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LABORATORY SERVICES

Animal Health Laboratory

AHL Newsletter

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In this issue:

Scrapie surveillance at the AHL – an update

Jim Fairles

Scrapie is a fatal, degenerative disease that affects the central nervous system of sheep and goats. It is a federally reportable disease with all suspected cases needing to be reported to CFIA. The AHL is involved in several testing programs associated with scrapie.

- **National Voluntary Scrapie Flock Certification Program** http://www.scrapiecanada.ca/home.html - The AHL is participating in this program and is able to accept samples from veterinarians and their clients.
- **National Survey of Scrapie Genetics in Canadian Purebred Sheep** - This program has recently been returned to a full user-pay system. The AHL is still able to accept samples for scrapie genotyping testing directly from veterinarians and their clients.
- **OMAFRA abattoir surveillance** – OMAFRA is conducting surveillance in Ontario through the testing of appropriate samples from sheep and goats through the meat inspection system.
- In order for CFIA to provide surveillance as comprehensively as possible in Ontario and Canada, they have also asked us to collect samples from mature sheep and goats that are submitted for post mortem, and submit for testing by CFIA. Since scrapie is a reportable disease, a positive result has consequences for the owner of the animal. We will be contacting the veterinarian and obtaining informed consent from the owner before submitting these samples to CFIA. *AHL*

Changes in this issue:

- We've moved to 4-color printing of the Newsletter - Figures will now be in color in the printed copy, as well as in the Web version, available at www.ahl.uoguelph.ca
- Replaced the microscope in the header above with the concept drawing of our new Pathobiology-AHL building - construction is well underway! Occupancy mid-2010.

We welcome your feedback for suggestions on improving the Newsletter.

Are there more diagnoses of sudden death recently?

Beverly McEwen

Would you find the answer to that or similar questions useful? We are all ready to gather and summarize those data, but we need your help to do it! "**Syndromic surveillance**" uses health-related data that precede diagnosis and signal a sufficient probability of a case or an outbreak. **All AHL submission forms have a section "body systems affected" with small check boxes to indicate the systems affected, or general problems such as sudden death or abortion.** The collection and analysis of pre-diagnostic information is a quick and efficient way to estimate clinical problems seen by veterinarians. When sufficient data are collected, trends in these data can be used to determine if there are potential health issues that need further investigation. It is particularly useful for those syndromes and diseases that do not yet have an etiology identified (remember pre-PCV2?).

So – are there more diagnoses of sudden death recently? We can't tell you without doing exhaustive and complicated data searches based on history, because depending on the commodity, **82% to 91% of the submissions do not complete the body systems affected section on the submission forms!** It doesn't cost anything extra to do this, takes little time, and will provide useful information regarding possible disease trends or health issues, in any species. *AHL*

AHL Continuing Education Session / Open House this coming April !

Do you have an interest in learning more about proper post mortem techniques? Test interpretation? Sample submission? The AHL will be holding an open house focusing on key areas of interest as determined by our practitioner survey during the 2009 OVMA Conference.

Proposed topics include:

- Practical cytology and hematology.
- Fundamentals of sample submission for bacterial culture, interpretation of results, including antimicrobial susceptibility testing.
- Investigation of suspected poisonings.
- Post mortem techniques focusing on appropriate sample submission.
- Application of molecular technology (PCR) and immunohistochemistry to diagnostic testing.

Information regarding the specific times and date will be forwarded shortly.

If you were unable to attend the OVMA conference to fill in our questionnaire, and would like to provide feedback on your particular area of interest, please contact either **Dr. Durda Slavic** at dslavic@lsd.uoguelph.ca, or **Dr. Kris Ruotsalo** at kruotsal@lsd.uoguelph.ca with your request. *AHL*

Animal Health Laboratory - Events Calendar

AHL Client Services is off to a busy start in 2009. We kicked off the year by attending the **OVMA Conference** at the end of January. Mid-February we travelled to London and the **OAVT Conference**, and we look forward to touching base with our poultry clients at the **2009 Poultry Show April 8-9 in London, Booth 507.**

Combined with clinic visits, we would like to thank all for meeting with us and providing feedback that will help in providing better service to you. Please forward any comments and ideas to info@ahl.uoguelph.ca.

