18 2 Spring 2007

FROM THE DIRECTOR

H. Leon Thacker, DVM, PhD

Good day from ADDL. It is always great to see Spring arrive, the newness that accompanies it is invigorating. Along with spring this year, we have been greeted with news of cats and dogs becoming ill and dying from toxic feedings, the etiology of which is yet in question. As of today, we have had only a few animals submitted with suspicion of death due to ingestion of a suspected commercial diet. We have not had definitive confirmation of death due to one of the suspected diets, but we have been alert to the gross and microscopic changes reported from other laboratories who have diagnosed the toxic disorder in

question. Our toxicology laboratory has the equipment and the published procedure for identifying the purported toxic agent, but have not pursued specific identification of the compound nor received definitive positive controls. The IVMA has a well prepared website for reference to the condition that is also linked on our website; updates of findings to come in this disease instance will be posted as they become available.

We have had a major change of personnel in the ADDL. After 31 years of service to the Laboratory, Dr. Bob Everson, Head Chemist of the ADDL Toxicology section retired February 28, 2007. Bob and his services will be missed in the Laboratory. During the time of his tenure here he held several concomitant positions including Chief of Computer Services '81-'86, editor of the ADDL Annual Report '81-'87, Laboratory Safety Chairman and, most recently, ADDL Quality Manager '00-'06. A search for Dr. Everson's replacement is underway. Our assistant chemist, Dr. Christina Wilson who has been with ADDL since 1995 and who has recently completed the requirements for the PhD degree, has expressed interest in the position and has submitted application for it.

I would here offer to the users of ADDL services sincere apologies for the delayed ADDL billing for the months of February, March and April. Purdue University has prepared for some time to change to a new main computer operating system for the campus. The adjustments to the new system are yet being made and incorporated. An unavoidable consequence of the initiation of this new system was delay in sending accounts payable from ADDL for a period of three months. I apologize profusely for this inconvenience; from days of practice I can imagine how much inconvenience this has brought to many of our submitters. I and ADDL thank you for your patience.

We wish you well for the spring weather and activities; this is a wonderful time of year. We are soon to be joined by an enthusiastic and most capable graduating Purdue School of Veterinary Medicine Class of '07. To the Class: Welcome to the wonderful and exciting world of veterinary medicine; we look forward to assisting you with your diagnostic needs.

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FINAL DIAGNOSIS: Rhododoccus equi in a goat

In each issue, we will feature a case submitted to ADDL that we hope will be of interest to you. **History:** An 11-month old female Boer goat weighing 40 kg was submitted alive to the Purdue Animal Disease Diagnostic Laboratory for necropsy. The submitter reported an approximately two month history of anorexia, incoordination, and stilted

gait. Other animals on the farm were not reportedly affected. At presentation, the animal was laterally recumbent, but could stand with assistance. If left alone, she would fall back into lateral recumbency. Sensation was present in all four limbs. There was no apparent head tilt or nystagmus.

Gross examination: A tan to yellow, friable mass replaced approximately 50% of the dorsal aspect of the second thoracic vertebral body. The mass protruded into the spinal canal and compressed approximately three centimeters of the overlying spinal cord. The dura in this area was thin and red. Additional firm, white to tan, well-circumscribed nodules were present in the lung and liver. These nodules ranged in size from 2 mm to 12 mm in diameter and were lamellar on cut section. The left submandibular lymph node was enlarged. On section, the node architecture was replaced by friable, tan exudate.

Histopathologic examination: The vertebral body mass was a granuloma, composed of epithelioid macrophages and multinucleate giant cells that frequently contained large numbers of intracellular bacteria. The granuloma was surrounded by a thin layer of fibrous connective tissue. The adjacent bony trabeculae of the vertebral body were lytic and there was а concurrent suppurative osteomyelitis. The mass protruded into the spinal canal and extended slightly over the intervertebral disc. The dorsal aspect of the intervertebral disc and the physis of the vertebral body exhibited signs of degeneration. The overlying thoracic spinal cord was compressed. Many axons were swollen and surrounded by edematous myelin sheaths. Occasionally, myelin sheaths lacked axons and were infiltrated by macrophages. These axonal lesions were bilaterally symmetrical, limited to the area overlying the mass, and were most severe in the ventral funiculi. The submandibular lymph node, liver nodules, and nodules had similar morphology lung

characterized by a necrotic center with multifocal dystrophic mineralization. The central necrotic mass was surrounded by epithelioid macrophages and multinucleated giant cells that contained numerous Gram'spositive bacteria within cytoplasmic vacuoles. The nodules were encapsulated by a layer of fibrous connective tissue infiltrated with moderate numbers of neutrophils. The surrounding normal parenchyma of the organs was compressed.

