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CASE I – 125/01 (AFIP 2789367)

Signalment: 10-month-old, ewe, Merino cross, ovine

History: This ewe was one from a flock of 420 grazing dry summer pastures. There had been approximately 25 deaths in the flock over the last 2 months. Sheep from the property had previously been diagnosed with white muscle disease. This mob was being fed silage and had been drenched for worms twice in the last 6 weeks. Some of the sheep had blocky firm wool clinically diagnosed as fleece rot.

Gross Pathology: This sheep was submitted in very thin body condition. There was severe osteopenia, most notably in frontal bones and appendicular skeleton, and there was heavy enteric parasitism with *Ostertagia* and *Nematodirus* spp. The animal had only poor quality forage in its rumen, there was papillary atrophy of the ruminal mucosa and the liver was swollen and fatty. The fleece over the rump was hard and segregated into irregular blocks ranging from 1-2 cm across. Removal of these blocky sections of fleece exposed raw, ulcerated areas. The wool was not discolored.

Laboratory Results: None.

Contributor's Morphologic Diagnosis: Pustular and proliferative epidermitis, chronic, multifocal, severe, with intralesional organisms typical of *Dermatophilus congolensis*.

Contributor's Comment: The skin lesions in this case represent an excellent example of cutaneous dermatophilosis. The lesions are characterized by multiple layers of serous to purulent exudate that have accumulated in the fleece above the epidermis, extensive epidermal hyperkeratosis and parakeratosis, multifocal

intraepidermal pustules, vesicles and ulcers and a mild, predominantly nonsuppurative, superficial, perivascular dermatitis. The most striking feature histologically is the alternating bands of keratin and exudate accumulating at the skin surface. The skin surface has been colonized by mixed bacteria but numerous banded filaments are present in serous vesicles and pustules adjacent to the epidermis. These are Gram-positive and typical of *Dermatophilus congolensis*.

Dermatophilosis is a well-recognised entity in livestock exposed to warm, moist climatic conditions. The clinical lesions are distinctive when involving the skin of mammals but in poikilotherms *Dermatophilus* species sporadically cause necrogranulomas in internal tissues. In this case, the lesions affecting the fleece were considered of minor importance, and the causes of debility were thought to be chronic malnutrition and enteric parasitism. However, the reason why some of this flock had "lumpy skin disease" remain unclear since at the time this part of Australia was experiencing one of the hottest and driest weather periods on record.

The interaction of the immune system with *D. congolensis* in providing protection against disease is not known in sheep. In cattle, recent studies have shown that immunity is strain-specific and that cell-mediated responses involving gamma-delta T-cells may be involved.

AFIP Diagnosis: Haired skin: Epidermitis, proliferative and exudative, multifocal, chronic, neutrophilic, marked, with lamellated hyperkeratosis and parallel chains of coccobacillus, Merino cross, ovine.

Conference Comment: *Dermatophilus congolensis* grows as branching filamentous mycelia composed of parallel zoospores. Under the appropriate conditions, zoospores transform into motile infectious cocci.

As Dermatophilus is not a highly invasive organism, it is generally unable to disrupt the normal physical barrier provided by hair or wool, the waxy superficial sebaceous layer, and the stratum corneum. Prolonged wetting of the skin as well as trauma (shearing or mounting injury) and ectoparasitism (tick infestation, biting flies) can allow an infection to become established.

The characteristic lamellated appearance of alternating hyperkeratosis and degenerate neutrophils is the result of cyclical invasion of the epidermis and hair follicle by the bacterium. Zoospores infiltrate the deeper cell layers of the disrupted epidermis in response to a CO2 gradient. Following the initial invasion and multiplication of the organism there is neutrophilic infiltration that inhibits any further progression. The epidermis overlying the neutrophils keratinizes while the underlying epidermis quickly regenerates resulting in a thick hyperkeratotic layer. Residual organisms then reinvade and the cycle is repeated.