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Its mission is to inform AHL clients and partners about AHL current activities, and laboratory-based animal disease events and disease trends.

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Our continued thanks to all of the non-author AHL clerical, technical, and professional staff who contribute to the generation of results reported in the AHL Newsletter.

Selected zoonotic pathogens and diseases identified at the AHL, 2008

Beverly McEwen, Durda Slavic, Davor Ojkic, Josepha DeLay, Hugh Cai, Margaret Stalker

Many new, emerging, and re-emerging diseases of people are caused by pathogens originating from animals, or are shared between people and animals. The AHL plays an important role in public health by identifying zoonotic pathogens (Tables 1 and 2). These are numerator data reliant upon submission biases to the diagnostic laboratory and cannot be regarded as population prevalence estimates. Monitoring programs are not included.

***Mycobacterium tuberculosis*, was identified in a dog submitted for necropsy and details of this case are reported in the December 2008 AHL Newsletter.** The zoonotic pathogens most frequently identified at the AHL since 1999 are *Leptospira spp.*, *Salmonella sp.*, *Streptococcus sp.*, and *Cryptosporidium sp.* Occupational exposure to pigs and horses is a risk factor for *S. suis* and *S. zooepidemicus* infections. Sporadic cases of dermatophytosis, cryptococcosis and blastomycosis are identified microbiologically and/or on cytology or histological sections.

In previous years, the numbers of isolates were tabulated, however, due to the increasing number of tests for selected pathogens, the number of cases will now be documented. For data prior to 2008, please refer to previous editions of the AHL newsletter. *AHL*

Table 1. Cases with selected zoonotic pathogens isolated and/or identified at the AHL, 2008

Agent	Bovine	Swine	Equine	Ovine	Caprine	Chicken	Turkey	Canine	Feline	Other	**2008**
Bordetella bronchiseptica		27	1					10	5	9	**52**
Campylobacter coli/ jejuni/ fetus subsp. fetus	2			5				7			**14**
Chlamydophila sp.				3	5					2	**10**
Clostridium difficile	1	7	14						1	2	**25**
Coxiella burnetii (Q fever)				2	13						**15**
Cryptosporidium sp.	127	4		5					1	7	**144**
Eastern equine encephalitis virus			10							2	**12**
Giardia sp.	10	3			1			38	3	1	**56**
Listeria monocytogenes	4	3		3	3					1	**14**
Methicillin-resistant *Staphylococcus aureus* (MRSA)			49					1		1	**51**
Mycobacterium tuberculosis								1			**1**
Rabies			1							3	**4**
Salmonella sp.	83	58	75	2	1	36	11	4	4	48	**322**
Streptobacillus moniliformis (rat bite fever)										1	**1**
Streptococcus suis	4	153			1						**158**
Streptococcus equisimilis		41	26					8		1	**76**
Streptococcus zooepidemicus	1	5	96					5		1	**108**
Toxoplasma sp.				4	2				2		**8**
West Nile virus			2							68	**70**
Yersinia enterocolitica		6									**6**

Table 2. *Leptospira spp*. seropositive cases identified at AHL, 2008, microscopic agglutination test (MAT)

***Leptospira spp*. serovar**	Bovine	Swine	Equine	Canine	Other & not specified
L. autumnalis	7	4	9	31	4
L. bratislava		5	7	8	
L .canicola	9	4		5	
L. grippotyphosa	12	5	2	47	
L. hardjo	5	3			
L. icterohaemorrhagiae	12	5	4	11	1
L. pomona	29	5	15	38	
Total	**74**	**31**	**37**	**140**	**5**

AHL Lab Reports

AVIAN/FUR/EXOTIC SPECIES

An outbreak of canarypox in an aviary following the introduction of newly acquired canaries

Marina Brash, Michael Taylor, Jan Swinton, Davor Ojkic

A canary breeder acquired a collection of canaries and placed them in a small facility with a variety of passerines including canaries and finches, a short distance away from his main aviary. The breeder routinely conducted daily chores in the main aviary first and then moved to the smaller facility that housed the new introductions where he finished feeding and cleaning for the day.

