



Newsletter

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From the Director:

“Who gets what?” You may have wondered how your submissions to the diagnostic laboratory find their way to one diagnostician or another. This is the result of several processes. Sometimes the assignment is made on the basis of a specialty such as Microbiology, Toxicology or Pathology. Other times it is based on which diagnostician happens to be on duty for the particular day when the specimens arrive. The fact is we are not all identical and we have varying areas of expertise and, in fact, somewhat different personalities and interests. Therefore we frequently shuffle cases amongst ourselves to try to take advantage of our diverse interests. We regularly consult with each other to bring a group mentality to problem solving. You may wonder if we are offended if you request a particular diagnostician. We are not. We certainly try to honor requests for a specific diagnostician. Sometimes that means doing the preliminary workup on a case and then consulting with the requested individual. Other times, we may be unable to completely honor the request because of workload or absences. In any case, we are always happy to try to involve the veterinarian that you request. Also, if you are wondering about whether you should raise questions about the diagnosis you received with regard to the logic, the diagnostic procedure, or with regard to who made the case assignment decision, feel free to call us. We will be happy to work through any concerns.

Diagnostic Update

The following are selected samples of cases submitted to the AzVDL during the fall months of September, October and November.



Bovine

An outbreak of **Bovine Respiratory Syncytial Virus** infection was diagnosed in a group of yearling calves shortly after arrival at a local feedlot. The outbreak was severe, with high morbidity and the death of six calves. Calves developed watery nasal discharge, marked respiratory distress and were unresponsive to treatment. It was an unusual case because of its severity and because all the dead calves succumbed to an uncomplicated viral infection without any indication of associated bacterial pneumonia. The calves came directly from the ranch in northern Arizona without being exposed to other cattle during shipping or after arrival. Two vaccinations (at the ranch and on arrival) failed to prevent the outbreak. However, all calves were copper deficient with liver copper levels between 8 and 10 ppm. Copper deficiency is a well known immune suppressive factor and is believed to have played a very significant role in the increased susceptibility to BSRV infection in this case. The selenium status of the calves was uncertain because of treatment with injectable selenium on arrival.

Vegetative valvular endocarditis due to *Arcanobacterium pyogenes* infection was diagnosed in

tissue samples collected from the field necropsy of a five-year-old Holstein cow. The cow was thin and found down in a wash pen. It died twelve hours later. Necropsy examination revealed large, cauliflower lesions on the pulmonary valve and the left atrioventricular valve. *Arcanobacterium (Corynebacterium) pyogenes* is a common isolate from bacterial endocarditis of cattle. The right atrioventricular valve is the valve most commonly affected. The left atrioventricular valve is second in frequency of involvement. The pulmonary valve is rarely affected. Bilateral involvement of the atrioventricular valves has been reported.

Probable **milk fever (parturient paresis or hypocalcemia)** was supported by testing of blood samples submitted from a Guernsey cow that was eight days post calving and had, according to the submitting veterinarian, experienced “multiple episodes of weakness in past week”. The cow had been successfully treated with calcium injections. The cow’s serum calcium level was subnormal (6.7 mg/dl) which is suggestive of milk fever (parturient paresis). However, it is reported that coliform mastitis occurring around the time of calving can also induce hypocalcemia.

In this issue:

Who will be looking at my submission?	page 1
Diagnostic case update - Sept. to Nov.	page 1
Toxicology notes	page 5
Mosquito-bite dermatitis	page 5
Water quality assessment	special insert

The nature of the feeding program prior to calving has been shown to influence the occurrence of milk fever by reducing parathyroid gland activity. For instance: it is reported that feeding of a diet containing more than 100 g/day calcium during the dry period is associated with an increased incidence of milk fever. Dietary phosphorus fed in excess of 80 g/day is also reported to be associated with an increased incidence of the disease since it will decrease intestinal absorption of calcium before calving. Diets high in the cations sodium and potassium are also reported to induce the disease. The affected cow was being fed alfalfa hay in which the calcium/phosphorus ratio was about 4:1. The "ideal" calcium/phosphorus ratio for cattle is 1.5-2.0:1.