Contributor: University of Melbourne, Veterinary Science Clinical Center, Werribee, Victoria, Australia

References: 1. Ambrose N, Lloyd D, Maillard J: Immune responses to *Dermatophilus congolensis* infections. Parasitol Today **15**:295-300, 1999
2. Haargis A, Ginn P: Integumentary System. *In*: Thomson's Special Veterinary Pathology, eds. McGavin M, Carlton W, Zachary J, 3rd ed., pp. 563. Mosby, St. Louis, MO, 2001
3. Hirsch D, Zee Y: Veterinary Microbiology, pp. 211-212. Blackwell Science, Malden, MA, 1999
4. Yager J, Scott D: The skin and appendages. *In*: Pathology of Domestic Animals, eds. Jubb K, Kennedy P, Palmer N, 4th ed., vol. 1, pp. 648-651. Academic Press, San Diego, CA, 1993

CASE II – CASE 1 (AFIP 2788384)

Signalment: Fifteen-year-old, neutered male, goat

History: This goat was part of a small herd used for antibody production. He had been on the same farm for 10 years, was kept at pasture, wormed monthly, immunized with antigen periodically and bled monthly for serum production. All goats appeared to be in good health until about a month ago, when one scoured and died, but no necropsy was done. Then two more goats scoured, this one died and a necropsy was done.

Gross Pathology: Liquid feces, subcutaneous edema, fluid in the abdominal cavity. There were fibrotic adhesions from the liver capsule to the diaphragm. Mesenteric lymph nodes were enlarged. A single *Cysticercus* sp. was present in the abdominal cavity. Two nematodes were present in the abomasum; one was *Haemonchus contortus*.

Laboratory Results: A Block of tissue (ileum) sent to the state diagnostic lab in Harrisburg, Pa for identification of *Mycobacterium* subtype by PCR revealed *Mycobacterium avium* subsp. *paratuberculosis* IS900.

Contributor's Morphologic Diagnosis: Johne's disease due to *Mycobacterium avium* subsp. *paratuberculosis*.

Contributor's Comment: Several areas of the mesenteric lymph node and other abdominal lymph nodes were examined. These showed severe lymphoid depletion with no normal lymphoid follicles or paracortical lymphoid tissue visible, severe macrophage infiltration with giant cell formation, moderate caseous necrosis of

cortical areas, and mild lymphocyte infiltration of the capsule. The ileum had villous atrophy and fusion, and the mucosal lamina propria was heavily infiltrated with macrophages/epithelioid cells with moderate lymphocytic infiltration of the submucosa. The cecal mucosal lamina propria contained numerous foci of macrophages/epithelioid cells with diffuse lymphocyte and plasma cell infiltration. The colonic lamina propria was moderately infiltrated with foamy macrophages and there were few crypt abscesses.

Ziehl-Nielsen stained sections of ileum, jejunum and mesenteric lymph node revealed that macrophages contained numerous acid-fast organisms. This is characteristic of Johne's disease due to infection with Mycobacterium avium subsp. paratuberculosis.

Johne's disease in goats has been less well characterized than in cattle, however, it has been described as causing chronic wasting with diarrhea occurring only terminally, as in this goat. Rugous thickening of intestinal mucosa, so frequently seen in cattle, is often absent at necropsy in goats, as in this case, despite the presence of distinct microscopic lesions with abundant epithelioid cells and acid fast organisms in much of the small and large intestine. This lymph node shows caseous necrosis with mineralization, which is frequently seen in goats, but not in cattle, with Johne's disease. The spectrum of lesions produced by this disease in goats in different parts of the world has been described recently. It has been shown that the Mycobacteria enter the gut through the "M cells", follicle-associated epithelial cells, over the Peyer's patches. Reservoirs of infection, in addition to domestic ruminants, are birds including domestic fowl, rabbits and deer. Recently evidence has emerged of the spread of *Mycobacterium avium* subsp. *paratuberculosis* on intestinal nematode larvae.