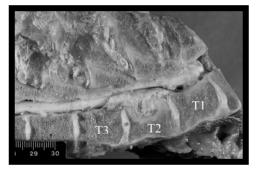
Bacteriology: An abscess from the vertebral mass, along with samples of the liver, lung, and lymph node nodules, were submitted for aerobic bacterial culture. *Rhodococcus equi* was isolated from the vertebrae, liver and lung. *Corynebacterium pseudotuberculosis* was isolated from the submandibular lymph node.

The most common cause of Comment: disseminated granulomas or abscesses in sheep and goats is Corvnebacterium pseudotuberculosis. When the disease is isolated to the lymph nodes, it is known as lymphadenitis. While caseous С. pseudotuberculosis was isolated from the lymph node, Rhodococcus equi was identified as the cause of the other granulomas. Rhodococcus equi was identified as the cause of the other granulomas. Rhodococcus equi is a common pathogen in foals where it can cause pneumonia, enteritis, abscesses, and Rhodococcus equi has also osteomyelitis. been documented to cause abscessed lymph nodes in swine, sheep, cats, cattle, and llamas. There have even been recent reports of Rhodococcus equi infecting immunosuppressed human patients with HIV/AIDS. In goats, R. equi is reported to cause disseminated abscesses or granulomas, osteomyelitis, pneumonia. pleuritis, and lymphadenitis.

R. equi is a pleomorphic. Gram's-positive. obligate intracellular bacterium most commonly residing in the soil where there is abundant avian or herbifore feces. The exact pathogenesis of *R. equi* infection in species other than foals is not completely understood. It is believed that infection may occur through ingestion or inhalation of the bacterium. Lesions in both the liver and lung of this animal would support both of these routes of entry. established, the organism Once may disseminate hematogenously throughout the bodv. Affected animals are largelv asymptomatic, as is seen with caseous lymphadenitis. until the abscesses or granulomas compress and/or destroy vital tissues (i.e. spinal cord)

Based solely on gross and microscopic morphology, it is quite difficult to differentiate between abscesses caused by R. equi or Corynebacterium pseudotuberculosis. Even with a Gram's stain, the morphology of the intracellular bacteria are similar. Bacterial culture is the most reliable method for differentiation. The high incidence of C. pseudotuberculosis and its characteristic gross appearance often discourage bacterial culture of these lesions when observed in the field. Because of this, the true prevalence of R. equi may be under-reported. Clinically, differentiation between the two organisms may be of benefit as the transmission and source of the organisms likely vary.

-by Dr. Robert Johnson, ADDL Graduate Student



A granuloma bulges from the second thoracic vertebra (T2) into the spinal canal where it compresses the spinal cord

References:

Davis et al: 1999. Disseminated *Rhodo-coccus equi* infection in two goats. Veterinary Pathology 36(4): 336-339.
Kabonga et al: 2005. Caprine vertebral osteomyelitis caused by *Rhodococcus equi*. Journal of the South African Veterinary Association. 76(3): 163-164.



Feline Inflammatory Bowel Disease

Inflammatory bowel disease (IBD) is a chronic gastrointestinal tract disorder involving the proximal or

distal portions of the gastrointestinal tract. Clinical signs may vary depending upon the site of involvement. As the pancreatic and biliary ducts are combined in cats, IBD may result in concurrent pancreatitis and cholangiohepatitis, called "triaditis". **Etiology:** The cause of IBD is not known, but thought to be the result of an appropriate immune response to a persistent, abnormal stimulus (luminal pathogen) or an abnormal, prolonged response to a normal stimulus (dietary protein and enteric microflora). Improper immune response against enteric bacteria is proposed to be an essential cofactor.

Signalment and clinical presentation: No gender, age, or breed predispositions exist for feline IBD. Mostly middle to old-aged cats are affected, but IBD has also been diagnosed with some frequency in cats less than one year old. Small intestinal IBD typically results in vomiting and weight loss whereas large intestinal IBD causes hematochezia. Other clinical signs include lethargy, anorexia and ravenous appetite. In cats with triaditis, icterus and palpable liver are seen. Severity of the signs does not correlate well with the severity of the infiltrate and the site involved.