Within a few days, the newly acquired, predominantly juvenile canaries were noted to have respiratory distress, started to die and were subsequently submitted for necropsy. The birds were dehydrated and in poor to fair body condition with reduction in the amount of breast musculature and increased prominence of the keel bone (Figure 1). Numerous raised white papules were present on the face, head and neck, and in some cases were scattered over the whole body (Figure 1). White opaque fluid was noted in the tracheal lumen, and the cranial portions of the lungs were congested and edematous (Figure 2). Spleens were sometimes enlarged and dotted with small white foci. Histologically, the skin lesions consisted of hyperplastic and hypertrophied epithelium with numerous large round eosinophilic cytoplasmic viral inclusions (so-called "Bollinger bodies") which are diagnostic of poxvirus (Figures 3a & b). Proliferative tracheitis and bronchitis with the characteristic poxviral inclusions were also noted. Poxvirus was seen by direct examination of the skin lesions with transmission electron microscopy.

All canaries housed in the small holding shed died. The newly introduced canaries were suspected to be incubating the poxvirus infection when received and, as a result of the extra stress associated with moving, the clinical signs were more severe. **There was no transfer of the poxvirus to the canaries in the main aviary because the breeder had implemented heightened biosecurity** by:

- isolating the new introductions from his main breeding stock,
- feeding and cleaning the original collection first, and
- never returning to the main facility after being in the small holding shed.

Canarypox can occur in 3 forms, including dry (cutaneous), wet (diphtheritic oral/respiratory), and the systemic form, which is usually associated with high mortality (up to 100%) in a short period of time. There is no effective treatment. Poxviruses are very resistant and can survive in dried scabs in the environment for months or even years. Persistent cutaneous lesions of avian poxvirus infection have been demonstrated and there is evidence that latent infections may be reactivated by stress. Until recently, vaccination using a commercial modified-live *Canarypox virus* vaccine was the preferred method of control, however this vaccine is no longer available in North America. This leaves canary breeders quite vulnerable to *Canarypox virus* infections. The loss of this vaccine coupled with the potential for long-term environmental viral persistence and protracted or latent infections places canary breeders at risk of introducing *Canarypox virus* each time new stock is introduced into the collection.

Heightened biosecurity measures are the only means by which this risk can be reduced. Such measures include:

- Purchase new stock from reputable breeders.
- Quarantine new introductions in a separate facility for 3-4 weeks.
- Perform daily chores in the main aviary first and then move to the isolation unit.
- Do not share clothing, footwear or equipment between facilities.
- Monitor new introductions for adaptation to their new home and for any signs of illness.
- When moving new stock into the main aviary, remember that this is another stress as group dynamics need to be resolved.
- If mortality occurs in the collection shortly after introducing new birds, postmortem examination may provide valuable information about the health of the flock. *AHL*

Figures on page 5 ⟶

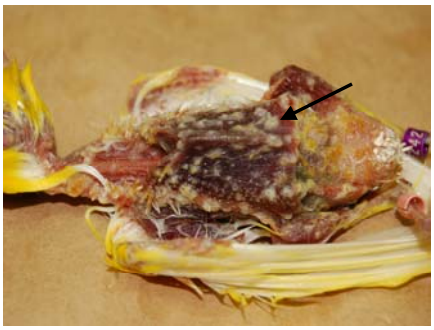

Figure 1. Multiple white plaques on head, neck and body (arrow). Canary is dehydrated, in poor body condition with prominent keel bone.