AFIP Diagnoses: 1. Lymph node: Lymphadenitis, granulomatous and necrotizing, multifocal to coalescing, severe, with intrahistiocytic bacilli, goat, breed not specified, caprine.
2. Lymph node: Lymphoid depletion, diffuse, moderate.

Conference Comment: Mycobacterium is an acid-fast, weakly gram positive, facultative intracellular bacillus. Mycolic acids and their esters make up the cell wall and account for its acid-fast properties. Mycosides (a glycolipid) protect the bacterium against lysosomal digestion within macrophages. The differential diagnosis discussed in conference included *Corynebacterium pseudotuberculosis*, *Rhodococcus equi*, and *Burkholderia pseudomallei*.

Corynebacterium pseudotuberculosis is a non-acid-fast, gram positive, facultative intracellular coccoid to filamentous bacterium. The cause of caseous lymphadenitis, *C. pseudotuberculosis* is found in the gastrointestinal tract and soil. Infection occurs through the contamination of cutaneous wounds with spread to regional lymph nodes. *C. pseudotuberculosis* releases an endotoxin with

phospholipase D activity that works synergistically with "*Rhodococcus equi* factors" which include phospholipase C and cholesterol oxidase.

Rhodococcus equi is a weakly acid-fast, gram negative, facultative intracellular coccobacillus. Suppression of phagolysosomal fusion helps facilitate intracellular survival. *R. equi*, which is present in the soil and feces, is most commonly associated with pyogranulomatous bronchopneumonia in foals. In goats, caseous abscesses may develop in the liver, spleen, lymph nodes, lung, and bones.

Burkholderia pseudomallei, the cause of melioidosis, is a saprophytic, gram negative, facultative intracellular bacillus. Similar to glanders (*Burkholderia mallei*), *B. pseudomallei* affects a wider host range and is associated with systemic suppurative abscessation affecting many internal organs. Contaminated feed and water is often the source of infection.

Contributor: Novartis Pharmaceuticals, East Hanover, NJ

References: 1. Barker I, Van Dreumel A, Palmer N: The Alimentary System. *In*: Pathology of Domestic Animals, eds. Jubb K, Kennedy P, Palmer N, 4th ed., vol. 1, pp. 250-251. Academic Press, San Diego, CA, 1991
2. Beard P, Rhind S, Buxton D, Daniels M, Henderson D, Pirie A, Rudge K, Greig A, Hutchings M, Stevenson K, Sharp J: Natural paratuberculosis infection in rabbits in Scotland. J Comp Pathol **124**:290-299, 2001
3. Corpa J, Garrido J, Garcia Marin J, Perez V: Classification of lesions observed in natural cases of paratuberculosis in goats. J Comp Path **122**:255-265, 2000
4. Daniels M, Ball N, Hutchings M, Greig A: The grazing response of cattle to pasture contaminated with rabbit faeces and the implications for the transmission of paratuberculosis. Vet J **161**:306-313, 2001
5. Hirsch D, Zee Y: Veterinary Microbiology, pp. 104-108, 129-134, 155-157. Blackwell Science, Malden, MA, 1999
6. Manning E, Steinberg H, Rossow K, Ruth G, Collins M: Epizootic of paratuberculosis in farmed elk. J Am Vet Med Assoc **213**:1320-1322, 1998
7. Pavlik I, Bartl J, Dvorska L, Svastova P, du Maine R, Machackova M, Yayo Ayele W, Horvathova A: Epidemiology of paratuberculosis in wild ruminants studied by restriction fragment length polymorphism in the Czech Republic during the period 1995-1998. Vet Microbiol **77**:231-251, 2000
8. Sigurdartottir O, Press C, Evensen O : Uptake of *Mycobacterium avium* subsp. *paratuberculosis* through the distal small intestinal mucosa in goats. Vet Pathol **38**:184-189, 2001
9. Sigurdartottir O, Press C, Saxegaard F, Evensen O: Bacterial isolation, immunological response, and histopathological lesions during the early subclinical phase of experimental infection of goats kids with *Mycobacterium avium* subsp. *paratuberculosis*. Vet Pathol **36**:542-550, 1999

