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Fall 2006

FROM THE DIRECTOR

H. Leon Thacker, DVM, PhD

Fall is here and time for another ADDL newsletter. A number of good things are for reporting this time including the excitement that has arisen from the return of Dr. Willie Reed to Purdue as Dean of the School of Veterinary Medicine. Willie completed his PhD in anatomic pathology at Purdue in 1982 and was pathologist in ADDL and head of our Avian Diagnostic Section until 1990 when he was recruited to Michigan State University as Director of the Michigan Animal Disease Diagnostic Laboratory. It will be good to have Dr. Reed back at Purdue.

The ADDL also has significant contribution to the veterinary pathology training program of Purdue. Over the years, several of these programs around the country have seen a decline in numbers. The number of pathology graduate students at Purdue has remained quite stable and the quality of our graduates has remained high. Three of our anatomic pathology graduate students took the qualifying examination of the American College of Veterinary Pathologists in September. All three students passed all sections of the exam. This is admirable as the overall pass rate of the ACVP exam is 40% or less. The annual meeting of the American Association of Veterinary Laboratory Diagnosticians was recently held in Minneapolis, MN. Two of our pathology students were awarded travel awards to the meeting and another of our students, Dr. Ikki Mitsui, won the graduate student poster presentation award which included a certificate and check for $500.

ADDL faculty and graduate students gave scientific presentations and contributed in other responsibilities to the Minneapolis AAVLD meeting. Dr. Roman Pogranichniy presented his research findings on a novel disease condition recognized in swine and thought to be caused by a pestivirus agent which is different and distinct from the causative agent of classical swine fever. In all likelihood, there will be further development of the significance of this agent as cause of economic losses in swine herds. During the meeting, Dr. Willie Reed's contributions to the success and activities of USDA-APHIS were recognized by receipt of USDA-APHIS administrator's award presented by Dr. Ron DeHaven, head of USDA-APHIS.

I hope this finds each of you enjoying the change of weather brought on by the fall season; it too is a beautiful time of year in Indiana. Have a good day, ADDL is here to serve you with the cutting edge diagnostics, it is our desire to provide you with the best veterinary diagnostics available.

FINAL DIAGNOSIS: Insulinoma in a cat

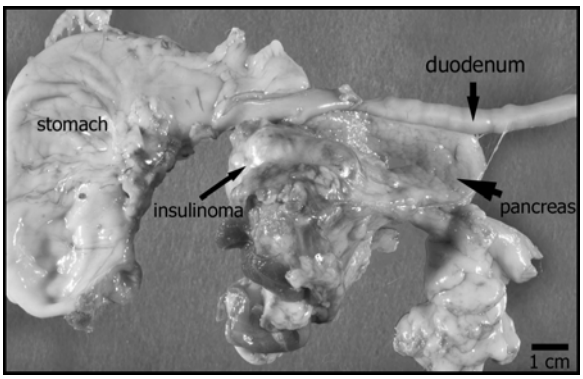
In each issue, we will feature a case submitted to ADDL that we hope will be of interest to you.

History: An 8-year-old castrated male domestic shorthair cat was presented to Purdue University Veterinary Teaching Hospital following a three week history of anorexia, weight loss, intermittent hematuria, and urinating outside of the litter box. Physical exam findings included muscle wasting, tachycardia (240 bpm), and hepatomegaly. Mildly increased liver enzymes (ALT and ALP) and markedly elevated lipase levels were noted on serum biochemistry. Complete blood count results showed a mild anemia and lymphopenia. Radiographs demonstrated urinary calculi and an enlarged liver with irregular margins. Several masses were identified in the liver and pancreas on abdominal ultrasound, along with enlarged gastric lymph nodes and mild peritoneal effusion. Cytology of liver mass aspirates was interpreted as adenocarcinoma with hepatic lipidosis. The cat was euthanized due to poor prognosis and submitted for necropsy examination to the Animal Disease Diagnostic Laboratory.