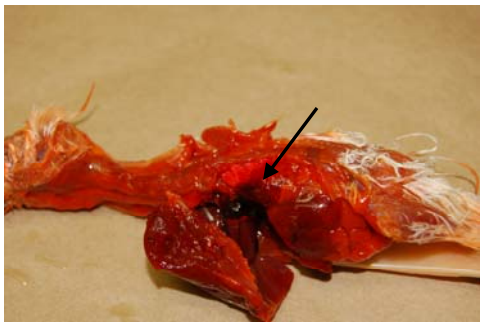

Figure 2: Cranial portion of lung is edematous and congested.

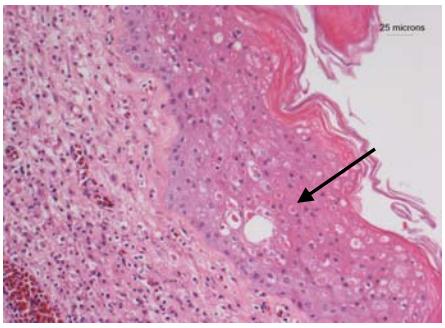

Figure 3a: Hyperplasia and hypertrophy of the epidermis with the typical large eosinophilic intracytoplasmic poxvirus inclusions.

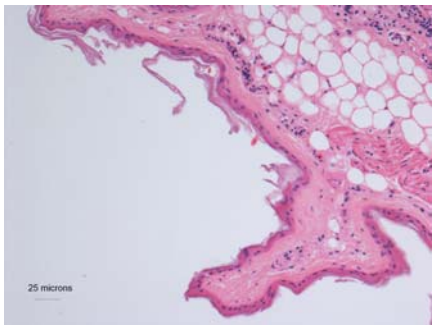

Figure 3b: Normal skin. Note the difference in the thickness of the epidermis.

Genetic characterizations of 2 low-pathogenicity avian influenza H5N1 viruses from Ontario *Yanlong Pei, Janet Swinton, Davor Ojkic, Shayan Sharif*

The genomes of 2 low-pathogenicity H5N1 avian influenza viruses (AIV) were cloned and genetically characterized. AIV A/Turkey/ON/84/1983 was isolated in Southern Ontario from commercial turkeys experiencing production problems. AIV A/Mallard/ON/499/2005 was isolated during a wild bird surveillance project from an asymptomatic duck in Eastern Ontario. Phylogenetic analysis showed that the 2 isolates sequenced in this study along with other North American isolates fall into 1 group, clustered separately from Euro-Asian isolates. Analysis of another 6 segments further demonstrated that the North American group clusters separately from the Euro-Asian isolates, which indicates that genetic exchange between Eurasian and North American isolates is limited. Individual gene comparisons demonstrated that the Ontario isolates were most similar to the viruses isolated from around the same time period and geographical area. **To our knowledge, A/Turkey/ON/84/1983 genomic sequence is the first and only available entire genomic sequence of a H5N1 AIV from domestic birds in Canada and USA.** A long deletion of 22 amino acids was identified in the stalk region of NA of A/Turkey/ON/84/1983 isolate, a characteristic mutation related to AIV adaptation to domestic birds. *AHL*