10. Whittington R, Lloyd J, Reddacliff L: Recovery of *Mycobacterium avium* subsp. *paratuberculosis* from nematode larvae cultured from the feces of sheep with Johne's disease. Vet Microbiol **81**:273-279, 2001

CASE III – V99-5910 (AFIP 2792728)

Signalment: Three-year-old, spayed female, Chow Chow, canine

History: This young adult female Chow Chow presented to the base veterinary treatment facility at Sigonella Naval Air Station, Sicily on 7 January 1999 with the chief clinical complaint of a generalized skin problem. The clinician's differential diagnosis included leishmaniasis and deep pyoderma.

Gross Pathology: None

Laboratory Results: 1. Multiple skin scrapes performed at presentation and on 23 February 1999 were negative.
2. Fungal culture performed on 23 February 1999 using dermatophyte test media was negative.
3. Hematocrit measured on 23 February 1999 was 21%.
4. Leishmania titer was measured on 12 January 1999 and found to be negative.
5. Multiple punch biopsies of skin lesions were submitted to Veterinary Laboratory Europe on 5 May 1999.

Contributor's Morphologic Diagnoses: 1. Haired skin and subcutis, site unspecified: Dermatitis, necrotizing, lymphohistiocytic, neutrophilic, and plasmacytic, diffuse, moderate to severe, with multifocal perivascular panniculitis, hemorrhage, and intrahistiocytic protozoal amastigotes, etiology consistent with *Leishmania* sp.
2. Haired skin, site unspecified: Follicular and epidermal hyperplasia and hyperkeratosis, orthokeratotic, diffuse, moderate, with lymphoplasmacytic, histiocytic, and neutrophilic periadnexal dermatitis and mural folliculitis, follicular ectasia, and intrafollicular mites, etiology consistent with *Demodex canis*.

Contributor's Comment: Leishmaniasis, a worldwide infectious disease of humans and animals, is caused by diphasic protozoans of the genus *Leishmania*. This zoonotic disease may present as cutaneous, mucocutaneous, or visceral forms. The disease is endemic in Mediterranean countries, such as the Italian island of Sicily. The most common species of *Leishmania* infecting dogs in Sicily is *L. infantum*. Sandflies are considered to be the vector of infection for animals; in Europe, the sandfly vectors are members of the genus *Phlebotomus*, while in North America the vectors are from the genus *Lutzomyia*. Leishmaniasis has been

reported in dogs, cats, and horses. Reservoir hosts for human infections include dogs, cats, and rodents.

Clinical leishmaniasis usually causes chronic systemic disease in dogs, and nearly 90% of animals have dermatologic abnormalities. Cutaneous manifestations of overt disease include generalized alopecia, exfoliative dermatitis with silvery-white scales, and skin ulceration and nodules as the disease progresses. Dermatologic lesions chiefly occur on anatomic locations on which sandflies feed, such as the muzzle, periorbital region, and ears. Histopathologic findings include hyperkeratosis and parakeratosis with crusting, moderate acanthosis, and multinodular to coalescing infiltration of the dermis by many foamy macrophages admixed with fewer lymphocytes, plasma cells, and neutrophils. Protozoal amastigotes are principally found with macrophages.

The most common clinical signs of visceral leishmaniasis include weight loss, decreased physical activity, chronic renal failure, lymphadenopathy, neuralgia, and ocular lesions. Other less common manifestations of disease include nephrotic syndrome, ascites, polyarthritis, epistaxis, ulcerative stomatitis, pericardial tamponade, and chronic colitis. Laboratory derangements associated with active disease include nonregenerative anemia, proteinuria, azotemia, and hyperproteinemia. Thrombosis may occur as a result of nephrotic syndrome caused by glomerulonephritis.