Campylobacter jejuni was isolated from the reproductive tract of a heifer sent to slaughter from a herd that had been experiencing reproductive failure. Reproductive failure in cattle due to campylobacter infection is generally associated with the venereally transmitted *Campylobacter fetus* ss. *venerealis* or, less commonly, *Campylobacter fetus* ss. *fetus*. The significance of *Campylobacter jejuni* as a reproductive pathogen is less than certain although it is reported that the organism has been isolated from aborted bovine fetuses and placentas.



Avian

A Macaw was presented with a history of severe dyspnea. The larynx was filled by exudate. The diagnosis was diffuse necrotizing inflammatory response affecting the larynx. A **mycotic tracheitis** was diagnosed produced by a non-*Candida albicans* yeast. The specific yeast could not be identified.

A die-off of several different types of birds in an aviary was consistently diagnosed as disseminated *Pseudomonas aeruginosa* infection. The source of the *Pseudomonas aeruginosa* is often a contaminated water supply but that was not specifically determined in this case.

Ten chickens from a flock of 280 birds of various breeds died after acute onset of illness characterized by swollen eyes, coughing and gasping. Gross necropsy examination results included mucoid ocular exudates and reddening of the conjunctiva. The trachea was diffusely reddened. Microscopic findings included an ulcerative conjunctivitis, rhinitis and tracheitis with intranuclear inclusions bodies consistent with herpesvirus infection. The disease is known by the common name **infectious laryngotracheitis** or LT. It is a contagious disease that is spread by the respiratory secretions of an infected bird. Severely infected birds suffer tracheal obstruction with fibrinonecrotic exudate resulting in characteristic gasping (pump-handle respiration). Death is often due to asphyxiation.

A male Amazon parrot died suddenly. Grossly, the bird was in thin body condition. The testes were markedly

enlarged, firm and pale tan. Each measured 5.0 by 3.0 by 2.0 cm. The small intestine was slightly thickened and the spleen was mildly enlarged and pale. Microscopically, the testes, small intestine and spleen contained multifocal to coalescing sheets of macrophages loaded with acid-fast bacteria consistent with *avian tuberculosis*.

Obstruction of the proventriculus/ventriculus due to impaction of a mass of synthetic fibers was diagnosed in a ten-year-old male cockatiel that died after being presented "depressed with labored breathing and decreased weight and muscle mass". Also found in the bird was **hemosiderosis** of the liver (liver tissue contained 1716 ppm iron).



Canine

Anticoagulant rodenticide poisoning (Bromadiolone) was diagnosed in a two-year old male Shepherd canine that was found dead in a pool with about one foot of water. According to the submitting veterinarian, the owner was "...concerned about poison. He barks a lot". Necropsy revealed dark red discoloration of the lungs and the tracheal lumen was filled with froth. Microscopic examination revealed pulmonary hemorrhage and edema. GC/MS screening of liver tissue and stomach contents was negative for toxins detectable by that method. However, this equipment has not been reliable to date in our hands for the detection of anticoagulant rodenticides. A sample of liver tissue was referred to another laboratory for anticoagulant screening using another technique. The sample was found to be positive for bromadiolone (100 ppb).

Cl. botulinum type C toxin was isolated from the blood of a three-year-old dog (breed not specified) with a history of ascending motor paralysis of several days duration, and a presumptive clinical diagnosis of botulism. The source of the intoxication could not be determined although the dog had access to open desert and presumably to wildlife cadavers. Botulism is very rarely diagnosed in dogs.

Canine **parvoviral enteritis** was diagnosed in five unrelated canine necropsies during this reporting period. Four of the cases were the typical hemorrhagic enteritis caused by Canine Parvovirus type II (CPV-2). The clinical course ranged from two to four days, and included vomiting, anorexia, diarrhea, and death. Two aspects of these cases that are note worthy are the age of the dogs and the basis for these diagnosis. The affected dogs ranged from 9 weeks to 3 years. The three-year-old was current in all vaccinations. This illustrates the fact that severe CPV-2 infections are occasionally diagnosed in well immunized adult dogs. In all four dogs there were typical histopathological lesions of necrosis of the intestinal crypt epithelium with collapse of the mucosa on which the diagnoses were based. In only two of the four cases viral particles could be observed in the intestinal content by electron microscopy. In the two cases in which an in-office ELISA test kit was used, the results were

