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Oncologists join Faculty at Veterinary Teaching Hospital

Have you recently removed a lump from one of your patients and had the pathology report indicate it was cancer? Did you wonder what the most up to date treatment is? Remember the good old days when you called Kansas State to talk to an oncologist or refer the case to the oncology department. That was great, but the oncologists left about 4 years ago. Well, the Kansas State Veterinary Teaching Hospital is ready to help you with animal cancer again.

Two oncologists are now faculty members. They are not new to Kansas State having arrived August 2007, but you may not know them. Mary Lynn Higginbotham, a native of Maryville, Missouri, completed her pre-veterinary education at Northwest Missouri State University and received her veterinary degree from the University of Missouri-Columbia. Dudley McCaw grew up in western Illinois and attended the University of Illinois for his pre-veterinary and veterinary degrees.


Mary Lynn Higginbotham


Dudley McCaw

Both completed oncology residencies at Missouri and are board certified by the American College of Veterinary Internal Medicine, specialty oncology. The oncology department at K-State also includes a resident, Kimberly Reeds (DVM Oklahoma State), a chemotherapy technician, Amy Juracek, and a radiation therapy technician Randy Juracek.

Within the teaching hospital, state-of-the-art diagnostics include computed tomography (CT), magnetic resonance imaging (MRI), and endoscopy. Mary Lynn and Dudley work closely with the surgeons, Emily Klocke and Heather Towle, to provide surgical treatment followed by chemotherapy when indicated.

Upcoming events include installation of a newer linear accelerator for radiation therapy and a new drug for the treatment of canine mast cell tumors. The linear accelerator should be fully functional this July.

Radiation therapy is indicated for several tumor types including nasal, brain, mast cell tumors, soft tissue sarcomas (e.g. fibrosarcomas, hemangiopericytomas, nerve sheath tumors), and pain palliation for bone tumors when amputation is not feasible.

The new drug for mast cell tumors is a tyrosine kinase inhibitor (similar to the human drug Gleevec) and has been shown to have a significant effect in 60% of mast cell tumors. Still awaiting FDA approval, this new drug is anticipated to be available this fall.

If you would like to consult about an oncology case, you can reach Mary Lynn or Dudley through the referral coordinator, Marsha, at 785-532-5555.

Canine leptospirosis common cause of acute renal failure

Kenneth R. Harkin, DVM, DACVIM

Although April showers may bring May flowers, they also may increase transmission of leptospirosis to our canine companions. Traditionally we expect to see more cases of leptospirosis in the fall, however, in Kansas we see cases of leptospirosis throughout the year. Leptospirosis may be one of the most under-diagnosed and under-appreciated diseases in veterinary medicine. We expect to see the classic clinical disease syndromes of acute renal failure and/or hepatic failure, but atypical presentations of leptospirosis may go undiagnosed. The importance of leptospirosis as a cause of acute renal failure cannot be emphasized enough. A 2008 study by Segev et al. published in the Journal of Veterinary Medicine identified leptospirosis as the etiology in 56 of 182 dogs with acute kidney injury. In that study, there were 50 dogs with ethylene glycol intoxication and 40 dogs with uncharacterized causes.

Leptospirosis is a spirochetal bacterial zoonosis that is found world wide, most often in wetter climates. In man, leptospirosis infections can be subclinical, self-limiting febrile disease with or without meningitis, or a severe and potentially fatal illness known as Weil's syndrome that presents as hemorrhage, renal fail-

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ure, and jaundice. As new communities encroach on areas inhabited by wildlife, the incidence of leptospirosis will continue to climb. In rural areas, pigs and cattle are the primary reservoirs of disease important for dogs. In suburban areas, rodents, raccoons, opossums, and other common wildlife are important reservoirs, and in the cities rats and raccoons are the main reservoirs.