Canine demodicosis, caused by the mite *Demodex canis*, occurs in generalized and localized forms. Both clinical forms are more common in juvenile and young adult animals, and disease tends to occur primarily in purebred dogs of many breeds; these clinical findings suggest an inherited immune deficit. Demodicosis also occurs in adult dogs in association with underlying metabolic disease or drug therapy, such as hypothyroidism, hyperadrenocorticism, glucocorticoid therapy, and cytotoxic chemotherapy. Clinical findings in localized demodicosis include small, scaly, non-pruritic areas of alopecia primarily on the face and forelegs. Generalized demodicosis affects extensive areas of the animal, with large zones of alopecia, erythema, scaling, and crusts. Typical histopathologic findings include follicular and epidermal acanthosis and hyperkeratosis, lymphoplasmacytic mural folliculitis and perifolliculitis, perifollicular granulomas, sebaceous adenitis, and intrafollicular mites. Furunculosis and bacterial folliculitis may complicate severe cases of demodicosis.

Both demodicosis and overt leishmaniasis in dogs are associated with underlying deficiency in cell-mediated immunity, and much of the literature attributes the immune dysfunction to T-lymphocyte deficits. Immunodeficiency probably promotes the incidence of concomitant infections; concurrent ehrlichiosis, babesiosis, dirofilariasis, and demodicosis is observed in dogs with overt

leishmaniasis. The negative *Leishmania* titer in the face of active disease with concurrent demodicosis strongly suggest immunosuppression in this dog.

AFIP Diagnoses: 1. Haired skin: Dermatitis, lymphoplasmacytic and histiocytic, multifocal, moderate, with intrahistiocytic amastigotes, etiology consistent with *Leishmania* sp., Chow Chow, canine.
2. Haired skin: Folliculitis and furunculosis, granulomatous, multifocal, moderate, with intrafollicular and intradermal mites, etiology consistent with *Demodex* sp.

Conference Comment: *Leishmania* sp. are protozoan in the order Kinetoplastida, family Trypanosomatidae. Flagellated promastigotes are introduced into the host during sandfly feeding and are engulfed by macrophages. Within macrophages, aflagellated amastigotes (characterized by a round nucleus with a perpendicular rod shaped kinetoplast) divide by binary fission eventually rupturing the cell to infect new cells. Phagocytosis by macrophages is enhanced by protozoal surface lipophosphoglycans that form a dense glycocalyx and bind C3b (an opsonin). Within phagolysosomes, lipophosphoglycans also scavenge free radicals while a proton transporting ATP-ase maintains the pH at an appropriate level. Additional survival strategies include inhibition of macrophage cell signaling pathways (via inhibition of protein kinase C activation) and inhibition of nitric oxide and cytokine production (via inhibition of IFN-gamma induced tyrosine kinase phosphorylation). Through unclear mechanisms T-cells become depleted while B cells proliferate. With fewer T-cells, macrophage effectiveness is reduced allowing widespread dissemination of the organism. Profuse B cell propagation can result in abundant circulating immune complexes that deposit resulting in vasculitis, polyarthritis, and glomerulonephritis. Also included in the propagation may be cryoglobulins resulting in ischemic necrosis of the distal extremities and autoantibodies associated with the development of immune-mediated thrombocytopenia and anemia.

Histologically, the three most common patterns include granulomatous perifolliculitis, superficial and deep perivascular dermatitis, and interstitial dermatitis. Additional gross findings include nasodigital hyperkeratosis, onychogryposis, generalized lymphadenopathy, and hepatosplenomegaly.

Demodex canis is an obligate parasite which spends its' entire life cycle on the host feeding on cells, sebum, and debris. Characterized by jointed appendages, an exoskeleton, striated musculature, readily identifiable mouthparts, and a fusiform body shape, it is relatively distinct.