SWINE

Coccidiosis in weaned pigs *Maria T. Spinato, Andrew S. Peregrine*

Enteric coccidiosis due to *Isospora suis* infection is a well-recognized cause of diarrhea in suckling pigs. In 2008, 17 cases of porcine coccidiosis were diagnosed at the AHL. Of these, only 8 submissions identified affected pigs as being 1-3 weeks old. **The remaining 9 cases involved pigs ranging from 4-10 weeks of age.** A majority of cases consisted of fresh and formalin-fixed tissues collected by the referring veterinarian. Five cases were comprised of 2-6 live pigs delivered to the AHL for complete diagnostic examination. While diarrhea was the most commonly-listed clinical sign, unthrifty or "fading" pigs was the sole presenting complaint in 3 of the weaned pig submissions, and the primary clinical sign in 4 additional cases in this age group. Differential diagnoses and requests for ancillary tests varied, and included *Transmissible gastroenteritis virus* (TGEV), porcine rotavirus, *E. coli* and/or *Clostridium* sp. in the suckling pigs, and TGEV, rotavirus, *Porcine circovirus 2* (PCV-2) and/or post-weaning colibacillosis in the weaned pigs. Only 1 of the weaned pig submissions listed coccidiosis as a potential rule-out.

Continued on page 6 →

Coccidiosis in weaned pigs cont'd from p. 5

Coccidiosis was diagnosed by characteristic histologic lesions and the presence of intralesional protozoa in all 17 cases. Fecal examinations for oocysts were not performed. Lesions consisted of mild to subtotal villus atrophy with occasional fused villi, associated with attenuation and focal erosion of villus epithelium. Intracellular coccidial forms, consistent with *Isospora suis*, were visible within villus enterocytes. Mature oocysts were rarely described. Lamina propria was edematous and infiltrated by eosinophils, in addition to increased numbers of lymphocytes and plasma cells. In more severely-affected sections, crypts were dilated, variably lined by attenuated to hyperplastic epithelium, and contained cellular debris. Of note are the 4 cases in which coccidia were seen in intestinal sections of only 1 or 2 animals, although all of the weaned pigs submitted in each of these cases had a significant degree of villus atrophy.

Immunohistochemical staining for TGEV and porcine rotavirus latex agglutination were negative in the 2 weaned pig submissions that requested testing for enteric viruses. Enterotoxigenic strains of *E. coli* were cultured in 4 cases, and 1 of these also had *Salmonella* sp. isolated from primary culture. Two additional cases identified *Salmonella* sp. only following enrichment culture, a finding that is more suggestive of carrier status than clinical disease. Immunohistochemical staining for PCV-2 was negative in the 4 cases in which the role of this potentially immunosuppressive virus was examined; 2 of these cases were however positive for PCV-2 by PCR. **Among the 9 cases in which coccidiosis was diagnosed in weaned pigs, 7 reports considered this infection the primary cause of unthriftiness,** whereas the remaining 2 cases identified pneumonia as the more clinically relevant disease.

Enteritis in pigs is often multifactorial, and etiologic confirmation is complicated by the delay between onset of clinical signs and diagnostic testing. In some pigs with coccidiosis, diarrhea may be evident for only 1-2 days; however, villus atrophy is reported to persist for up to 14 days post infection. This is a significant period during which malabsorption of nutrients could certainly contribute to reduced growth rates. A recent study was carried out to identify the age-related prevalence of coccidia infections in 1 large swine research herd in Ontario. Based upon fecal flotation of pigs from 1-8 weeks of age, 35% of all positive samples were from pigs aged 25-40 days. As the prepatent period of *I. suis* is very short, with oocyst excretion commencing as early as 5 days post-infection and lasting approximately 5 days, most of these older pigs were likely infected after leaving the nursery. There are virtually no studies that examine the occurrence and pathogenesis of coccidiosis in weaned pigs. **It is possible therefore, that coccidiosis may be an underrecognized and contributing cause of enteritis and unthriftiness in this age group.** *AHL*

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Mundt HC, et al. *Isospora suis*: an experimental model for mammalian intestinal coccidiosis. Parasitol Res 2006; 98:167-175.

Todd C, et al. Prevalence and age-dependent occurrence of coccidia infections in pigs up to 8 weeks of age. OVC Summer Leadership and Research Program Poster Presentations, 15th August 2007, Guelph, Ontario.