Although we still see occasional cases of dogs infected with serovars Canicola and Icterohemorrhagiae, the most important serovars today are Grippotyphosa, Pomona, and, to a lesser degree, Bratislava. A number of additional serovars are present in the United States and have been documented in individual cases, but they don't currently appear to be a significant issue. Infection with a serovar that is not tested for and does not cross-react can lead to a false negative diagnosis by serology.

There are three classical presentations: hemorrhagic syndrome, icteric syndrome, and uremic syndrome, although the hemorrhagic syndrome is uncommon in dogs. Dogs may have one, two, or all of these syndromes and any serovar can produce any clinical picture.

Common clinical signs include arthralgia or myalgia (this may be the initial presenting complaint), vomiting and diarrhea, icterus, depression or lethargy, hematochezia or melena, intussusceptions, polyuria/polydipsia (may not be azotemic), dyspnea (from pulmonary hemorrhage or pneumonitis), oculonasal discharge, cough, and uveitis.

Common laboratory findings include mild anemia, which often confuses the diagnosis, making the clinician think that chronic renal failure is present; thrombocytopenia, which is typically mild; leukocytosis (most do not have a left shift); azotemia (elevated BUN, creatinine, phosphorous); elevated serum alkaline phosphatase (this is often dramatic with minimal increase in serum alanine transaminase when seen in combination with renal failure); elevated ALT when liver disease occurs in the absence of renal failure; and hyperbilirubinemia.

A high index of suspicion is important in the diagnosis of leptospirosis. The diagnosis can be confirmed by serology, especially when a fourfold rise in titers over a 2 to 4 week period or a single high titer with appropriate clinical signs and response to therapy are documented. Initial titers may be negative (can get early peak leptospiremia in 2 to 4 days, i.e. clinically ill, but antibodies are not evident until 10 days post-infection), titers may stay low if treated early and with appropriate antibiotics, and titers can revert to negative in 30 to 45 days if treated appropriately. Culture of blood or urine is difficult to do and requires special media and handling. Darkfield microscopy of urine has a very low yield.

Polymerase Chain Reaction (PCR) testing on blood or urine allows for very early diagnosis. This test identifies pathogenic leptospires, although it does not currently identify the serovar. This test is available through the diagnostic laboratory at Kansas State University. The PCR assay at KSU will detect most known pathogenic serovars of the genus Leptospira.

Contact the diagnostic laboratory 785-532-5650 for submission information. Additionally, information can also be found online at *www.vet.ksu.edu*, then follow the link to "Diagnostic Lab" and then to "Submission Forms". Write in "Leptospirosis PCR" or "Leptospirosis Serology". The Diagnostic Lab will establish an account for you after you submit the sample. This author currently recommends the combination of the serology and PCR to maximize the diagnostic sensitivity for leptospirosis.

The therapy for leptospirosis is straightforward. Management of dogs with acute renal failure is facilitated by the fact that most cases are in polyuric renal failure. Typically, fluid deficits are replaced over 6 to 10 hours and then a fluid rate at 2 to 3 times maintenance is continued. Of critical importance is monitoring of fluid balance, which can be achieved by monitoring central venous pressure, weight (we frequently weigh these dogs 3 to 4 times per day), and urine output (subjectively or quantitatively). Reported mortality rates range from 17–22%, and delayed and inappropriate therapy has the greatest impact on outcome.

Antibiotic therapy typically consists of initiating therapy with ampicillin (22 mg/kg IV q8h) for the acute disease until oral doxycycline is tolerated. As soon as the patient can tolerate oral medications, the patient is switched to doxycycline (5 mg/kg PO q12h or 10 mg/kg PO q24h (may use up to 20 mg/kg PO q24h)). I currently recommend a minimum of 3 weeks and a maximum of 4 weeks. This step is required to clear the leptospires from the renal tubules. For those patients that can tolerate doxycycline from the beginning, there is no need to use ampicillin. It is important to point out that first-generation cephalosporins are ineffective.