The most consistent histologic finding in active demodicosis is a mural folliculitis. The mural infiltrate is composed primarily of CD8+ T-lymphocytes while the infiltrate in the surrounding perifollicular dermis is composed of equal numbers of CD8+ and CD4+ cells with fewer CD21+ B-lymphocytes. With resolution the mural folliculitis subsides initially while the perifolliculitis tends to

persist. Additional histological findings include follicular and epidermal hyperkeratosis, follicular epithelial degeneration, and follicular pigmentary incontinence.

Contributor: Veterinary Laboratory Europe, CMR 402, APO AE 09180, Landstuhl, Germany

References: 1. Brandonisio O, Panaro M, Sisto M, Acquafredda A, Fumarola L, Leogrande D: Interactions between Leishmania parasites and host cells. Parassitologia **42**:183-190, 2000
2. Caswell J, Yager J, Parker W, Moore P: A prospective study of the immunophenotype and temporal changes in the histologic lesions of canine demodicosis. Vet Pathol **34**:279-287, 1997
3. Cunningham A: Parasitic adaptive mechanisms in infection by Leishmania. Exp Mol Pathol **72**:132-141, 2002
4. Gardiner C, Fayer R, Dubey J: An Atlas of Protozoan Parasites in Animal Tissues, 2nd ed., pp. 1-5. Armed Forces Institute of Pathology, Washington, DC, 1998
5. Gardiner C, Poyton S: An Atlas of Metazoan Parasites in Animal Tissues, pp. 56-58. Armed Forces Institute of Pathology, Washington, DC, 1999
6. Gross T, Ihrke P, Walder E: Diseases of the dermis. *In:* Veterinary Dermatopathology, pp. 186-188, Mosby-Year Book, St. Louis, MO, 1992
7. Gross T, Ihrke P, Walder E: Diseases of the hair follicles. *In:* Veterinary Dermatopathology, pp. 262-266, Mosby-Year Book, St. Louis, MO, 1992
8. Hargis A, Ginn P: The integumentary system. *In:* Thomson's Special Veterinary Pathology, eds. McGavin M, Carlton W, and Zachary J, 3rd ed., pp. 571-572, 575-576, Mosby, St Louis, MO, 2001
9. Koutinas A: Clinical considerations on canine visceral leishmaniasis in Greece: a retrospective study of 158 cases (1989-1996). J Am Anim Hosp Assoc **35**:376-383, 1999
10. Mozos E, Perez J, Day M: Leishmaniosis and generalized demodicosis in three dogs: a clinicopathologic and immunohistochemical study. J Comp Pathol **120**:257-268, 1999
11. Orndorff G, Cooper B, Smith W, Ryan J: Canine visceral leishmaniasis in Sicily. Mil Med **165**:29-32, 2000
12. Scott D, Miller W, Griffin C: Muller and Kirk's Small Animal Dermatology, 6th ed., pp. 457- 474, 535-538. W.B. Saunders, Philadelphia, PA, 2001
13. Slappendel R, Ferrer L: Leishmaniasis. *In:* Infectious Diseases of the Dog and Cat, ed. Greene C, 2nd ed., pp. 450-458, WB Saunders, Philadelphia, PA, 1998

CASE IV – UFSM-1 (AFIP 2789831)

Signalment: Two-year-old, female, mixed breed, bovine

History: Sixty two-year-old cattle (40 females and 20 males) and six pregnant cows were placed in an 80-hectare pasture in October 1999. This pasture was heavily invaded by the plant *Ateleia glazioviana*. From January 2000 up to the date of this necropsy (July 2000), eight cattle (two cows, a steer and five heifers) died. Three of those animals presented sudden death and the others ran a chronic clinical course characterized by depression, subcutaneous dependant edema, engorged and pulsating jugular vein. Three abortions were reported among the pregnant cows. One heifer was necropsied.