Diagnosis: Diagnosis of IBD is by histologic exam and exclusion of other causes of gastrointestinal inflammation. Clinical differential diagnoses include:

- a) Endocrine disease (hyperthyroidism, exocrine pancreatic insufficiency)
- b) Food intolerance
- c) Infectious enteritis
- d) Chronic parasitism
- e) Neoplasia

Baseline tests: Baseline laboratory tests should include complete blood count, serum biochemistry panel, urinalysis, serum free T4 concentration, FeLV/FIV test, fecal analysis (zinc sulfate flotation, direct smear, fecal wet mount, ELISA for antigen detection). Changes in protein, liver enzymes, cholesterol, and potassium have been noted in affected cats. Biochemical evidence for pancreatic and hepatic involvement may be seen in cats with triaditis. Changes in the CBC are not specific, but hemoconcentration, leukocytosis, neutropenia, eosinophilia and basophilia have been noted.

Imaging procedures: Survey and contrast radiographs are not helpful in detecting IBD, but can be used to rule out other conditions (e.g. obstruction, intra-abdominal masses). Ultrasound can be used to choose appropriate procedures (endoscopy or exploratory laparotomy) for the collection of a biopsy specimen.

Biopsy: Histologic examination of the duodenum and colon is required for definitive diagnosis. Endoscopic or full-thickness surgical biopsies may be used for histological evaluation. It is important to obtain multiple

tissue samples from each site and from different areas (e.g. duodenum, jejunum, ileum, colon) of the alimentary tract. The limitations of endoscopy are that only mucosal biopsy can be performed (the disease could be deeper) and several areas of the digestive tract cannot be reached by this method.

Full thickness biopsy may be warranted if endoscopic biopsies are nondiagnostic and other causes of vomiting and diarrhea have been ruled out. If laparotomy is performed, biopsies of stomach, duodenum, jejunum, ileum, liver and pancreas should be obtained. Full thickness colonic biopsies should be avoided (endoscopy is the preferred method for colon).

Histopathologic findings: Increased numbers of inflammatory cells are present in the lamina propria. Typically, all cell lines are present but the type of IBD is named for the predominant cell type.

- Lymphoplasmacytic IBD is the most common form. The lamina propria is infiltrated with lymphocytes and plasma cells
- Eosinophilic IBD is the second most common form. It is characterized as diffuse or focal infiltration of mature eosinophils into one or more layers of the intestinal tract. This form of IBD may be one component of feline hypereosinophilic syndrome.
- Suppurative forms of IBD are uncommon; when present, an infectious etiology should be investigated.
- Regional granulomatous gastroenteritis is rare and typically manifests as a thickened, partially stenotic segment of bowel. This form must be differentiated from granulomatous inflammation caused by fungal disease, intestinal parasites, feline infectious peritonitis, viral infection, foreign material, toxoplasmosis, and neoplasia.

Other histologic findings include an increased number of intraepithelial lymphocytes and altered mucosal architecture (villous fusion or atrophy, edema, fibrosis, and dilation of lacteals). The submucosa is not commonly affected, except in aggressive forms (e.g., eosinophilic and regional granulomatous gastroenteritis).

Grading IBD: IBD is histologically graded into mild, moderate and severe, based on the severity of the inflammation in the biopsy sample (endoscopic and full thickness biopsy).

- Mild: Inflammation without architectural distortion
- Moderate: Inflammation with separation and distortion of glands or crypts and mild villous blunting
- Severe: Inflammation with multifocal epithelial necrosis, marked separation of glands or crypts, fibrosis, and marked villous blunting and fusion

Dilemma: A significant dilemma is to distinguish between severe lymphoplasmacytic gastroenteritis and early lymphosarcoma. In these cases, immunohistochemical staining of lymphocytes can help to determine the phenotype of the infiltrating lymphocytes (monomorphic populations of lymphocytes are suggestive of lymphosarcoma).

Treatment: Treatment is aimed at removing the antigenic source of inflammation and/or suppress the cell mediated inflammatory response in the gastrointestinal tract.

- Nutritional therapy: The diet should contain a single, highly digestible, gluten-free, novel protein (the one that animal has not been previously exposed to) and reduced amounts of food additives. High fat diets should be avoided. If colitis is present, consider high fiber diets containing either insoluble or soluble fiber
- Pharmacologic therapy: Nutritional therapy alone for moderate to severe IBD is seldom successful and most animals require pharmacological therapy.

-by Kazuhisa Miyakawa, ECFVG Student

-edited by Dr. Vimala Vemireddi, ADDL Graduate Student

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ADDL test results are available on the Internet. Call ADDL at 765-494-7440 to set up an account or log on to

<u>www.addl.purdue.edu</u>, click on Online Reports tab and follow instructions under "Request Info"



Dr. Robert Everson, ADDL's analytical chemist. retired from Purdue on February 28, 2007. A graduate of University and Cornell Purdue University, Dr.

