



Newsletter

Volume 5, Issue 1

March 2000

Quarterly Publication of the Arizona Veterinary Diagnostic Laboratory

From the Director:

We are often reminded that “how” we report results is just as important as the results obtained. We occasionally have some difficulties and we also are faced with increasing options with regard to reporting information. We are always happy to report results by telephone if that service is requested. We do not do so routinely because some veterinarians prefer that we communicate by other methods as a means of saving telephone time for the veterinarian or veterinarian’s staff. Do not hesitate to check the box that requests a telephone response on our submission form but understand that we will not respond by telephone unless requested, or if we have questions about history or other aspects of the submission. Our next means of communication is fax transmission. This is a fairly efficient method but is slightly slower because it requires typing and proofing the document before it can be sent. Reports are sent by fax without phone calls if the report is complete on the same day. Letters are the final backup and are sent on all cases. Problems arising through the use of these communication methods mentioned are usually the direct result of errors on our part. Our administrative staff does their best to keep these types of mistake to a minimum. Other concerns result from loss of phone messages left in veterinarians offices or on answering machines. It is also unfortunate that some fax transmissions simply do not find their destination. Even letters are sometimes misfiled and do not reach the individual who is concerned. Our diagnosticians attempt to respond to a veterinarian in all instances even if the case is billed to the owner. The laboratory is in the process of trying to upgrade our computer capabilities and hoping that we will ultimately be able to handle data more efficiently, and provide some Internet access to test results. You will be informed as we progress. The bottom line is that we try very hard to communicate in a timely fashion. If you feel that there is a problem with timeliness or with the way we communicate information, please give me a call.

Robert D. Glock, Director

Group G Streptococcal Infections in Animals

Human streptococcal infections by group A streptococci have reemerged in recent years as an important pathogenic entity. There has been a significant increase in the number of reports of severe group A streptococcal infections, particularly those associated with a toxic shock-like syndrome. Necrotizing fasciitis (infections by the “flesh eating bacteria”) are probably one of the better-known examples of this kind of infection.

Although group A streptococci (*S. pyogenes*) are human pathogens, group G streptococci are fast becoming their animal counterpart. We are seeing more of these infections in dogs and cats, including more “classical” infections as well as the infections associated with toxic shock and very often “sudden death”. The classical infections include abscesses, skin infections, mastitis, venereal infections and reproductive disease, abortions, and neonatal septicemias. The largest increase has been, however, in septicemias of adult animals and necrotizing fasciitis, conditions most often associated with toxic shock and a rapid course.

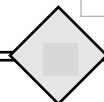
We have also isolated group G streptococci from exotic species (septicemia in an adult Giant Anteater) and wildlife (necrotic fasciitis in a raccoon). The literature reports a wide range of severe human infections produced by these organisms. Very little is known however, about the epidemiology

of these infections, the potential for cross-species infections, or the reasons for the increase in the incidence of clinical disease. The limitations of the traditional phenotypic criteria (biochemical reactions, serologic groups) to characterize and classify streptococcal strains is one of the major reasons for the paucity of our knowledge in these areas. The application of modern molecular tools to the typing of isolates and the characterization of virulence genes now allows accurate and pathogenetic studies. Those molecular tools are available in our department and we would like to apply them to the study of streptococcal infections in companion animals in Arizona, and to develop diagnostic tools of clinical value. Help us by referring cases of suspected streptococcal infections to allow us to build a representative library of isolates and accumulate epidemiological data. Call us at 520-621-2356 for any questions on submissions or to discuss specific cases.

Carlos Reggiardo, DVM, PhD

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Diagnostic Update

The following are selected samples of cases submitted to the AzVDL during the winter months of December, January, and February.

Bovine



A crossbred heifer was found dead in a feedlot pen. The gross necropsy revealed anteroventral consolidation of the lungs, mediastinal hemorrhage, hemorrhage in the papillary muscles of the heart, foci of hemorrhage in the brainstem and fibrin in multiple joints. Histologic lesions in the brain were compatible with **thromboembolic meningoencephalitis** due to *Haemophilus somnus* infection. Pneumonia, myocarditis and fibrinous arthritis were also compatible with this infection.

Steers (approximately 400 lbs.) originally from Mexico were processed in a feedlot, held for 10 days then placed on pasture. Over a one-month period the owner lost 17 animals from an illness characterized by progressive difficulty in walking, followed by recumbency and inability to rise. The animals continued to eat but had to “creep” to the feed bunk. At necropsy, two of the steers had severe, multifocal, fibrinous polyarthritis with periarticular edema and severe broncho pneumonia with abscesses. *Mycoplasma* sp. was isolated from the joints and lungs. The isolate is currently being typed by PCR but is probably *Mycoplasma bovis*. This disease appears to be increasing in frequency in feedlot cattle. Young calves are most commonly affected. The disease usually develops in multiple animals within a few weeks of comingling in the feedlot. Reported morbidity ranges are from 18-85% and mortality rates are from 3-50%. The disease also occurs in dairy calves at dairies with ongoing mycoplasmal mastitis.