Gross examination: Subcutaneous tissue and visceral organs were icteric. The pancreas, liver, and gastric/pancreatic lymph nodes contained multifocal to coalescing masses, characterized by pale tan, firm, often umbilicated, nodules measuring up to 6 cm in diameter. The masses extended into the parenchyma of affected organs and, occasionally, had brown, friable, central necrosis on cut section.

Histopathologic examination: Masses in the pancreas, liver, and lymph nodes effaced normal tissue parenchyma and were composed of solid packets or nests of neoplastic cells separated by moderate fibrovascular stroma. The cells occasionally organized around central lumina, forming irregular acini or ductules. Features of the tumor cells included polygonal to columnar cell shape, moderate amphophilic cytoplasm, round to oval nuclei with stippled chromatin, and single distinct nucleoli. Mitotic figures were common with an index of 44 per ten high power fields (400X magnification). Hemorrhage, necrosis, and mixed inflammation were present multifocally in the masses. Portal veins contained tumor emboli, and micrometastases were identified in lung and adrenal cortex.

Immunohistochemistry: Tissue of origin could not be determined based on morphology alone in this tumor. Differential diagnoses included carcinoma (pancreatic or biliary) or malignant neuroendocrine tumor (pancreatic islet cell carcinoma). Immunohistochemical (IHC) tests utilized in this case consisted of antibodies against neuron-specific enolase, PGP 9.5, and insulin. Neuron-specific enolase and PGP 9l5 were both positive in tumor cells, supporting a diagnosis of neuroendocrine tumor, likely of islet cell origin. Insulin IHC was also positive, indicating the production of insulin in tumor cells and confirming the diagnosis as beta-cell carcinoma (insulinoma).



Gross photograph depicting the large insulinoma disrupting the pancreatic body.

Comment: Insulinoma is a tumor derived from pancreatic beta-cells, which are the insulin-producing cells in the islets of Langerhans. Insulinoma is rarely documented in cats, with only five reports identified in the literature, and is uncommon in dogs. Interestingly, three of the five cats reported with insulinoma were Siamese.

In this type of tumor, neoplastic beta-cells produce insulin and can autonomously secrete the hormone, resulting in hyperinsulinemia. Excess insulin causes hypoglycemia, which is the most consistent hematologic or serum biochemical abnormality in animals with insulinoma. The classical manifestation, best characterized in the dog, is episodic neurological signs such as weakness, ataxia, syncope, or seizures during fasting or exercise when hypoglycemia becomes most severe. After eliminating other causes of hypoglycemia, demonstration of inappropriate levels of insulin (i.e. normal or increased in the face of hypoglycemia) can help confirm the diagnosis of insulin-secreting neoplasia. Ultrasound of the pancreas can assist in identification of the mass, although the tumors are commonly small (<3 cm) at the time of diagnosis, and can be difficult to detect. Exploratory laparotomy may be warranted for definitive diagnosis.

Treatment for insulinoma can include surgery, medical management, or both. Surgery is rarely curative, and can create new challenges by inciting pancreatitis, the most common post-operative complication. Medical management consists of feeding frequent small meals, supplementing with glucocorticoids to counteract the hyperinsulinemia, and limiting excitement and exercise. The goal is to keep glucose levels from falling dangerously low, rather than maintaining normal levels at all times. Prognosis is poor due to challenging case management and high incidence of metastasis of this tumor.