HORSES

Equine abortion caused by *Arcanobacterium hippocoleae*

Durda Slavic, Tony van Dreumel

An aborted male equine fetus was submitted for diagnostic workup to the AHL. The fetus was aborted at the ninth month of gestation and was the third abortion in a herd of 40 Thoroughbred mares.

Post-mortem examination revealed the fetus was appropriately developed for the stage of gestation with no visible gross lesions. The only significant gross lesions were observed in the placenta. The amnion was cloudy and the chorion appeared thickened and leathery in some locations. Appropriate tissues were collected and submitted for bacteriology, virology and histopathology.

Microscopic examination of the spleen revealed marked lymphoid depletion and central hyalinosis of follicles. There were no lesions present in any of the other tissues examined with the exception of placenta. The proprial capillaries of the chorionic villi were markedly congested. There was marked fibrosis and edema of the stroma of the chorioallantois. Focal areas of necrosis surrounded by a mixture of inflammatory cells, mainly neutrophils, were evident in some locations. Areas of perivascular hemorrhage and fibrinocellular exudates were also present. Some of the stromal veins were thrombosed. The amnion was edematous. Special stain for *Nocardia spp.* was negative but Brown-and-Brenn Gram stains revealed large number of gram-positive rod-shaped bacteria in the exudate and on the surface of the allantois.

No *Equid herpesvirus* was detected in lung and thymus using fluorescent antibody testing. Furthermore, no virus was isolated in cell culture. **Bacterial culture of lung, stomach content and placenta, however, yielded a large number of *Arcanobacterium hippocoleae* as determined by 16S rRNA sequencing.** *A. hippocoleae* is a gram-positive rod-shaped bacterium first isolated from vaginal discharge of a horse. More recently, however, large numbers of *A. hippocolea*e were isolated from lung, stomach content and placenta of an aborted American Quarter Horse foal. In addition, necrosuppurative placentitis with large number of gram-positive bacteria was present. Continued on page 7

Equine abortion caused by *A. hippocoleae* - cont'd from p. 6

Based on these findings, **it appears that *A. hippocoleae* may play a role in equine reproductive tract disease, primarily as a placentitis.** More work, however, is required to establish the full host range of this organism and its pathogenic potential. *AHL*

References

Bemis AD, et al. Isolation of *Arcanobacterium hippocoleae* from a case of placentitis and stillbirth in a mare. J Vet Diagn Invest 2008;20:688-691.

Hoyles L, et al. *Arcanobacterium hippocoleae* sp. nov., from the vagina of a horse. Int J Sys Evol Microbiol 2002;52:617-619.

Pastern leukocytoclastic vasculitis in the horse

Margaret J. Stalker, Tony van Dreumel, Anthony Yu

Several cases of equine pastern leukocytoclastic vasculitis have been diagnosed by skin biopsy over the past year at the AHL. **Leukocytoclastic vasculitis of the pastern is a specific form of pastern dermatitis unique to horses, primarily affecting nonpigmented lower extremities of mature animals.** The syndrome is under-recognized and occurs sporadically, typically affecting a single animal on a horse farm. Lesions may be limited to a single leg, even though other legs may also lack pigment. There is no apparent breed or sex predilection, but cases usually develop during the summer months, implicating ultraviolet light in the development of this disease (although the disease is not a form of cutaneous photosensitization and is considered rather to be a **'photo-aggravated' vasculitis**). The pathogenesis is incompletely understood, but appears to involve deposition of circulating immune complexes in the walls of cutaneous vessels of the distal limbs, with subsequent development of vascular injury and corresponding thickly crusted lesions in the overlying skin.