An 800-cow dairy experienced an abortion storm in cows pregnant 180-220 days. The cows were clinically normal. The fetuses showed no gross lesions but contained microscopic lesions of encephalitis, myocarditis and myositis consistent with *Neospora caninum* infection. Dogs have now been identified as the definitive host of this parasite, shedding oocytes in their feces. Contamination of feedstuffs fed to cattle is the route of transmission to the cow. On this dairy, dogs were allowed access to the stored feed area.

Cows, steers and heifers grazing alfalfa pastures in central Arizona developed severe dermatitis, skin necrosis, crusting and peeling of unpigmented skin with a sharp line of demarcation with pigmented skin areas. The unpigmented conjunctival membranes of the eyes were also inflamed. **Photosensitization** was suspected. Skin biopsy specimens were supportive of that diagnosis. Serum chemistry analysis revealed no evidence of hepatic injury in these animals and suggests a primary photosensitization process. Alfalfa is periodically involved in cases of photosensitization in live-

stock but the component of the plant responsible for this activity has not been identified. This year, photosensitization has been seen in Arizona horses, cattle and sheep. The horses were consuming Arizona grown baled hay. The cattle and sheep cases were all on alfalfa pasture.

A hyperemic abomasum containing lots of sand and rocks plus inflamed intestines were found at necropsy of a five-year old Holstein cow that died suddenly. The diagnosis was *E. coli septicemia* based on the gross lesions and the inflammatory lesions observed in the kidney, heart and small intestine. *Escherichia coli* with very similar appearance and sensitivity patterns was isolated from multiple tissues. Older animals are generally considered to be resistant to *E. coli* infections but it appears that some increased susceptibility may have precipitated a disseminated infection in this cow.

Botulism due to *Cl. botulinum* type C was diagnosed in a group of 800# steers that exhibited posterior weakness progressing to paralysis. The steers had been recently castrated by banding. The diagnosis was confirmed by detection and identification of type C toxin in the serum of an affected steer by the mouse protection test. Botulism as a result of wound infections in humans or contamination of necrotic lesions in horses has been well documented. The present case illustrates the possibility of similar cases in cattle.

Equine



Eight horses in a herd of 45 developed chronic gingivitis that was unresponsive to treatment. A biopsy of one of the horses revealed pyogranulomatous gingivitis with intralesional plant material. Prior exposure to hay containing sharp **grass awns** was suspected as the cause.

A seven-year-old Quarterhorse gelding had a two-month history of cutaneous nodules developing on the trunk. Hematology and blood chemistry results were unremarkable. A biopsy of the masses revealed cutaneous amyloidosis with an infiltrating population of lymphoid cells. Mild pleomorphism and occasional mitoses were seen. Immunohistochemical stains demonstrated that the lymphoid cell population was entirely composed of T-lymphocytes. A diagnosis of **cutaneous amyloidosis and probable T-cell lymphoma** was made.

A one and one-half year old Thoroughbred colt had a 10-day history of illness, which began as pneumonia and stiffness. The horse developed progressive depression, head pressing and blindness. A CSF tap contained 4000 cells/ul consisting largely of neutrophils. Bacteria were possibly present. Treatment included antibiotics, dexamethasone, DMSO, orgotein and vitamin E. The horse died and was necropsied. The meninges were dark red. The lateral, third and fourth ventricles and mesencephalic aqueduct were di-

lated and filled with yellow purulent exudate and clumps of fibrin. Cultures of the lateral ventricles of the brain yielded *Chryseobacterium meningosepticum*. The brain lesions were consistent with a **bacterial meningitis/ependymitis**. The isolate was an unusual one but the organism (previously known as *Flavobacterium meningosepticum*) is recognized as a sporadic cause of CNS infection in animals.

Coccidioides immitis was isolated from a draining tract in a horse. The only history available to us stated: “limb draining tract since summer, operated on once”. These lesions usually result from disseminated coccidioidomycosis, which although of rare occurrence in horses, should always be considered in these types of conditions. Other mycotic infections occasionally seen in chronic draining infections in our area are sporotrichosis, pythiosis and phaeohyphomycosis.