The interesting features in this case include the absence of neurological signs and the inability to document hypoglycemia (cat was euglycemic on

presentation). One plausible explanation may be that , although the neoplastic cells produced insulin as indicated by positive insulin IHC staining, the hormone was either non-functional or not secreted from the tumor cells. It is also possible that hyperinsulinemia and hypoglycemia did occur, but glycose failed to reach levels low enough to manifest as neurological signs.
-by Dr. Pamela Mouser, ADDL Graduate Student

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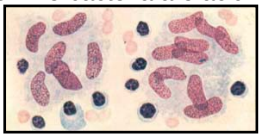
Bovine Tuberculosis

Bovine tuberculosis is an important infectious disease worldwide that threatens the lives and livelihood of those people associated with the cattle industry. Many countries, including the United States, are trying to identify and prevent the spread of this disease through testing and eradication programs. Bovine tuberculosis is a zoonotic disease that causes respiratory disease in both cattle and humans. The organism can be transmitted to humans through infected unpasteurized milk or the inhalation of bacteria at the time of slaughter..

Bovine tuberculosis is spread through aerosolized droplets or ingestion once it is established in a herd of cattle. The incubation period can range from months to years with the severity depending on the immune system of each individual animal. Infection results in chronic disease; animals typically present with clinical signs during times of increased stress or as they age. The organs that can be affected include the lungs, liver, spleen, lymph nodes and intestines. The clinical signs include moist cough, dyspnea, weight loss, anorexia, lymphadenopathy and diarrhea.

Mycobacterium bovis is the organism that causes bovine tuberculosis. The bacteria are acid fast, filamentous, curved rods. The bacteria usually enter the respiratory system of a cow and settle in the lungs. Macrophages in lungs are then responsible for phagocytizing the organism. The organism replicates intracellularly after it has been taken up by the macrophages. A granuloma or tubercle forms as the body tries to wall off the infected macrophages with fibrous tissue. The granuloma is usually 1-3 cm in diameter, yellow or gray, round and firm. On cut section, the core of the granuloma consists of dry yellow, caseous, or necrotic cellular debris. The infection can spread hematogenously to lymph nodes and other areas of the body and cause smaller, 2-3 mm in diameter, tubercles. The formation of these smaller tubercles is known as “miliary tuberculosis”. The histological lesions consist of necrotic cells in the center of the tubercle surrounded by epitheloid cells and multinucleated giant cells all encapsulated by collagenous connective tissue. The necrotic core of cells can often become calcified as the tubercle matures.



Diagnosis of bovine tuberculosis can be accomplished through gross examination, histology, acid-fast staining, culturing the bacteria, and PCR. In the live animal, interferon gamma testing, enzyme linked immunosorbent assay (ELISA) testing, and tuberculin testing can be performed. The interferon gamma test is a whole blood immunoassay that detects the presence of the cytokine produced by natural killer cells in response to mycobacterial protein. However, the most common *in vitro* test used in the United States is the tuberculin skin test.

Tuberculin skin testing is based on T-cell response to mycobacterial proteins. The caudal-fold tuberculin test is performed using 0.10 cc of a purified protein derivative. This is injected intradermally in the skin of the tail. The injection site should be examined for a reaction 72 hours post inoculation. If there is a reaction, such as a discolored raised area, the animal is classified as a responder and undergoes a comparative cervical tuberculin test. The test is more sensitive and helps to determine whether the animal is infected with *M. bovis* as opposed to *M. avium* or *M. paratuberculosis*. This test is performed by injecting 0.10 cc of a more potent purified protein derivative intradermally into the cervical skin. If the animal responds to this injection, it is classified as a reactor; the animal will be euthanized and a necropsy should be performed to determine the extent of the infection.

Currently, treatment of bovine tuberculosis is not recommended due to its infectious nature. If an animal is found to be infected, it should be culled from the herd. However, there are some preventative measures available. One way to ensure that cattle do not become infected is to eliminate any possible interaction with deer. A population of white-tailed deer was found to harbor *Mycobacterium bovis* in lower Michigan in 1995. White-tailed deer can be infected with tuberculosis infection and spread it to uninfected cattle through nose to nose contact, aerosol droplets, or indirect contact. The indirect contact is usually a result of cattle ingesting feed that has been contaminated by deer saliva. It is recommended that any feed for cattle be protected and stored away from deer. Other programs to control the deer population, such as

hunting and banning feeding, have been implemented to decrease the density of deer and the population of affected deer.

