypertrophy. In extensively-affected areas a central eosinophilic zone of 'compact necrosis' containing contracted dark neurons is bordered by a zone of neuropil vacuolation. Virchow-Robin spaces around medium-sized vessels in the cortex and the leptomeninges of the sulci are infiltrated by small to large numbers of eosinophils and mononuclear leukocytes.

**Pig 2: Forebrain:** As for Pig 1 but more severe.

**Contributor's Morphologic Diagnosis:** Brain: Necrosis, cerebrocortical, eosinophilic, acute, laminar, severe.

**Contributor's Comment:** Acute water deprivation syndrome (also designated salt poisoning, or water intoxication, as disease develops after water supply is restored). The history, clinical signs and histological changes are pathognomonic for this syndrome.

In pigs with water deprivation syndrome, there is commonly marked depression and cortical blindness, leading to typical seizures that begin with a twitching of facial muscles, jerking upwards motions of the head, retropulsion to a dog-sitting position, falling to the side, and paddling with the limbs (1). The two characteristic histological changes are of laminar necrosis and eosinophilic infiltration of the cerebral cortex (1). The lesions observed in these cases are acute, and probably of no more than 26 hours duration (the time interval between water supply restoration and necropsy). The degree of eosinophilic infiltration of Virchow-Robin spaces and the leptomeninges of the cerebral cortex varies between these two pigs: more severe in Pig 1 than in Pig 2 (possibly partly due to a difference in lesion age between the two pigs). However within the same pig the numbers of eosinophils also vary amongst different areas of the cortex, possibly reflecting relative differences in age of the lesion in these areas. Mononuclear leukocytes are prominent in the perivascular cuffs in affected areas, regardless of the number of eosinophils present.

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**AFIP Diagnosis:** Brain, cerebrum: Necrosis, cortical, laminar, sub-acute, multifocally extensive, with mild eosinophilic meningoencephalitis, Large White, porcine.

**Conference Comment:** The pathophysiology of excessive dietary sodium intake without access to adequate free water is well documented in pigs. With developing hypernatremia, brain cells have a compensatory mechanism of rapidly accumulating electrolytes (sodium, potassium and chloride) to prevent the movement of intracellular fluid into the hyperosmolar cerebrospinal fluid and plasma. This mechanism prevents intracellular water from following concentration gradients that would cause cellular dehydration and, consequently, brain shrinkage. Such a situation could lead to disruption of cerebral blood supply in the calvarium and cerebral infarction, inhibition of energy pathways, or cerebral edema.

When pigs are allowed access to water again, they often overindulge and sodium concentration in extracellular fluid is lowered rapidly through dilution effect. Intracellular osmolarity is now greater and water moves into neurons along a concentration gradient. Cerebral edema and the clinical signs of salt toxicity, or water deprivation syndrome, are then manifested. In known cases of water deprivation, access to water or replacement with intravenous fluids should be gradual. Despite these measures, the prognosis is generally poor.

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**References:**

Summers BA, Cummings, de Lahunta A. *Veterinary Neuropathology*. St. Louis MO: Mosby; 1995: 254.

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**CASE III – 37236 (AFIP 2996898)**

**Signalment:** 7 yr old, female, Jersey, bovine.

**History:** Sudden onset of depression, ataxia, hypermetria and stupor progressing to bilateral blindness with constricted pupils. Death occurred 36 hours after symptomatic treatment.

**Gross Pathology:** Brain: On cut surface of thalamus there was a moderately well circumscribed approximately 20mm diameter, grey, mottled soft, depressed area. There were multiple bilateral locally extensive dark red poorly circumscribed soft foci in the cerebral cortex at the level of the midbrain and basal ganglia. There was multifocal thickening and opacity of the meninges with prominent blood vessels. Other organs: The lungs were firm, multinodular and had a yellow/red mosaic appearance on cut surface affecting 90% of the lung mass. There was marked thickening of the interlobular septae and subpleural space. The endometrium was diffusely thickened, tan and friable with few visible caruncles and no foetus.