negative. This inability to confirm CPV-2 infections by fecal ELISA or EM is a common occurrence in dogs that are sick for a couple of days or longer. It is usually due to neutralization of the virus by antibody. The fifth case of parvoviral enteritis was diagnosed in a five-day-old Shih Tzu puppy. Microscopically the tips of the villi of the intestinal epithelium exhibited prominent, basophilic intranuclear inclusions consistent with Canine Parvovirus type I (CPV-1) or Minute Virus of canines. This is not to be confused with canine parvovirus type II that is the cause of the hemorrhagic enteritis in dogs. This virus is antigenically distinct from CPV-2 and of somewhat uncertain pathogenicity. Although usually sub-clinical, it has been associated with abortions and neonatal deaths.

A six-year-old spayed female Dachshund died following a three-day history of illness. Initial signs included pyrexia, vomiting, diarrhea, seizures and leukopenia. Treatment included phenobarbital and antibiotics. Two days later, the dog developed a heart block with atrial premature contractions. She was depressed, mildly tachypneic and had an enlarging abdomen. Cardiomegaly and pulmonary edema were evident on thoracic radiographs. The dog was euthanized. Necropsy examination by the referring veterinarian revealed slightly bloody peritoneal fluid, a slightly edematous pancreas, dark loops of ileum with serosal hemorrhages, hemorrhages on the thoracic wall, hemorrhagic apical lung lobes, an enlarged right ventricle of the heart and hemorrhage in the papillary muscle of the mitral valve. **Suppurative myocarditis** was found on microscopic examination of submitted tissues. Also there was a suppurative enteritis and mesenteric lymphadenitis. Tissues for culture were not available but the lesions suggested a bacterial etiology, probably originating in the intestine with hematogenous spread to the myocardium.

An eleven-year-old, spayed female Chocolate Labrador was presented with **glossitis**. The tongue was so swollen that the airway was partially obstructed necessitating a tracheotomy. The swelling of the tongue continued to worsen and the dog was euthanized. At necropsy, the tongue was red, swollen and turgid. It protruded severely from the mouth and filled the oral and pharyngeal cavities. On cut surface, there was a 1.0 by 5.0 cm abscess filled with red, purulent exudate extending from the hyoid region rostrally. The adjacent muscle of the tongue was mottled and red. *E. coli* and *Streptococcus sp* (group G) were recovered from the abscess. Other significant lesions included a 2.0 cm diameter adrenocortical adenoma and bilateral atrophy of the adrenal cortices. How the infection became established in the tongue was not clear but a foreign body penetration or wound to the tongue seems plausible. The adrenocortical adenoma was apparently functional since it was associated with bilateral atrophy of the adrenal cortices. Hyperadrenocorticism resulting in increased susceptibility to infection was probably a contributory factor.

Aldicarb poisoning in dogs in the Tucson area was diagnosed on two occasions during this reporting period. One involved a three-month-old Shepherd mix that died shortly after the acute onset of foaming at the mouth, chewing, uncontrollable shaking, and profuse diarrhea. The other was a four-year-old male Chow mix that also became acutely ill. Stomach contents of both contained tiny, black "poppy seed" type granules. Testing of the brain tissue from the Chow mix yielded subnormal acetylcholinesterase activity (1.7 μ mole/min/g) that following incubation subsequently reactivated to a much higher level (5.5 μ mole/min/g), which is suggestive of carbamate intoxication. Aldicarb is a carbamate insecticide which is used in agriculture under the names Temik® and Sanacarb®.

Septicemia due to a mixed gram-negative infection (*Klebsiella sp.* and *E. coli*) was diagnosed in a three-day-old Great Dane puppy that was part of a litter of three. One of the pups was stillborn and the other two were apparently born healthy. The pup that was necropsied began to "fade" soon after birth and ultimately died. Lesions were typical of a septicemia and *Klebsiella sp.* and *E. coli* were isolated in cultures of brain, lung, and bone marrow.