Everson worked in the Office of the Indiana State Chemist from 1963-1975 before joining the staff at ADDL in 1975 where he started the first Toxicology Section.

Dr. Everson retired from the Naval Reserve in 1994 at the grade of Captain after completing 33 years of service.

ADDL's annual reports for Fiscal Years 2005 and 2006 are available on our web page (www.addl.purdue.edu). If you prefer a hard copy, please email me at yankovil@purdue.edu or call ADDL at 765-494-7440 and one will be sent to you.



Canine Pheochromocytoma

Pheochromocytoma is endocrine tumor derived from rare а chromaffin cells. It is the most common neoplasm of the adrenal medulla and develops often in dogs and cattle. The tumor can be unilateral or bilateral, infrequently functional and has no reported breed or sex predilection. Pheochromocytomas in dogs are usually benign, however, with potential for local invasion. Occasionally, they are malignant with metastasis to distant organs, especially the liver, regional lymph nodes or lungs. This neoplasm is considered malignant when metastasis is present in non-chromaffin cells. Affected dogs are usually older, with a mean age of 11 years. Pheochromocytomas are of neuroectodermal origin, arising from chromaffin cells, which produce and secrete catecholamines. Epinephrine and norepinephrine are the catecholamines most commonly produced, with dopamine produced less frequently. Catecholamine release in functional pheochromocytomas is usually paroxysmal. This may be attributed to changes in blood flow, chemicals, or drugs, or direct pressures on the tumor (abdominal palpation) since the tumors have no innervation.

Clinical signs associated with pheochromocytoma are often vague and intermittent, and may mimic more common disorders such as hyperadrenocortisism, diabetes mellitus. hepatic or renal disease, or other neoplasms. Clinical signs frequently observed include collapse, lethargy, weakness. anorexia, vomiting, panting, weight loss, anxietv. restlessness, polyuria, polydipsia, diarrhea, abdominal distention, hind limb edema, epistaxis, seizures or acute blindness. The clinical signs are generally associated with catecholamine excess and systemic hypertension. Elevation of blood pressure induced by sudden release of catecholamines can precipitate acute congestive heart failure, pulmonary edema, myocardial infarction, ventricular fibrillation cerebral and hemorrhage. Nonfunctional tumors can also produce clinical signs by their space occupying nature.

Pheochromocytomas occasionally result in secondary conditions including invasion and thrombosis of the caudal vena cava, aortic thromboembolism, spontaneous rupture of the tumor, paresis secondary to spinal cord compression, arrhythmias, cardiac hyper-

trophy, arteriolar sclerosis, systemic hypertension, or hyperadrenocortisism. About 15-38% of pheochromocytomas invade the caudal vena cava, causing clinical signs of ascites, hind limb edema, or distention of the caudal epigastric veins. Ultrasound and Doppler imaging can be used to identify obstruction of the caudal vena cava. Aortic thromboembolism is fairly uncommon in dogs, and occurs due to endothelial damage, vascular stasis, and hypercoagulable states. Rarely, pheochromocytomas may rupture sponand cause retroperitoneal taneously hemorrhage or periumbilical ecchymoses (Cullen's sign). Systemic hypertension occurs in 43-70% of canine patients with pheochromocytoma, and concurrent hyperadrenocortism has been found in 12% of reported cases.

Antemortem diagnosis of pheochromocytoma can be difficult because the tumors are often episodic non-functional or in nature. Abnormalities in routine laboratory testing are often non-specific, but may be useful to rule out concurrent disease. The CBC may reveal anemia (from chronic disease or blood loss) or a transient thrombocytopenia if the patient has platelet consumption from episodic hemorrhage. The leukogram often reveals leukocytosis characterized by a mature neutrophilia due to either catecholamine induced by demargination of neutrophils or necrosis/inflammation within the tumor. Serum blood chemistry occasionally reveals a mild azotemia, hypercholesterolemia, hypoalbuminemia, and elevated ALT and ALP. Abdominal radiographs and ultrasound can be used to detect abdominal masses, and thoracic radiographs should be taken to detect distant metastasis. A pneumoperitoneogram may aid in tumor visualization. Caudal abdominal venography can be useful to diagnose local tumor invasion or tumor related thrombosis, and contrast urography can identify invasion into the cranial pole of the kidney. Human medicine frequently uses advanced imaging techniques (CT, MRI, or metaiodobenzylguanidine scanning) and serum or urine catecholamine metabolite assavs to obtain a diagnosis. These methods are of limited use in veterinary medicine because of their unavailability, expense, the need for general anesthesia, and lack of reference ranges. A definitive diagnosis of pheochromocytoma can only be obtained through histopathology; therefore, the diagnosis is often obtained postmortem.