**Histopathologic Description:** Tissues examined: Brain, spleen, liver, heart, lung, kidney, mesenteric lymph node, uterus, abomasum, small and large intestine. The most significant lesion within all of the tissues relate to the severe thromboembolic necrotising vasculitis. This is particularly well demonstrated in the submitted section of cerebrum and thalamus. In these sections there is a generalised severe vasculitis with occasional thrombi and fibrinoid necrosis of vessel walls. These are most severe where meningeal vessels run deeply into the sulci. Surrounding these are perivascular infiltrates of inflammatory cells mostly comprised of macrophages with lesser numbers of neutrophils. There are locally extensive areas of necrosis, oedema and fibrin deposition surrounding blood vessels with large numbers of 4-8  $\mu\text{m}$  in width, non-septate, non-dichotomous branching organisms (Zygomycete). These are also visible within affected vessels/thrombi with PAS staining technique.

**Contributor's Morphologic Diagnoses:**

1. Thalamus, cerebrum: Meningoencephalitis, necrotising, thromboembolic, acute, severe, locally extensive with malacia, vasculitis with fungal hyphae, Jersey, bovine, etiology Zygomycete (probably *Mortierella wolfii*).
2. Uterus: Endometritis, necrotising, thromboembolic, severe, subacute, diffuse with hyphae.
3. Lung: Interstitial Pneumonia, necrotising, thromboembolic, pyogranulomatous, severe, subacute, diffuse, lobular, with hyphae.
4. Liver. Vasculitis, necrotising, severe, acute, multifocal with perivascular hepatocyte necrosis and hyphae.
5. Spleen. Splenitis, Fibrinonecrotic, severe, acute, focal with hyphae.

**Contributor's Comment:** The aetiological agent is likely to be *Mortierella wolfii*, a common cause of bovine abortion in New Zealand. These are usually sporadic and have been associated with feeding of poorly ensiled silage. Pathogenesis is still poorly understood but infection is believed to gain entry by ingestion which results in haematogenous spread of the organism to various tissues including the lungs and placenta, the latter resulting in abortion. Approximately 20% of affected cows develop fatal thromboembolic pneumonia. Neurological symptoms are a rare presentation and there is only one unpublished report of gross lesions in the brain

with natural infection in cattle. However, fetal lesions will sometimes include encephalitis, hepatitis and splenitis.

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**AFIP Diagnosis:** Brain, cerebrum: Vasculitis, necrotizing, thromboembolic, acute, multifocal, severe, with fungal hyphae, Jersey, bovine.

**Conference Comment:** The contributor provides an excellent case of a necrotizing mycotic vasculitis with striking cerebral lesions. Conference attendees agreed that the lesions strongly resemble those of thrombotic meningoencephalitis (TME) caused by *Histophilus somni*. Only after fungal hyphae were recognized within the blood vessels and cerebrum did attendees consider another cause. Attendees considered the morphology to be most consistent with a *Zygomycete*.

Zygomycetes are ubiquitous saprophytic molds associated with water, soil, decaying matter, and substrates high in carbohydrates. Zygomycetes are opportunistic invaders; predisposing factors include antibiotic therapy, ruminal acidosis (grain overload), reflux of acidic abomasal contents and erosive viral disease such as infectious bovine rhinotracheitis (IBR) or bovine pestivirus (BVD-mucosal disease). Focal or disseminated infections can occur in cattle of all ages. Zygomycotic hyphae are described as 3-25  $\mu\text{m}$  wide, rarely septate, with thin non-parallel walls, non-dichotomous irregular branching, and focal bulbous dilatations. Frequently, hyphae are collapsed, folded and twisted and resemble a ribbon. Hyphae may be angiotropic, and perineural invasion is common.

In this case, hyphae stained very poorly with GMS, which was considered unusual in the experience of those in attendance. The hyphae are easily visualized; however, when stained with PAS.