Thrombosis of the portal vein and associated venous drainage with extensive infarction of the small intestine was diagnosed in an eight-year-old neutered male Labrador weighing 77 pounds. The dog had a history of a "hepatic lymphoma" and had been treated using antineoplastic chemotherapeutic agents several months prior to its death. It was described as being in complete remission but was subsequently presented "hypercalcemic with melena and hematemesis". Necropsy revealed infarction of most of the small intestine, beginning 30 inches distal to the pylorus and ending about 26 inches anterior to the ileoceocolic valve. There was no evidence of volvulus. The portal vein and associated venous drainage were distended with dry, clotted blood. A cause was not apparent. Idiopathic thrombosis of the portal vein has been reported in dogs.



Feline

A severe, chronic **hepatotoxicity** was the cause of death of a two-year-old DSH female from the Morenci area. The hepatic lesions were those of multifocal nuclear (karyomegaly) and cytoplasmic gigantism (megalocytosis) of hepatocytes, hepatocyte necrosis, and mild bile duct proliferation and fibrosis. There was also karyomegaly of the tubular epithelium of the kidneys. This type of lesions is occasionally observed in ruminants or horses in Arizona following the ingestion of plants containing pyrrolizidine alkaloids. It can also be observed in pigs, calves, and rarely other species such as dogs following chronic exposure to aflatoxins. But we had never observed similar lesions in cats. The history

available to us indicates that this animal was only fed commercial cat food and there was no known exposure to toxic plants.

A seventeen-year-old female cat was presented with a history of pleural effusions on the right side of the thorax. There was diffuse pleuritis and amorphous appearing lesions in the lung. The diagnosis was **bronchiogenic carcinoma** with metastasis to the kidney. Bronchiogenic carcinomas are not an unusual finding as a cause of pulmonary disease in older cats and dogs.

Strychnine was detected by thin layer chromatography in the stomach contents of a three-year-old male DSH cat that died suddenly. The owner reported that the cat came in the house with a strange look to its eyes that evening and was found dead the following morning. At necropsy the stomach contained parts of a rodent and kernels of millet seed stained with green dye. This apparently represents an instance of secondary poisoning of the cat after it ingested a rodent poisoned with strychnine containing bait.

Feline infectious enteritis (panleukopenia) was diagnosed in a two-month-old Bengal kitten that died after a history of diarrhea. Twenty cats were in the household and nine had died during the preceding two months. A vaccination history was not available.



Wildlife

Salmonellosis with an accompanying empyema of the lateral ventricles of brain was diagnosed in a four-year-old female Black-footed ferret that had been sick for about three days. The rehabilitator submitting the case indicated that the animal was unexpectedly found dead with no premonitory clinical signs being reported. Considerable purulent material was present in the lateral ventricles of brain and a group B *Salmonella* was isolated in cultures of bone marrow and lateral ventricle.

Severe Staphylococcal pneumonia was diagnosed in an adult female prairie dog that the rehabilitator reported to be "lethargic, dehydrated, thin, and having trouble breathing". The lungs were bilaterally consolidated and sank in fixative. Gram-positive coccoid organisms were evident in gram-stained sections of pneumonic lung tissue and heavy growths of *Staphylococcus aureus* were isolated from lung tissue.



Exotics

Mycotic dermatitis due to an unidentified, branching, septate fungus was confirmed in a Brook's kingsnake. The snake was being treated for a suspected

fungal dermatitis when it died. The skin contained multifocal areas of proliferative dermatitis characterized by flaking, deformation and brown discoloration of the scales. Microscopically, there was diffuse hyperkeratosis of the epidermis and invasion of the keratin by fungal hyphae. Mycotic dermatitis in reptiles is associated with poor sanitation and warm, wet conditions. Saprophytic fungi such as *Fusarium* and *Geotrichum* are the usual cause.

A female tetra had several, large, circular cyst-like structures on the lower jaw. Histologic examination of the affected tissue contained granulomas with numerous acid-fast bacteria consistent with **piscine tuberculosis**. *Mycobacterium fortuitum* and *M. marinum* are the usual species involved. All species of fish in both fresh and saltwater aquariums are susceptible. Clinical signs are variable and may include, anorexia, emaciation, vertebral malformations, exophthalmos and loss of coloration.