Clinical signs of the acute phase of the disease include multiple, clearly demarcated areas of erythema, oozing and crusting often restricted to white areas of the distal limbs (Figure 1), particularly the lateral and medial aspects of hind pasterns. These lesions may progress to non-pruritic, painful areas of erosion, ulceration and crusting, often with disproportionate edema of the affected limb. The main clinical differential diagnoses for dermatitis restricted to nonpigmented skin include primary or secondary systemic photosensitivity dermatoses. Investigation of potential ingestion of primary photosensitizing plants (e.g., St. John's wort, buckwheat, or perennial rye grass), and evaluation of liver function to rule out secondary hepatogenous photosensitization (e.g., Alsike clover ingestion or pyrrolizidine alkaloid poisoning) is warranted. Differential diagnoses for dermatitis of the distal limbs that is not restricted to nonpigmented skin include primary irritant or allergic contact dermatitis, pastern folliculitis/pyoderma (e.g., *Staphylococcus* infection, dermatophilosis), chorioptic mange, dermatophytosis, *Malassezia* infection, immune-mediated dermatitis (e.g., pemphigus foliaceus) and neoplastic conditions (e.g., sarcoids). **Obtaining a complete history, thorough physical examination, skin scrapings, skin cytology and biopsy of primary skin lesions early in the course of disease development may increase the likelihood of reaching a definitive diagnosis.**

On histologic evaluation of biopsies, acute lesions include leukocytoclastic vasculitis of the small vessels of the superficial dermis with vessel wall necrosis and thrombosis, while chronic changes include vasculopathy with hyalinization and thickening of vessel walls. The overlying epidermis is often hyperplastic, eroded or ulcerated, and in chronic cases may develop papillomatosis.

Clinical management includes identifying and eliminating potential trigger factors while reducing exposure to sunlight by stabling during daylight hours or wrapping limbs, institution of immunomodulatory therapy including pentoxifylline and topical or systemic glucocorticoids, along with treatment of any concurrent secondary infections. With early identification and aggressive therapy, the prognosis for these patients is favorable. *AHL*

Reference

Akucewich L, Yu A. Equine pastern dermatitis. Compendium: Equine Edition, CE article 2. July/August 2007:214-227.

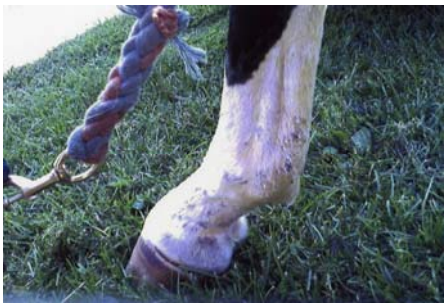

Figure 1: Note crusted lesions on nonpigmented areas of the distal leg. (Photo courtesy of Dr. L. Kouwenberg, Harwich Veterinary Clinic).

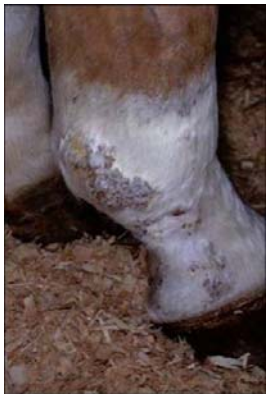

Figure 2: Note marked edema of the proximal limb. (Photo courtesy of Dr. A. Yu, OVC).

COMPANION ANIMALS

Neck pain in dogs, a 6-year retrospective

Janet L. Shapiro, Brian Binnington, Beverly McEwen

Neck pain in dogs is a subjective diagnosis based on observing an abnormal gait, and changes in demeanor when the cervical spine and para-spinal structures are palpated or manipulated. It occurs in many medical conditions related to the nervous and non-nervous soft tissues of the cranium and cervical vertebral column.

From Jan 1, 2003 to Dec 31, 2008, 26 dogs were submitted for necropsy to the AHL, Guelph and Kemptville, for which neck pain was a prominent presenting clinical sign. Dogs ranged in age from 15 wk to 12 yr, with a mean age of 5.28 yr, and a median age of 4 yr. Six of 24 dogs were mixed-breed, and 18 were purebreds, including 2 each of Yorkshire Terriers, Great Danes, German Shepherds, and 1 each of Bull Mastiff, Beagle, Dalmatian, Rhodesian Ridgeback, Wheaten Terrier, Cavalier King Charles Spaniel, Bernese Mountain Dog, Pug, Collie, Golden Retriever, Boxer, Rottweiler, and Dogue de Bordeaux; 12 were male,14 were female.