For additional lesions caused by Zygomycetes, readers are encouraged to review conference 12, case 4, 2005-2006 for a case of mycotic rumenitis in a calf.

Readers are also encouraged to review conference 2, case 4, 1989-1990 for a case of thrombotic meningoencephalitis (TME) in a bovine. As a point of clarification, TME is formerly known as thromboembolic meningoencephalitis (TEME) and is caused by *Histophilus somni*, also formerly known as *Haemophilus somnus*.

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  5. di Menna E, Carter M.E and Cordes D.O (1972): The identification of *Mortierella wolfii* isolated from cases of abortion and pneumonia in cattle and a search for its infection source. *Research in Veterinary Science* **13**, 439-42.
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**CASE IV – 04-2379-5 (AFIP 2983612)**

**Signalment:** Three month-old pig.

**History:** Pig coming from a feeder pigs operation in which 1.5% of the animals had a posterior paralysis.

**Gross Pathology:** Gross lesions were not observed.

**Laboratory Results:** A teschovirus was isolated from the spinal cord.

**Histopathologic Description:** Microscopic lesions were confined to the central nervous system. They were located mainly in the gray matter of the medulla and ventral horns of the spinal cord. In the spinal cord sections submitted, there is a perivascular cuffing and neuropil infiltration by lymphocytes, plasma cells and histiocytes. Glial nodules mixed with lymphocytes and plasma cells, and neuronal degeneration are also present. Less severe focal lesions are sometimes present in dorsal horns.

**Contributor's Morphologic Diagnosis:** Nonsuppurative poliomyelitis

**Contributor's Comment:** Lesions of nonsuppurative polioencephalomyelitis affecting mainly the medulla and ventral horns of the spinal cord are very suggestive of a teschovirus (enterovirus) encephalomyelitis (1, 2). Lesions caused

by teschoviruses are principally located in the gray matter of the brain stem from the hypothalamus through the medulla, and ventral horns of the spinal cord (2). In the present case, the presumptive diagnosis was confirmed by the culture of a porcine teschovirus from the spinal cord. The porcine teschoviruses (formerly porcine enteroviruses CPE group I) comprise at least eleven distinct serotypes (3). The most severe form of polioencephalomyelitis caused by these viruses is called Teschen disease, and is produced by highly virulent serotype 1 strains (4). Milder forms of the disease called Talfan disease, enzootic paresis and North American encephalomyelitis are caused by less virulent serotype 1 strains or other serotypes of porcine teschoviruses (4). All forms of the disease can affect pigs of all ages.

The hemagglutinating encephalomyelitis virus (HEV) causes a nonsuppurative polioencephalomyelitis localized mainly in the pons, medulla and dorsal horns of the upper spinal cord (5). Affected pigs are usually less than three weeks of age (2, 5). The infection causes two clinical syndromes which may coexist in the same herd. One of them is characterized by anorexia and vomiting with some of the affected pigs becoming emaciated. This syndrome is called vomiting and wasting disease (2, 5). The other syndrome is characterized by nervous signs and lesions which may resemble those caused by teschoviruses. Demonstration of the viruses is essential for the differential diagnoses.

Several viruses cause a nonsuppurative encephalomyelitis in pigs, but the location of the lesions observed in the present case is very suggestive of teschovirus infection.

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**AFIP Diagnosis:** Spinal cord: Poliomyelitis and radiculoneuritis, nonsuppurative, multifocal, sub-acute, moderate with mild non-suppurative meningitis, porcine.

**Conference Comment:** Attendees noted lesions affecting predominantly the ventral horns of the spinal cord gray matter and agreed that the pattern supports the diagnosis of Teschen disease. Although not visible in all sections, attendees also noted non-suppurative inflammation affecting adjacent ventral nerve root ganglia.