Vaccination for the prevention of bovine tuberculosis is another option that is being investigated. A vaccine was created for *M. bovis* in the 1920s by Calmette and Guerin. The BCG vaccine has undergone some scrutiny due to variable efficacy. The vaccine reduces the severity of the disease, usually allowing the bacteria to infect only a few lymph nodes, but does not prevent infection. The vaccine can also cause false-positive caudal fold skin tests, which can cause confusion when testing is performed. There are studies researching the efficacy of combining the BCG vaccine with mycobacterium protein and DNA. The goal is to develop an enhanced vaccine that can provide protection against the disease. The vaccine would be given to wildlife in an effort to prevent the spread of the disease to cattle.

Bovine tuberculosis poses a significant risk to human and herd health. The only way to be protected from the disease is through prevention. It is important to limit the exposure of the herd to other infected cattle or wildlife. Testing and eradication of infected animals is the current method of control, though additional research is currently being explored in the areas of vaccinations and other possible preventative measures.

-by Rose Paylor, Michigan State University Veterinary Student
-edited by Dr. Pam Mouser, ADDL Graduate Student

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ADDL Schedule

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

2006
November 23-24.....Thanksgiving
December 22-26.....Christmas

2007
January 1.....New Year
January 15.....Martin Luther King

ADDL test results are available on the Internet. Call ADDL at 765-494-7440 or email addl@purdue.edu to set up an account.

ADDL News

ADDL welcomed three new graduate students July 1, 2006.

Dr. Fenton Dr. Johnson Dr. Webster

Dr. Kent Fenton is a 1998 graduate of the Western College of Veterinary Medicine, Saskatoon, Saskatchewan and was most recently employed by Feedlot Health management Services, Okotoks, Alberta, Canada

Dr. Robert Johnson is a 2006 graduate of the Purdue University School of Veterinary Medicine.

Dr. Joshua Webster graduated from the Michigan State University College of Veterinary Medicine in 2003 and earned in PhD in Pathobiology and Comparative Medicine from MSU this past summer.

Graduate students **Dr. Pam Mouser** and **Dr. Dinesh Singh** were awarded travel grants by the American Association of Veterinary Laboratory Diagnosticians to attend its annual meeting in Minneapolis, MN, October, 2006. Both will be presenting scientific papers at the meeting.

Serology technicians **Cheryl Parker, Brenda Turner**, **Alice Hardebeck**, and **Cheryl Chapple** have successfully completed proficiency testing for Brucellosis, Equine Infectious Anemia, Bluetongue, Bovine leukosis. These proficiency tests are provided by the USDA's National Veterinary Services Laboratory

Our congratulations to **Drs. Kim Maratea**, **Ingrid Pardo** and **Phaedra Cole** for successfully completing the American College of Veterinary Pathologists certification qualifying exam.

Blister Beetle Poisoning:

Cantharidin toxicosis in Equines

Awareness is the best prevention

There are more than 200 species of blister beetles, each varying in size, shape and color, but the most common is genus i*Epicauta* which commonly contaminates alfalfa hay causing toxicosis in horses. Blister beetles range from 0.5-1.5 inches in length and can be black, brown, gray or even yellow and black in color. Most have non-bulging eyes that follow the contour of their head, and the head is often bent downward. The legs are long, with claws on the tarsus that have blades, teeth or spines. The most important feature found commonly among all species is that the first portion of the thorax is narrower than either the head or wing and there are six abdominal plates on the beetle's underside. Early identification in alfalfa bales may help to prevent ingestion by horses, therefore preventing cantharidin toxicosis.