The author's current recommendation is that dogs that are considered at risk should be vaccinated yearly with a multivalent vaccine (four serovars). Unfortunately, "at risk" is a vague term. We see leptospirosis in a number of dogs who do not fit the typical risk categories of farm dogs, dogs allowed to roam, hunting dogs, dogs living in suburban areas that have high wildlife traffic in their yards, and dogs living in urban areas where rodents are a significant problem. Many of our patients are small breed dogs that live within the city limits and go no further than the confines of their own backyard.

Concerns about high reaction rates to leptospirosis vaccines were shown to be unfounded in a 2005 study by Moore et al. published in JAVMA. They showed that the rate of adverse events from the DA2PP-L vaccine given alone was no higher than for the rabies vaccine given alone. They did note that with any vaccine, smaller dogs had a greater risk and the risk increased with the number of vaccines given. Other studies have demonstrated that vaccines provide durable protection (at least 52 weeks) despite the lack of demonstrable titers and that the vaccines almost completely eliminate the carrier state.

Leptospirosis should remain on the top of the differential list for any case of acute renal failure in dogs unless another etiology can be identified and treatment for leptospirosis should be initiated pending confirmation of the diagnosis by serology and/or PCR. Leptospirosis is a common disease in dogs in Kansas and including this vaccine in the protocol should be considered, especially in areas where the disease has been identified with regularity.

Interpretation of canine and feline serological titers

William Fortney D.V.M.

Historically our profession has considered vaccination a relative innocuous medical procedure as evidenced by the fact that in most states, most vaccines can still be purchased over the counter. The standard operating procedures in veterinary practices has been "when in doubt, vaccinate the patient" believing the benefits of vaccinations always outweigh possible risks.

However, emerging awareness of acute and chronic adverse reactions to vaccines is changing the way vaccine decisions are being made in small animal practices. The current trend is to carefully assess each patient's disease risk to determine if vaccine(s) are necessary and if so, which vaccine(s) would be appropriate. The use of serological titers can be a valuable tool in making those vaccine decisions.

Vaccine-induced immunity is a multifaceted process involving antigen processing, humoral immunity, cell-mediated immunity, local immunity and cell memory. Predicting whether a patient is protected against a disease based solely on a single serum titer is a gross oversimplification of this complex immune process. But-despite the drawbacks, the use of serum titers in making logical and informed vaccine–related decisions is gaining some popularity especially in animals with histories of a previous vaccine reaction, or in those individuals prone to allergic reactions, or in those specific cases where concerns of "over vaccinations" persist.

When interpreting any serum vaccination titer, consider the following:

• Titer results will vary depending on the laboratory and testing methodology used.

• Even among the experts, there is no clear consensus on exactly what titer is considered protective for each specific disease.

• Titers measure the patient's immune status at a single point in time and may not necessarily reflect the patient's future immune status one month or one year later.

• A positive titer will protect against development of the clinical signs of that disease in the patient, however it may not prevent the patient from becoming infected or even transmitting the agent.

• Titers only measure circulating humoral antibodies and not cell-mediated immunity, local immunity, cell memory or the anamnestic response to a viral challenge. Therefore patients with low titers may, in fact, be protected. Serological titers can be useful in determining whether an animal is protected against a specific disease (positive or protective titer) at that time.

Canine

There is an excellent correlation between a "positive" titer and protection against viral challenge with canine distemper virus (CDV); canine adenovirus 1(CAV1); and canine parvovirus2 (CPV), and rabies (RV).

CDV: SN > 1: 32
CAV1: SN > 1: 32
CPV2: HI > 1: 80

Leptospirosis

Serological titers are of some benefit in determining acute and chronic Leptospirosis infections. However, because of the various serovars involved in the disease, combined with the shorter duration of vaccine-induced protection with the current vaccines, serological titers are not indicative of protection against Leptospirosis.

Feline

In cats there is an excellent correlation between a "positive" titer and protection against challenge with the feline panleukopenia virus (FPL) and rabies virus (RV) but only a good correlation with feline herpesvirus (FHV1) and feline calicivirus (FCV) protection.