Avian



Sudden death occurred in a seventeen month old Quaker parrot that had been on a diet of “pellets, fruit, and veggies”. Necropsy revealed an enlarged, pale liver. Microscopic examination revealed moderate to severe, diffuse hepatocellular **lipidosis** and **hemosiderosis**. Analysis of liver tissue revealed an excessive level of iron (2098 ppm).

A chronic **purulent peritonitis** was the cause of death of an adult budgie. A large (1.0 by 0.5 cm) pedunculated **Sertoli cell tumor**, loosely attached to the testicles, was present in the abdominal cavity and may have precipitated the serosal infection.

Visceral gout was diagnosed in a five year old female parakeet which had been “fluffed for 2 weeks”, holding up the right leg. There were heavy deposits of urates in the kidneys, joints, and synovial sheaths of the tendons of the feet. The lesions were more severe in the joints and tendon sheaths of the right leg and foot. The precipitates formed whitish, hard nodules in the feet, some of which were ulcerated.

Canine



A five-year-old, spayed female Chihuahua died following a 24 hour history of acute onset of bloody diarrhea. There was a history of a recent diet change. Hemorrhagic gastroenteritis (HGE) was suspected. At necropsy the small intestine was diffusely reddened and had thick red/black contents. Microscopically, there was diffuse necrosis and hemorrhage of the mucosa with large numbers of gram-positive bacilli covering necrotic villi. *Clostridium perfringens* was isolated from the intestine and PCR analysis revealed that the isolate was an enterotoxigenic type A. *Clostridium perfringens* has been

implicated in the pathogenesis of the clinical syndrome known as **canine hemorrhagic gastroenteritis** but there is not much information that exists on the genotype or toxin producing characteristic of the isolated strains. This information is important in trying to understand the nature of this disease. The AzVDL has the capability to genotype *Clostridium perfringens* isolates and determine their spectrum of toxin producing capabilities, if any. We encourage submission of culture material from suspect cases of infection with this agent. Please contact the laboratory for additional information on samples required.

A six-year-old, castrated male Beagle developed renal failure and died acutely within a three-day period. Past pertinent history included testicular coccidioidomycosis six months prior to death. This condition had been treated with castration and itraconazole. At necropsy, both kidneys were pale and swollen. The spleen contained multiple, firm tan nodules and a few capsular scars. Microscopically there was severe **glomerular amyloidosis** in the kidneys with protein casts in tubules. The splenic lesions were granulomas centered around spherules of *Coccidioides immitis*. Thrombosis of a branch of the pulmonary artery was visible in the lung. Amyloidosis in dogs is of the secondary or reactive type meaning that it is usually associated with a chronic inflammatory condition. In this dog, the chronic granulomatous inflammatory process in the spleen stemming from the *Coccidioides immitis* infection was the underlying cause. Vascular thrombosis is occasionally seen in association with glomerular amyloidosis. The hypothesis is that glomerular leakage caused by the amyloid results in the loss of antithrombotic peptides (antithrombin III) and causes a hypercoagulable state.

Disseminated infection with *Encephalitozoon cuniculi* was the cause of severe neurologic signs and death in a litter of five-week-old Chihuahua puppies. This is a microsporidian parasite, which is a common infection of rodents and rabbits but is also a rare cause of neurologic disease in litters of puppies. The signs may resemble those of canine distemper virus infection. The transmission is primarily fecal-oral but transplacental transmission is thought to occur in foxes.

A six-year-old, male, German Shorthair Pointer died following an undefined history of “physical limitation”. At necropsy, the dog was thin. The thoracic cavity was filled with thick red fluid containing small yellow granules (**pyothorax**). The mediastinum was covered by velvety red tissue. The lungs were collapsed and the left diaphragmatic lobe was adhered to the left chest wall. A **grass awn** was found in the lung near the pleural surface of the left diaphragmatic lobe in the area of the adhesion. Bacterial cultures yielded a mixed population of *Actinomyces* sp. and *Pasteurella multocida*. The grass awn was identified as that of Slender Grama grass, *Bouteloua repens*.

A wolf-hybrid of unstated age died unexpectedly. Past per-

tinient history included recently diagnosed hypothyroidism and treatment with Soloxine. At necropsy, the posterior papillary muscle of the left ventricle of the heart contained a large dark red focus which extended into the adjacent left ventricular free wall. Microscopically the dog had multiple lesions of atherosclerosis involving arteries of the heart and spleen. One of the affected coronary arteries contained a thrombus. A focally extensive infarct, corresponding to the dark red focus, was present in the myocardium. Also present was severe lymphocytic thyroiditis. True **heart attacks** (myocardial infarctions) are rare in dogs, primarily since coronary artery disease/atherosclerosis is uncommon. However, atherosclerosis may develop in hypothyroid dogs.