Gross Pathology: The subcutaneous tissue (SC) of the head, submandibular region, ventral neck and brisket was marked swollen. At cut surface, the SC of these regions was gelatinous and translucent. Clear yellow fluid was present in the abdominal cavity (≅ 20 L) and in the pericardial sac (≅ 1 L). The margins of the liver were rounded and the hepatic capsule thickened. The cut surface had a marked lobular pattern (nutmeg liver). The mesentery and the wall of the large intestine were thickened by clear fluid that imparted a gelatinous aspect to those structures; the lymphatics in the mesentery and intestinal serosa were distended. Mesenteric lymph nodes were enlarged and moist. There were firm, white to yellow areas in the myocardium. These areas could be seen through pericardial surface but are better appreciated at cut surface.

Laboratory Results: None available.

Contributor's Morphologic Diagnosis: Myocardium, degeneration and necrosis with myofiber loss and fibrosis, chronic.

Other morphological diagnosis (slides not included):
Liver, centrolobular congestion, hepatocellular degeneration and bridging fibrosis (nutmeg liver).
Brain, white matter, intramyelinic edema (*status spongiosus*).

Etiology: poisoning by *Ateleia glazioviana*.

Contributor's Comment: *Ateleia glazioviana* (Leguminosae) is a tree most commonly found in woods and roadsides and invading pastures. In Brazil it is found in the southern states. The poisoning by the ingestion of the fallen leaves of *A. glazioviana* or its sproutings is described in cattle of southern Brazil and induces three forms of clinical manifestations including i) abortions, ii) a clinical form characterized by nervous signs such as depression (lethargy) and blindness and iii) cardiac failure. There is considerable overlapping among these clinical manifestations. The three clinical forms can be seen in the same outbreak but there are outbreaks where only a single clinical presentation occurs. Affected

cattle are more than one-year-old and there is no predisposition for breed or sex. The three forms of the toxicosis were reproduced in our laboratory by the feeding of *A. glazioviana* to cattle and sheep. The heart failure induced by the ingestion of *A. glazioviana* (both experimental and spontaneous field cases) are associated with a toxic cardiomyopathy manifested clinically in two ways. In one of them the animal simply "drops dead" with no premonitory signs, generally after being forced to exercise. This form is generally referred to as "sudden death". In the other form, previous signs of congestive heart failure precede death (as is the case of the heifer of this report). These signs include subcutaneous dependant edema (brisket edema) and an engorged and pulsing jugular vein. In cattle presenting this form, morbidity is 10%-60% and lethality is virtually 100%. Necropsy findings in cattle affected with the congestive heart failure include changes in the myocardium characterized by irregular, sharply demarcated, firm white areas of discoloration. These areas can be seen through the epicardium but are best seen in the cut surface of the heart. Edema in various tissues and body cavities can be seen. The liver is frequently enlarged and congested and has a nutmeg aspect particularly evident on the cut surface. In the "sudden death" form, lesions are restricted to the myocardium. Histologically, the primary lesion consists of chronic degenerative cardiomyopathy characterized by loss of myocytes and fibrosis. In the lethargic form, the basis for the neurological signs, such as depression and blindness, is intramyelinic edema (spongy degeneration) which is most probably caused by the direct effects of the plant since it occurs in the absence of hepatic lesions. Spongy degeneration (*status spongiosus*) can be found also associated with cases of cardiac failure. The toxic principle of *A. glazioviana* remains undetermined. Degeneration, necrosis and fibrosis induced in the myocardium by *A. glazioviana* are striking lesions, although not pathognomonic. In Brazil, two other plants (*Tetrapterys acutifolia* and *T. multiglandulosa*) cause similar clinical disease and lesions in cattle. However *A. glazioviana* poisoning and *Tetrapterys* spp. poisoning occur in regions of the country widely apart from each other.