FPL: HI > 1: 16
FCV: SN > 1:16
FHV1: SN > 1:16

Serological titers can be used to identify potentially susceptible animals (a negative titer).

• Because the titer only measures circulating humoral immunity, patients with negative titers may or may not be protected if challenged and therefore may be considered possible candidates for re-vaccination.

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Human tuleremia acquired from bite of a healthy cat

Jerome C. Nietfeld, D.V.M., Ph.D.
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Each year at the K-State Veterinary Diagnostic Laboratory we isolate Francisella tularensis, the cause of tularemia, from numerous cats dying of tularemia. Recently we received the body, minus the head, of a kitten to be cultured for Francisella tularensis. Several weeks previously the kitten had bitten the owner on the finger. At the time of the bite, the cat was healthy and it remained so until 10 days later when it was hit by a car and killed. The kitten's head was removed and submitted to the K-State Veterinary Diagnostic Laboratory to be tested for rabies virus. The fluorescent antibody (FA) test for rabies was negative. Subsequent to the negative rabies test, the owner was diagnosed as having tularemia and F. tularensis was isolated from the infected bite wound. The cat's body had been frozen and it was submitted to see if we could confirm that it was infected. The kitten did not have any gross changes typical of those in cats dying of tularemia. Except for broken ribs, hemorrhagic lungs, and blood in the thorax, the kitten was grossly normal. Tissues that are normally culture positive for F. tularensis from cats that die of tularemia, such as spleen, liver, lymph nodes, and lungs, were culture negative. Because the head was no longer available, the mouth could not be cultured.

At one time the most common source of tularemia for humans was handling or skinning infected rabbits. Currently, the most common source of tularemia in humans is tick bites because rabbit hunting is far less popular. Cat bites have always accounted for a small percentage of human infections. Many cases of cat-to-human transmission are from cats with clinical signs of tularemia, but there are case reports of humans developing tularemia after being bitten by healthy cats that remained healthy. In these reports, F. tularensis was isolated from both the bite wound and the mouth of the healthy cat. Evidently some cats do not become systemically infected after eating an infected rabbit, but they can carry the bacterium in their mouth for an unknown period. This is another reason why people should seek medical attention following bite wounds.

Fatal myocarditis caused by parvovirus in a litter of pups

Jerome C. Nietfeld, D.V.M., Ph.D.
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Recently we had a case where canine parvovirus type 2 (CPV-2) caused myocarditis and acute death of a litter of 11 puppies, something that has been rare since the early 1980s. The puppies ate well and were playful and apparently healthy until about 3 weeks of age when individually they began to develop acute respiratory distress followed rapidly by death. The mother and the last four puppies were brought to the K-State Veterinary Medical Teaching Hospital. One of the puppies died in route to the hospital. The other three were seemingly healthy at presentation, but over the next week each developed acute dyspnea and died soon after the onset of clinical signs. At necropsy, the lungs of all four puppies were diffusely wet and dark red, but they were not firm and they floated in the formalin. The left ventricle of the heart was mildly dilated. Microscopically, there was nonsuppurative myocarditis with degeneration and loss of myocardial fibers in all four puppies. Scattered myocytes contained basophilic intranuclear inclusions bodies. This combination of nonsuppurative mycocarditis and intranuclear inclusions is considered to be diagnostic of parvoviral myocarditis in neonatal puppies. In addition, CPV-2 was demonstrated in the heart muscles by immunohistochemical staining. Microscopic changes in the lungs consisted of lightly staining protein and foamy macrophages within alveoli, which are consistent with acute pulmonary edema secondary to acute heart failure. All other tissues were normal.