A five-year-old female Dalmatian was presented with a history of sudden death. The history included treatment for valley fever since being diagnosed when the dog was quite young. The immediate cause of death was **suppurative pyelonephritis and septicemia** caused by hemolytic *E. coli*. Additional observations included chronic cardiomyopathy possibly related to long-standing infection with *Coccidioides immitis*. There was a focal cerebral granuloma with *Coccidioides immitis* present. This is a good example of chronic but controlled infection with *Coccidioides immitis*.

A seven-month-old male Shar Pei was presented with a history that indicated that the dog had been lame and had been treated with anti-inflammatory agents. Mucous membranes were very pale. The cause of death was toxemia and shock resulting from **intestinal strangulation** with a majority of the small intestine herniated through a small tear near the base of the mesentery.

Severe acute myocardial necrosis and hemorrhage of unknown etiology was diagnosed in a seven-year-old female American Eskimo canine. The dog had been treated with Rimadyl for mild hind limb weakness and soreness but became recumbent and unable to walk. It was presented dystonic and in rigid paralysis to the attending veterinarian. Severe pulmonary edema developed over the four hours that the animal was alive at the clinic. The condition was unresponsive to treatment. Extensive dissection of the coronary vessels of the heart did not reveal any evidence of thromboembolism. An etiology for the lesions was not determined.

Septicemia, due to mixed bacterial infection, was diagnosed in necropsies of two puppies that were described as four-day old Polish Shepherds. Clinical signs included lethargy and reluctance to nurse and lesions were compatible with septicemia. Cultures of lung tissue, a swab of heart chamber, and bone marrow yielded a mixed growth of *Staphylococcus aureus* and β hemolytic *E. coli* from one puppy. Group G streptococci and *E. coli* were obtained in cultures of lung and bone marrow from another puppy.

Feline



Nocardiosis of the subcutis and musculature of the ventral cervical area was diagnosed in a two-year-old spayed female feline. The submitting veterinarian noted that the cat had been under treatment for a swollen face for about three weeks and had a history of being in a catfight. Cardiac arrest occurred while the animal was being treated. Necropsy revealed a large, firm, irregular, roughly circular, yellowish-brown lesion with caviated areas that contained malodorous turbid fluid in the subcutis of the ventral cervical area. The lesion surrounded the anterior trachea and larynx structures. Microscopic examination revealed numerous gram positive, beaded, filamentous organisms in the inflamed lesion. Cultures of the lesion yielded moderate growths of *Nocardia asteroides*.

Feline herpesvirus infection along with a concurrent **feline leukemia** virus infection was diagnosed in an eight-week-old female Bengal kitten. According to the history provided by the submitting veterinarian, the kitten was part of a litter of six and had been sick "all her life." Death occurred spontaneously. The queen had a history of having "sickly kittens" in prior litters. Clinical signs were reported to be chronic upper respiratory disease and "failure to thrive." Lesions included severe tracheitis, mild pneumonia, gastritis, cryptitis of the colon, and severe multifocal myocardial necrosis. Feline herpesvirus was isolated from the tracheal mucosa. Fluorescent antibody staining of bone marrow was positive for feline leukemia virus.

Probable acute heart failure was diagnosed in a two-year-old neutered male domestic shorthair that was found dead shortly after being walked by its owner. The owner also noted that the cat was "possibly sleeper" during the last week. Gross and microscopic lesions were consistent with those of acute heart failure although there were no microscopic lesions in the myocardium.

A severe necrotizing enteritis produced by **feline panleukopenia virus infection** was the cause of death of a recently adopted, stray 5 month old kitten. The evolution of the lesions suggested a course of several days duration but the received history did not indicate any diarrhea, and formed feces were present in the rectum. Calicivirus was also isolated from the lungs although respiratory lesions were minimal.

Wildlife



Lead poisoning was diagnosed in a male Golden Eagle that was estimated to be two years old. The bird was picked up along side a street in the city of Tucson and was described as being lethargic, had difficulty standing, and had respiratory noise over the thoracic inlet.

The bird was otherwise in good flesh. The whole blood lead level was extremely high (8.5 ppm, values in excess of 0.4 ppm are reported to be diagnostic of lead intoxication in birds). The bird died the next day and analysis of the liver tissue revealed a concentration of 14.3 ppm lead. ("Normal" ranges for liver lead levels in waterfowl are 0.05 - 0.5 ppm. Toxic values are those in excess of 6 ppm). This is the second known case of lead intoxication in a raptor from the Tucson area. The source of the toxicant was not determined.