As mentioned by the contributor, there are a number of viruses which can cause non-suppurative encephalomyelitis in pigs. Below is a list of viral agents which can cause inflammatory disease of the nervous system in pigs, with additional information adopted from references 1 and 9:

- Porcine Enteric Picornaviruses
  - Genus: *Teschovirus* (10 serotypes) polioencephalomyelitis associated with highly virulent PTV serotype 1 (Teschen disease); and less virulent PTV serotype 1 (Talfan disease and benign epizootic paresis)

- CNS lesions especially numerous in the ventral columns of the spinal cord, cerebellar cortex and brain stem; neurons show progressive diffuse chromatolysis, focal gliosis and perivascular lymphocytes, especially over the cerebellum
- Genus: *Enterovirus* (2 serotypes) no neurological disease has been observed
- Encephalomyocarditis Virus (family: *Picornaviridae*; genus: *Cardiovirus*): Congestion with meningitis, perivascular infiltration with mononuclear cells and some neural degeneration, nonsuppurative encephalitis and myocarditis has been described in swine fetuses with natural EMCV infection
- Porcine Adenovirus: Meningoencephalitis with perivascular infiltration and microglial nodule formation
- Pseudorabies (porcine herpesvirus 1): nonsuppurative meningoencephalitis and ganglioneuritis in the gray and white matter, also cervical and thoracic spinal cord, mononuclear perivascular cuffing and meninges thickened by mononuclear cells; intranuclear inclusion bodies in neurons, astrocytes and oligodendroglia
- African Swine Fever (family: *Asfarviridae*, genus: *Asfivirus*): Intense congestion in the meninges, choroid plexus, and encephalon
- Classical Swine Fever (family: *Flaviviridae*, genus: *Pestivirus*): Hyperemia of the brain blood vessels
- Porcine Cytomegalovirus (porcine herpesvirus 2): Hemorrhage and gliosis occur throughout the central nervous system, with a predilection for the choroid plexus, cerebellum, and olfactory lobes
- Hemagglutinating Encephalomyelitis Virus (family: *Coronaviridae*): nonsuppurative encephalomyelitis; perivascular cuffing; gliosis, neuronal degeneration, most pronounced in the gray matter of the pons Varolii, medulla oblongata, and dorsal horns of the upper spinal cord; neuritis of the peripheral sensory ganglia, particularly the trigeminal ganglia, also occurs
- Japanese Encephalitis and West Nile Viruses (family: *Flaviviridae*, genus: *Flavivirus*): Diffuse nonsuppurative encephalitis and spondylitis
- Porcine Reproductive and Respiratory Syndrome Virus (family: *Arteriviridae*): mild lymphohistiocytic leukoencephalitis or encephalitis involving the brainstem, cerebellum or cerebrum; multifocal gliosis; perivascular cuffing by lymphocytes and macrophages


- Eastern Equine Encephalomyelitis Virus (family: *Togaviridae*, genus: *Alphavirus*): encephalitis; perivascular cuffing by neutrophils early in disease followed by lymphocytes; neuronal necrosis; neuronophagia; glial nodules and malacia
- Rabies (family: *Rhabdoviridae*; genus: *Lyssavirus*): CNS changes range from mild vasculitis and focal gliosis in the brain to extensive meningoencephalitis and marked neuronal degeneration in the brain and spinal cord; Negri bodies are frequently absent in rabid swine
- Paramyxoviruses:
  - Blue eye paramyxovirus (BEP): reported only in Mexico; encephalitis and corneal opacity in piglets
  - Menangle virus: only one known outbreak; Australia 1997; mummified, autolyzed and fresh stillborn piglets with multiple congenital defects and extensive degeneration and necrosis of gray and white matter of the brain and spinal cord, intranuclear and intracytoplasmic inclusion bodies in the neurons of the cerebrum and spinal cord; nonsuppurative meningitis
  - Nipah virus: confined to Southeast Asia, zoonotic, epidemic in 1998-1999 in Malaysia resulting in significant human mortality; incases with neurological disease there is nonsuppurative meningitis rather than encephalitis

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\*Sponsored by the American Veterinary Medical Association, the American College of Veterinary Pathologists and the C. L. Davis Foundation.