Canine parvovirus-2 appeared suddenly in dogs on several continents in 1978 and rapidly spread around the world. For the first few years the virus was associated with epizootics affecting dogs of all ages as it spread into naive populations. As immunity became widespread due to natural infection and vaccination, the virus became enzootic causing disease in young dogs as they lost maternal immunity and in scattered naive dogs. Canine parvovirus-2 replicates in cells with high mitotic activity. If infected in utero, puppies can develop generalized infection, and CPV-2 can be found in virtually all tissues. During the first 15 days of life, striated muscle cells of the heart are susceptible to infection and damage. During the initial epizootics, acute myocarditis was common in young puppies born to naive bitches. The most common clinical presentation was acute dyspena and pulmonary edema followed quickly by death of puppies 3 to 8 weeks old. Occasionally, puppies four to five months old from litters that had experienced an outbreak of parvoviral myocarditis died acutely and had lesions of chronic, nonsuppurative myocarditis. Mortality in affected litters ranged from 20 to 100%. It was rare to see both myocardial and enteric diseases in the same dog or in the same group of dogs, and this case fit that pattern in that no clinical or pathologic evidence of enteric disease was present. After just a few years, myocarditis essentially disappeared because almost all bitches were immune and their colostrum had CPV-2 specific antibodies that protect their offspring during the critical first couple of weeks. This is the first case we know of at the K-State Diagnostic Lab in at least 10 years.

Blue-green algae toxicosis in dogs

Brad DeBey and Deon van der Merwe

Two dogs that accessed a pond in August became ill. Serum chemistry revealed elevated liver enzymes. One of the dogs died and was submitted for necropsy. The suspected diagnosis was blue-green algae toxicosis because microscopic examination of the water from the pond revealed blue-green algae of the genus Microcystis. Microcystin, a toxin produced by Microcystis, was present in the water at a high concentration. Postmortem testing of tissues from the dog revealed severe hepatic necrosis, compatible with ingestion of a hepatoxin from blue-green algae. The abdomen contained unclotted blood, indicating intra-abdominal hemorrhage attributed to coagulopathy secondary to lack of production of clotting factors by the damaged liver.

Blue-green algae are cyanobacterial organisms that derive energy from chlorophyll. At least eight genera of blue-green algae have toxic properties. Those that are most commonly associated with animal poisoning include Anabena, Aphanizomenon, Oscillatoria and Microcystis. A wide range of mammals and birds are susceptible to blue-green algae toxins. Rapid growth and accumulation of algae in water is favored by high nutrient availability and warm weather. Algal overgrowth results in a green, floating scum. Dead algae tend to remain on the surface as part of the scum. Gentle constant wind from one direction may cause drifting and accumulation of the algae along the windward shore, concentrating the algae and toxins. Most toxic algal blooms occur in late summer or early fall. Animal poisoning could occur if the algae dies in the water and the liberated toxin is consumed with the water, or if the blue-green algae cells are consumed. Toxin is released when the cells reach the acidic environment of the stomach.

The toxins produced by blue-green algae that are toxic to the liver are named microcystins. Microcystis algae are clinically the most important cause of algae-associated hepatotoxicity, although other blue-green algae including Anabaena and Oscillatoria, can also produce microcystins. Microcystin-LR usually is the predominant toxin in pond water contaminated with Microcystis. Anabaena, Aphanizomenon and Oscillatoria blue-green algae can cause death by production of a neurotoxin called anatoxin. Anabaena can also produce a cholinesterase-inhibiting toxin (with a mechanism similar to toxicity by organophosphate insecticides).

If blue-green algae toxicosis is suspected, samples should be collected from the surface of the water where the algae are concentrated. The sample should be chilled and submitted to the laboratory. If it will be more than a few hours until microscopic examination, preserve a separate sample by adding one part of 10% formalin to 9 parts of the water sample. A presumptive diagnosis can be made if animals showing typical clinical signs have had access to water with algal growth and microscopic examination of the water reveals toxic algae. A confirmatory diagnosis is made by identification of typical microscopic liver lesions and identification of the toxin in water samples or gastrointestinal content by laboratory methods such as HPLC.