Lead poisoning was diagnosed in a Bald Eagle from the Flagstaff area. Clinical signs included lethargy, emaciation, and seizures followed by death. Liver lead level in the bird was in the 15-16 ppm range. Consumption of waterfowl whose flesh contains lead shot pellets is thought to be the main source of exposure for raptors suffering from lead poisoning.

A **necrotizing fasciitis** of the left rear leg produced by a beta hemolytic, group G *Streptococcus* sp. was the cause of death of a young raccoon caught in NW Phoenix. The animal had been captive for several weeks and treated for tail injuries. Group G streptococcal infections are being diagnosed in our laboratory with increasing frequency in dogs and cats in conditions which include reproductive infection, abortion, neonatal septicemia, sepsis, and necrotizing fasciitis similar to the human infections by "flesh eating bacteria" (group A streptococci). The present case is our first isolation of these agents from wildlife. (Please see "From the Arizona Veterinary Diagnostic Laboratory" on page 5.)

Owl herpesvirus infection was the cause of death in two adult Great Horned Owls received in the month of December. Both birds had multifocal areas of necrosis in the esophageal mucosal, liver, and spleen characteristic of the disease.

Exotics



A four-year-old male Pot-bellied pig died after being treated for two weeks for a blocked urethra. **Urethral obstruction** was caused by a 5-millimeter diameter urolith lodged in the urethra near the pelvic flexure. There were two other uroliths in the bladder. The uroliths were composed of calcium carbonate. Urethral obstructions are quite common in Pot-bellied pigs. Clinical management can be quite difficult.

We received a twenty-eight year old female baboon that died quite suddenly in a zoo. The animal had been observed to be anemic and icteric. **Amyloidosis of the pancreatic islets** may have been significant but this possibility cannot be confirmed after death. Additional interesting but non-fatal observations in this animal included relatively mild chronic glomerulonephritis, adrenocortical hyperplasia, teratomas of both ovaries, and leiomyoma of the uterus.

Small Ruminants



Club lamb fungus infection was the diagnosis in biopsy specimens obtained from a Suffolk lamb. The history included crusts, scales, and alopecia with multiple lesions. Fungal hyphae were identified in hair follicles and the dermis surrounding the hair follicles. This condition is of undetermined etiology with regard to species but it has been widely recognized in the Southwestern United States. It causes significant problems primarily for lambs at show time.

compiled by Greg Bradley, Bob Glock, Carlos Reggiardo, T. H. Noon

Comments on Diagnostic Update can be directed to Dr. Greg Bradley via e-mail at: azvdl@ag.arizona.edu

Arizona Veterinary Diagnostic Laboratory

Access to the AzVDL: Take Tucson Interstate 10 to the Miracle Mile exit #255. Travel approximately 1/4 mile on the south bound frontage road between Miracle Mile and Grant Rd. Turn west onto the farm at the signed entrance.

The University of Arizona
College of Agriculture

Department of Veterinary Science/Microbiology
2831 N. Freeway

Tucson, Arizona 85705-5021

Fax: (520) 626-8696 Phone: (520) 621-2356

email azvdl@ag.arizona.edu

We welcome any subscriptions, comments or suggestions.

Editor:

Barbara Hiers

Contributing editors:

Greg Bradley

Robert D. Glock

Ted H. Noon

Carlos Reggiardo

Faculty and Staff of the AzVDL

Director: Robert D. Glock DVM, PhD

Diagnosticians:

Greg Bradley DVM, Diplomate ACVP

Ted H. Noon DVM

Carlos Reggiardo DVM, PhD, Diplomate ACVM

Administrative Staff: Barbara Hiers

Sarah Swanson

Necropsy: Melvin Perry

Owen Halferty

Chemistry: Dana Perry Betzer MS

Barbara Rickert

Histology: Esther Kerr MT(ASCP)

Andrea Perez, Histotechnologist

Isaac Cordova

Microbiology: Natalie Furrey MT(ASCP)

Victoria Zhitnik

Virology: Mark Shupe

Thea Meeker

Adjunct Faculty:

Sharon Dial DVM, PhD

Howard Frederick PhD, PAS

Emeritus Faculty: Raymond E. Reed DVM, Diplomate ACVP

Issued in furtherance of Cooperative Extension work, acts of May 8 and June 30, 1914, in cooperation with the U.S. Department of Agriculture, James A. Christenson, Director, Cooperative Extension, College of Agriculture, The University of Arizona.

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The University of Arizona
Dept of Veterinary Science and Microbiology
Arizona Veterinary Diagnostic Laboratory
2831 N. Freeway
Tucson, AZ 85705-5021