A 30+ year-old female polar bear was euthanized following a history of progressive weight loss, lethargy and stiffness that was unresponsive to therapy. Necropsy examination results included **degenerative joint disease** of both coxofemoral joints with rupture of the round ligament in the right joint. The left lung was replaced by firm tan-colored tissue, which microscopically was diagnosed as **bronchoalveolar carcinoma**. In addition, there was a small pancreatic adenocarcinoma and multifocal thyroid carcinoma. Skeletal muscle, especially the intercostal muscles and diaphragm contained myriads of tiny white foci. These corresponded to encysted parasites of *Trichinella* sp. **Trichinosis** is common in polar bears. The infection is spread by the consumption of carrion or garbage containing remains of infected mammals such as rodents or seals.

A disseminated infection with *Nocardia* sp. was diagnosed in an adult, male Gila Monster. The animal had a history of chronic sinusitis from which *Nocardia* had been isolated. Chronic anorexia and weight loss followed diagnosis and euthanasia was performed. Necropsy lesions reported by the referring veterinarian included numerous pale nodules throughout the lungs. Microscopically, the nodules corresponded to multifocal to coalescing granulomas with branching, filamentous, gram-positive bacteria in the lungs, heart, liver, and adrenal gland.

Two Mohave Rattlesnakes were submitted. They had been euthanized because of observation of severe oral hemorrhages. There was also a history of swelling of the venom glands. Both snakes had diffuse red to purple hemorrhagic areas in the mouth and especially in the area of the fangs. There was also hemorrhage in the venom glands of both snakes and some exudate could be expressed. One snake had hemorrhages in the posterior abdominal fat and in the kidneys. The oral mucosa had

extensive epithelial erosion in addition to the hemorrhage observed grossly and there were diffuse infiltrations of mixed inflammatory cells. One snake had numerous large basophilic inclusions distending the nuclei. Inflammatory lesions were also observed in the venom glands. The diagnosis was **glossitis** and **venom gland adenitis** probably resulting from initial infection with an unidentified virus and secondary infection by bacteria. *E. coli* and *Proteus* sp. were identified but may be secondary invaders. Tissues from these animals have been forwarded to a laboratory that specializes in diseases of snakes and particularly virology of snakes for additional testing

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

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

Notes from the toxicology lab

GC/MS analysis of an unknown whitish substance found in a split-open hot dog bait that had been tossed into a homeowner's yard revealed it to be bendiocarb, which is an insecticide sold under various trade names. The submitting veterinarian reported that a number of similar baits had been tossed into various yards in this Arizona community.

A potentially toxic level of nitrate was found in a sample of poor-quality hay containing a considerable amount of a pigweed-like contaminant that, according to the owner, had been bought and fed to two cows that were subsequently found dead. No necropsy was performed. Testing of the pigweed-like plant material contaminating the hay revealed that it contained a potentially toxic level of nitrate (values of 1% or higher of the total ration are considered potentially toxic for ruminant animals). A definitive diagnosis was not possible due to the absence of rumen content or clear body fluid for nitrate analysis. This case illustrates the need to necropsy the animals involved and rule out other possible causes of death.

by T. H. Noon, Dana Perry-Betzer, and Barbara Rickert

Feline mosquito-bite dermatitis

Three cases of this interesting entity were diagnosed by skin biopsies submitted to the AzVDL during the late summer. The histologic features were characteristic of the disease and resemble those of the feline eosinophilic granuloma complex. The gross lesions of feline mosquito-bite dermatitis include papular eruptions and erosions with crusting and depigmentation of the skin on the bridge of the nose. Hyperkeratosis and sometimes hyperpigmentation may be seen on the pads of the feet. The pad lesions may include fissuring and depigmentation. Lymphadenopathy and mild fever may be noted in some cats. There is a seasonal occurrence with most cases occurring during the summer and resolving by winter. The lesions also tend to resolve within a week if the cats are confined indoors or hospitalized so that exposure to mosquitoes does not occur. They will redevelop if exposure to mosquitoes resumes. A hypersensitivity reaction to an antigen in the mosquito bite is hypothesized to be involved in the pathogenesis. Two of four cats tested in the original study describing this disease reacted to mosquito extract injected intradermally and read at 20 minutes.

Mason KV, Evans AG. 1991. Mosquito bite-caused eosinophilic dermatitis in cats. JAVMA 198: 2086-2088

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.

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