In all but 1 case, neck pain was associated with lesions of the brain and/or spinal cord. **Neoplasia** was diagnosed in 10/25 cases, either primary (9 cases - meningioma, astrocytoma, ependymoma, multilobular osteosarcoma of the skull) or metastatic (1 case-carcinoma).

Inflammation of the meninges, brain or spinal cord was diagnosed in 5/25 cases, and included granulomatous meningoencephalitis (Beagle and Pug), idiopathic meningitis, spinal myelitis and meningitis associate with filamentous bacteria, and myeloencephalitis with vasculitis consistent with Beagle pain syndrome in a mixed-breed dog.

Cervical myelopathy due to intervertebral disc disease was diagnosed in 2/25 cases.

Ischemic necrosis and hemorrhage was diagnosed in 3 cases; 1 case each of atlanto-occipital malformation and subluxation, fibrocartilagenous embolism, and DIC. **Idiopathic degenerative myelopathy** including the cervical cord was diagnosed in 2 cases.

Two interesting cases submitted to AHL-Kemptville are featured below.

A 15-wk-old mixed-breed dog was diagnosed with 1-wk clinical history of fever, depression and neck pain exacerbated by lateral flexion. Treatment included anti-inflammatory drugs and antibiotics. Eleven days after presentation, it experienced seizures and died. Necropsy revealed myeloencephalomeningitis with fibrinoid vasculitis, necrotizing fibrinoid polyarteritis, and multifocal myocardial degeneration and necrosis consistent with the syndrome referred to as "**Beagle pain syndrome**", "canine juvenile polyarteritis syndrome", "aseptic suppurative meningitis" and "steroid-responsive meningitis-arteritis". The condition is most frequently reported in beagles; however, it has been reported in other purebreds and mixed-breed dogs. Most cases occur in 6-24 mo-old dogs and clinical signs can include pyrexia, reluctance to move, neck pain, extended head and neck, or a hunched posture. No microorganisms are identified and bacteriology is negative. The arteritis occurs most commonly in the spinal meninges, coronary blood vessels and mediastinal arteries. It is thought to be immune-mediated, with some cases being steroid-responsive.

A 4 yr-old mixed breed dog was presented with a sudden onset of shifting forelimb lameness and pain on left shoulder extension that responded well to treatment with prednisone, but recurred when medication was withdrawn. Over a 9-mo period, forelimb lameness became severe, and shoulder muscles atrophied. No neurological deficits were detected. The dog was euthanized. Necropsy revealed a solid mass with a soft center in the C6/7 intervertebral space. The mass was approximately 3 times the width of the cervical intervertebral discs, and expanded the C6/7 intervertebral space to 9 mm, bulging into the spinal canal. **The mass was histologically consistent with a chordoma.** No spinal cord lesion was detected. Chordomas are rare, solitary, slow growing neoplasms of skeletal tissue found in adult humans, ferrets, cats, dogs, rats, and mink. They arise primarily within the vertebral body, where remnants of the notochord, a fetal remnant derived from mesodermal tissue, may be found. Chordomas can also originate within any vertebral body, C2 in particular, being commonly affected in humans. Extraskeletal development of a chordoma within the spinal cord is a rare manifestation of this neoplasm. In dogs, chordomas appear to have a low metastatic potential, but can be locally invasive. Literature regarding chordomas in the dog is scarce, but at least 6 other cases, 2 of which involved the cervical spine, have been reported. These involved a 7-yr-old Belgian shepherd, at C3, and a 12-yr-old Shetland sheepdog, at C6/7. *AHL*