Horses are particularly susceptible to cantharidin, with the minimum lethal dose 1 mg/kg of the horse's body weight. Experimentally, as little toxin as 0.45 mg/kg of body weight has been fatal. One of the most important characteristics of this toxin is that it can exert its effects in the absence of the blister beetle bodies. Also, cantharidin withstands degradation by heating or drying, making it difficult to remove the toxin even during processing of alfalfa bales or alfalfa pellets where the beetles are commonly found.

Cantharidin is odorless and colorless, so it is important to monitor alfalfa hay for early detection of the blister beetles or their parts, as a preventive measure. If gone unnoticed within the alfalfa hay, once ingested it is highly irritating, causing acantholysis of the gastrointestinal tract, especially of the esophagus and nonglandular portion of the stomach, and of vesicles in skin or mucous membranes of horses. Cantharidin acts by altering mitochondrial metabolism via its inhibition of protein phosphatase, which is involved in the control of cell proliferation, activity of membrane-associated channels and receptors, and modulation of protein kinases and phosphatases. The inhibition of protein phosphatase 2A causes an increase in permeability of endothelial cells in a time- and concentration-dependent fashion by enhancing the phosphorylation of endothelial regulating proteins.

Clinical signs begin to appear 6-8 hours after ingestion of cantharidin. The affected horse may experience colic due to the irritation and vesicle

formation in the gastrointestinal tract or because of decreased contractility, hypomotility and ileus. Also, it may be restless, irritable, sweating, have diarrhea and/or submerge its muzzle in water, (a common sign of cantharidin toxicosis). Cantharidin toxicosis also causes mucosal hemorrhage and inflammation of the urinary tract, which may manifest itself as signs of hematuria, stranguria and/or dysuria. The cardiovascular system is less frequently affected but, clinically, a horse may present with syndoronous diaphragmatic flutter (SDF). This is caused by alteration in membrane potential of the phrenic nerve and its discharge in response to electrical impulses generated during myocardial depolarization. The nervous system is less commonly affected, but an affected horse may present with aggressive behavior, seizure-like muscle activity secondary to colic, or muscle fasciculations. Most commonly, the horse presents with colic, depression, fever, dehydration, gastritis, esophagitis, and oral ulcers.

Laboratory findings can also be helpful in diagnosing cantharidin toxicosis. Serum calcium is usually markedly decreased and may remain low for prolonged periods. This hypocalcemia may be manifested clinically as SDF, tremors, or abnormal facial expressions, such as clamped jaws with lips drawn back. The serum magnesium concentration is also usually low, while creatinine kinase can increase markedly within the first 24 hours after ingestion. In acutely affected horses, urinalysis reveals markedly decreased specific gravity, often less than 1.101, and hematuria with or without myoglobinuria. Also, in acute cases, horses are frequently hyperglycemic and analysis of peritoneal fluid may reveal increased protein, greater than 4 g/dl, with normal numbers of white blood cells and fibrinogen levels. If the toxin has caused renal tubular necrosis and/or hypoproteinemia, there may be increases in serum urea nitrogen, approximately 50-70 mg/dl, and increases in creatinine, approximately 2-10 mg/dl. Total protein may be normal or increased during the first 24 hours, but then drops dramatically. Most commonly, the horse's laboratory findings include hypocalcemia, hypomagnesemia, and azotemia.

Cantharadin toxicosis can be confirmed using high pressure liquid chromatography (HPLC) to detect and quantify cantharidin in the urine of live or dead horses, and in the gastric contents, liver, or kidneys of dead horses. It is best to submit at least one pint of stomach contents or 20 ml of urine on ice for analysis.

At necropsy. erosions in the oral cavity, esophagus and stomach may be seen, as well as ulcerated to pseudomembranous enteritis. The most commonly reported gross pathologic lesions include necrosis and ulceration of the squamous lining of the distal esophagus, forestomach and urinary bladder.