There are no specific antidotes to the toxins of blue-green algae. Treatment is symptomatic and supportive. Inducing vomiting, in appropriate species, may be helpful if it can be done soon after ingestion. After 1 to 2 hours, vomiting will have minimal effect on toxin absorption. Activated charcoal and cathartics help to bind toxins in the gut and reduce absorption. Bathing may be beneficial if the skin and hair are contaminated with algae. Animals should be monitored for liver function and, if needed, treated aggressively with fluids and corticosteroids to support liver function and prevent shock. Neurologic symptoms may require seizure control and/or respiratory support. Anabaena poisoning may require atropine treatment.

Alternative water must be supplied and animals must be prevented from further access to the suspected toxic water source. Algaecides, such as copper sulfate, may be used to prevent and treat algal blooms under appropriate conditions. After treatment of algal blooms, the water may remain toxic for several days. The prognosis is poor to grave in animals with signs of severe liver failure.

Acute respiratory syndrome of alpacas

Jerome Nietfeld, D.V.M., Ph.D.

Since the early summer of 2007 large numbers of alpaca farms have experienced outbreaks of acute respiratory disease that has been termed acute respiratory syndrome (ARS) or Snots. The syndrome began on the east coast and has spread to the west coast. Many people believe that alpaca shows and sales have been important in the spread of this new syndrome. Clinically and epidemiologically the syndrome appears to be infectious, and most people experienced with the disease believe it to be caused by a virus.

The reported clinical signs are nasal discharge, coughing, sneezing, open mouth labored breathing, flared nostrils, and hic-up sounds.[1] Approximately 50% of exposed alpacas do not become sick. About 40% become sick but respond well to treatment and approximately 8% become more severely ill and need aggressive treatment, but recover.

To date about 2% of the cases have been fatal. At the recent annual meeting of the American Association of Veterinary Laboratory Diagnosticians (AAVLD) there was a presentation describing the pathology in eight alpacas: six pregnant females, one adult male, and one cria.[2]

Grossly, there was pulmonary congestion and edema with pleural effusions. Microscopically, there was fibrinous interstitial pneumonia and, in some cases, vasculitis and inflammation. No significant bacteria were isolated and all viral tests were negative.

In a second presentation it was announced that a coronavirus was isolated from the lung of an affected alpaca.[3] The coronavirus is genetically different from previous alpaca origin coronaviruses. It has not been proven that the new coronavirus is the cause of alpaca ARS, but alpacas from farms that have had an outbreak of ARS are 50 times more likely to be serologically positive for antibody to the new coronavirus. Work continues to determine the role of this new virus in alpaca ARS

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and to determine whether other agents are involved.

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Continuing Education

May 27-28, 2009
International Conference on the Use of Antimicrobials in Cattle Production

June 7-10, 2009
71st Annual Conference for Veterinarians
KVMA Veterinary Trade Show
Heartworm University

August 7
3rd Annual Camelid Health and Management Conference

August 15-18
23rd Meeting of the American Society for Rickettsiology

October 10-11
16th Annual Exotic Animal Medicine Conference

October 30-31
Veterinary Career Opportunities Workshop

December 11
2nd Annual Small Ruminant Conference

For complete, up-to-date conference information visit our Web site at: *www.vet.ksu.edu* and click on Continuing Education.

Upcoming Events

June 10-13
K-State Animal Science Leadership Academy, Manhattan

June 5
KSU Youth Horse Judging Camp – Beginning Section, Manhattan

June 8-9
KSU Youth Horse Judging Camp – Advanced Section, Manhattan

June 16-18
Developing and Implementing Your Company's HACCP Plan, Manhattan

"Champion" Livestock Judging Camp A, Manhattan

June 19-21
"Champion" Livestock Judging Camp B, Manhattan

June 22-24
"Champion" Livestock Judging Camp C, Manhattan

July 10-11
2009 Dr. Bob Hines Swine Classic, Manhattan

August 13
K-State Beef Conference, Manhattan

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