A disease with similar clinical signs (brisket edema due to cardiac failure) occurs in cattle from one of the Brazilian states were *A. glazioviana* poisoning occurs and should be included in the differential diagnosis. The disease is known there as "swollen brisket disease" and, although thought to be caused by ingestion of a poisonous plant, its etiology is still unclear. Although swollen brisket disease and *A. glazioviana* poisoning in cattle share similar clinical signs, the epidemiology and gross and microscopic lesions in the heart differ and allow one to distinguish between them. The clinical course of swollen brisket disease does not include abortion; the disease occurs only in cattle over three to four-years-old that are raised at altitudes between 1,100 and 1,400 meters; it rarely if ever induces sudden death; and swollen brisket disease does not induce extensive fibrosis in the myocardium. The clinical signs and cardiac lesions resulting from the ingestion of *A. glazioviana* are somewhat similar to those observed in ionophore poisoning in cattle. The epidemiology and the distribution of lesions in the heart and skeletal

muscles should help in differentiate between the two conditions. In ionophore poisoning lesions can be found in the skeletal muscle; these changes do not occur in *A. glazioviana* poisoning.

Comparable diseases in cattle are also reported in Africa and Australia; these diseases are characterized by a severe form of toxic degenerative cardiomyopathy caused by poisonous plants which have to be eaten by cattle in large amounts for prolonged periods of time. In Africa the disease is known clinically by the Afrikaans sobriquet "gousiekte" (quick disease) and is caused by a group of poisonous plants which includes *Pachystigma* spp., *Pavetta* spp. and *Fadogia monticola*. In Australia *Acacia georginae* is the poisonous plant involved. Frequently the poisoning in cattle by coffee senna (*Senna occidentalis*, formerly *Cassia occidentalis*) is included in the differential diagnosis of all the cardiomyopathies discussed above. From our experience we do not think this is valid. Coffee senna poisoning in cattle is very prevalent in southern Brazil and what follows are our observations from a large series of clinical cases and necropsies of cattle affected by this poisoning (experimentally or spontaneously). We have never observed any clinical sign related to cardiac dysfunction in coffee senna poisoned cattle, at least none anywhere similar to those observed in *A. glazioviana* poisoning. In coffee senna poisoned cattle, gross lesions in the myocardium are either absent (most of the times) or are very mild. Microscopically, these lesions, when present, are mild and acute, never associated with fibrosis; while degenerative/necrotic lesions in the skeletal muscle are marked.

The spongy degeneration found frequently in the brains of cattle poisoned by *A. glazioviana* are similar to the ones observed in other plant poisonous such as *Helichrysum argyrosphaerum* and chemical substances like closantel.

AFIP Diagnoses: 1. Heart: Cardiomyocyte degeneration and loss, diffuse, with fibrosis and multifocal mild histiocytic myocarditis, mixed breed, bovine.
2. Heart, cardiomyocytes: Sarcocysts, several.

Conference Comment: The contributor has provided a complete review of this entity. The differential diagnosis discussed included nutritional deficiencies (vitamin E/selenium, copper), toxicities (ionophores), and infectious agents (*Clostridium chauvoei*, *Fusobacterium necrophorum*).

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References: 1. Gava A, Barros C: Field observations of *Ateleia glazioviana* poisoning in cattle in southern Brazil. Vet Human Toxicol **43**:37-41, 2001

2. Kellerman T, Coetzer J, Naudé T: Plant Poisoning and Mycotoxicoses of Livestock in South Africa, pp. 83-130, Oxford University Press, Cape Town, South Africa, 1988
3. Peixoto P, Loretti A, Tokarnia C: "Doença do peito inchado", *Tetrapterys* spp. poisoning, briskett disease and St. George disease: A comparative study. Pesq Vet Bras **15**:43-50, 1995
4. Tokarnia C, Peixoto P, Döbereiner J: Poisonous plants affecting heart function of cattle in Brazil. Pesq Vet Bras **10**:1-10, 1990
5. Van der Lugt J, Olivier J, Jordaan P: Status spongiosis, optic neuropathy, and retinal degeneration in *Helichrysum argyrosphaerum* poisoning in sheep and a goat. Vet Pathol **33**:495-502, 1996
6. Van Vleet J, Ferrans V: Cardiovascular System. *In*: Thomson's Special Veterinary Pathology, eds. McGavin M, Carlton W, Zachary J, 3rd ed., pp. 214-220. Mosby, St. Louis, MO, 2001

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