Grossly, pheochromocytomas are often large (10 cm or greater in diameter), multilobular,

firm, and encapsulated, and typically replace most of the affected adrenal gland. A thin rim of adrenal cortex may surround smaller neoplasms. Large tumors are often locally invasive into the tissues surrounding the affected adrenal gland, most notably the caudal vena cava where they may form a thrombus. Pheochromocytomas may also be located at extra-adrenal sites where they are called paragangliomas. On cross section, the tumors are often red-brown to yellow (as a result of areas of hemorrhage and necrosis), and are calcified in approximately 10% of cases. Histologically, the cells vary from small, round or polyhedral cells to large pleomorphic cells with multiple hyperchromatic nuclei. Tumor cells are often arranged in irregular cords that are separated by fine fibrovascular The cytoplasm is usually pale septae. eosinophilic and finely granular. Cytoplasmic granules can be observed by techniques using Muller, Zenker, Orth, or other dichromatecontaining fixatives. A valuable diagnostic aid is the Henle chromoreaction, which can be performed by applying Zenker's solution to the flat-cut surface of a freshly resected tumor. In a pheochromocytoma, the catecholamines will be oxidized, forming a dark brown pigment within 20 minutes of application.

The treatment of choice for canine pheochromocytoma is surgical removal. Medical therapy can be used for nonresectable or metastatic disease, or to stabilize the patient prior to surgical excision. Medical therapy is generally aimed at correcting hypertension systemic and cardiac arrhythmias. The prognosis is guarded; however, survival of up to one year has been reported in 50% of dogs with uncomplicated cases following surgical removal of the tumor. Pheochromo-cytoma should be considered in cases which have vague clinical signs and concurrent hypertension. Perhaps with more awareness of this condition, it can be diagnosed and managed more successfully. -by Dr. Kelly Baete, Class of 2004

-edited by Dr. Gopa Gopalakrishnan, ADDL Graduate Student

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Error Correction

An editorial error occurred in the Winter 2007 edition of the Diagnostic Forum in an update to a previously published article titled "Ascarid Migration in Turkey Livers". It should have read: With the advent of fenbendazole use in turkey feed (14.5 grams/ton feed for six consecutive days) ascarid migration and liver condemnations have been greatly reduced.

Purdue ADDL and Heeke ADDL will be closed on the following University holidays.

May 28	Memorial Day
July 4	Independence Day
September 3	Labor Day
November 22-23	Thanksgiving
December 24-25	Christmas
December 31-January 1	New Year's

New from Virology/Serology

PRRSV sequencing analysis on ORF5 region is now available at a cost of \$150.00/test.

Other new tests are in development – you will be notified as soon as they become available.

ADDL will return EIA charts to submitting veterinarians via Federal Express Overnight delivery. Cost will be \$12.00 for week-day delivery and \$25.00 for Saturday delivery. This request must be made at the time of submission.

Bone Marrow Fat Analysis

To Support a Diagnosis of Starvation/Malnutrition

Summary: Making a definitive diagnosis of starvation in animals is difficult because there are few quantitative measures of starvation available at post-mortem examination. The Section of Toxicology and Analytical Chemistry has developed a method which can be used to relate severely decreased bone marrow fat to clinical malnutrition.

Method: In this method, the fat content is determined from the bone marrow of the right femur using a modification of the AOAC published procedure for crude fat analysis. This procedure involves drying the sample *in vacuo* and extracting the bone marrow fat using pentane.

Average bone marrow fat content for normal adult cattle, dogs, sheep, pigs and horses:

Species	n	Mean	Range
Cattle	18	91.54%	83.21-101.22%
Dogs	15	81.53%	65.23-98.48%
Sheep	7	91.31%	83.35-95.52%
Pigs	4	81.27%	72.35-91.52%
Horses	13	85.90%	62.06-99.33%

Note: These average values should be used in conjunction with the results of a complete post-mortem evaluation for the diagnosis of malnutrition/starvation.

Cases Submitted to the ADDL:

Average bone marrow fat content in selected cases suspected of malnutrition (as of Nov, 2006)

Species	n	Mean	Range
Cattle	6	18.82%	0.47-67.11%
Sheep	1	7.89%	7.89%
Goats	5	14.12%	1.52-40.12%
Cervids	2	1.53%	0.21-2.86%
Horses	3	47.24%	37.91-64.44%

Submission: To submit a sample for analysis, submit a whole femur from the affected animal.

-by C. Wilson, Interim Chief Analytical Chemist K. Meyerholtz, Toxicology Technician

S. Hooser, Head, Toxicology Section