Histologically, sheets of epithelium lifted from the serosal surface with normal epithelium in between can be seen, as well as hemorrhagic, ulcerative cystitis that appears as desquamation of epithelium, hyperemia, and marked hemorrhage in the bladder. Renal tubular necrosis is also visible. Occasionally, ventricular myocardial necrosis, which appears as foci or streaks in the papillary muscles and under the epicardium, may be seen both grossly and histologically.

There is no specific antidote for cantharidin toxicosis, so the treatment is usually directed at cantharadin removal, reduction, and immediate symptomatic therapy. The fatality rate can be as high as 65%, but with aggressive therapy, can be reduced to 20%. Horses with a toxic dose can die within 3-18 hours of onset, but, if they survive for 72 hours, recovery is more likely. Calcium and magnesium supplementation for prolonged periods of time is almost always indicated, but their administration should be carefully monitored and linked to serum chemistries. If the horse is exhibiting signs of gastritis, often indicated by submerging the muzzle in water repeatedly, sucralfate can be administered as a protectant. Non-steroidal anti-inflammatories (NSAIDs) can alleviate pain and protect against endotoxemia, but should be used with caution because NSAIDs are toxic to the kidney if the horse is dehydrated and if renal damage has occurred. The horse should also be stall rested for 5-10 days.

Prevention is the most effective way to avoid cantharidin toxicosis. The first cutting of hay is often free from blister beetles because the adults do not emerge until late May or June (in the southwest and southern plains, if cut before mid-May). Also, it is important not to crimp the hay during cutting so that the beetles can escape rather than get trapped and incorporated into the hay. Cutting the alfalfa at 10% or less can decrease the chance of poisoning because beetles are attracted to flowering plants. Scouting the fields for beetles and treating with a short residual insecticide before cutting helps to prevent blister beetle infestation. Sevin XLR has been has been used for prevention of infestation by blister beetles and other toxic insects. Carbaryl and parathion have also been commonly used to kill blister beetles, but have a pre-harvest waiting period that does not give them adequate residual activity to kill blister beetles that enter the field from spray time until just before harvest.

Blister beetle, or cantharidin, toxicosis is an important disease that should be considered when horses present with colic or acute death soon after ingestion of alfalfa. A definitive diagnosis may be determined if there is a history of feeding alfalfa or alfalfa-containing products, laboratory findings of hypocalcemia with or without hypomagnesemia, identification of blister beetles in hay or GI contents, and gross identification of ulcers in the distal esophagus, stomach and urinary bladder on necropsy. Confirmation using HPLC to determine the

presence and amount of cantharidin in stomach contents or urine can be used. In order to prevent cantharidin toxicosis, proper cutting of alfalfa, surveying of fields and use of an insecticide, if necessary, are recommended.
-by Cindy Echevarria, Ross Student
-edited by Dr. Steve Hooser, ADDL Toxicologist

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On the Road

Drs. Tom Bryan and **Ching Ching Wu** attended the National Poultry Improvement Plan biennial conference in Portland, OR, September, 2006

Dr. Greg Stevenson traveled to Bangkok, Thailand and Manila, Phillipines to present papers on swine diseases, September, 2006

Dr. Roman Pogranichniy traveled to the Slovak Republic to present a paper at the 2nd International Scientific Conference on Infectious and Parasitic Diseases of Animals,and continued to Hungary, Serbia, and the Czech Republic to consult with virologists.

Dr. Roman Pogranichniy attended the National Animal Health Laboratory Network Methods Technical Working Group meeting in Fort Collins, CO, September, 2006

Drs. **Steve Lenz** and **Steve Hooser** attended the Elanco-sponsored meeting on rumensin in Indianapolis, IN, August 2006

Margaret Gelhausen, Heeke ADDL Bacteriology Technician, attended the annual Association of Veterinary Microbiologists meeting in Gatlinburg, TN, August, 2006.

Dr. Steve Hooser attended the Merck Summer Research Scholars national meeting in Baton Rouge, LA, August, 2006