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Bacterial Valvular Endocarditis in a Black Bear from Labrador

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ABSTRACT: In fall 1991, a radio-collared black bear (*Ursus americanus*) in northern Labrador (Canada) died from valvular endocarditis caused by coagulase-positive *Staphylococcus aureus*, with widespread dissemination of the infection to other organs shortly before death. Apparently, this is the first reported case of bacterial valvular endocarditis in a wild black bear.

Key words: Bacterial valvular endocarditis, black bear, case report, *Staphylococcus aureus*, *Ursus americanus*.

A unique barren-ground population of black bears (*Ursus americanus*) is found in northern Labrador (Canada) between ca. 57°30′N and 60°00′N (Veitch and Harrington, 1996). Twenty-nine bears (21 males; 8 females) were radio-collared between May 1989 and November 1993 as part of a study on the ecology and behaviour of this population. On 8 November 1991, one of these animals, a 9-yr-old male, was found dead at the west end of Okak Bay, on the Labrador coast (57°31′N, 62°29′W). This animal had been immobilized six times to deploy and remove satellite-tracked radiocollars (Telonics Inc., Mesa, Arizona, USA) between 18 May 1989 and 7 April 1991. Its age had been determined from a count of cementum annuli in the lower left first premolar extracted at first capture. During the last capture preceding death, dermatitis was observed beneath the collar. It was treated with Hibitane™ topical antibacterial and antifungal ointment (Ayerst Laboratories, Montreal, Quebec, Canada) and an intramuscular injection of 20 ml Penlong XL™ antibiotic solution (Rogar/STB, London, Ontario, Canada). A 24 hr activity sensor built into the radio-collar indicated that the bear had been alive until at least 19 October 1991.

The frozen carcass was shipped to the Atlantic Veterinary College (University of Prince Edward Island, Charlottetown, Prince Edward Island, Canada) and necropsied on 18 November 1991. The animal weighed 105.5 kg and was in good body condition with normal muscle mass and abundant fat reserves. The abdominal cavity contained approximately 1 l of blood. The digestive tract contained little ingesta. The left perirenal area was expanded to approximately three times that of the right side due to marked retroperitoneal hemorrhage. In the affected area, clotted blood was attached to the renal capsule, and hemorrhage was present within the renal parenchyma. The cranial pole of the right kidney was also hemorrhagic. The right and left aortic semilunar valvulae had small irregular friable masses (approximately 1.0–1.5 × 1.5 × 0.3 cm) consistent with deposits of fibrin attached to their free edges, and a small area of endocardium beneath the dorsal valvula was roughened (Fig. 1). Gross lesions were not detected in other organs.

Brain, heart, lung, liver, kidney, adrenal, and skeletal muscle were collected for microscopic examination. These tissues were fixed in 10% neutral buffered formalin, dehydrated in graded alcohol and xylene, and embedded in paraffin blocks; 5 μm-thick sections were stained with hematoxylin and eosin (Luna, 1968). Selected sections were also stained with a modified Brown and Brenn Gram stain (Luna, 1968). Sections of cerebrum were treated with an avidin-biotin complex immunoperoxidase stain (Haines and Chelack, 1991), using polyclonal antisera against *Toxoplasma gondii* (rabbit polyclonal antiserum, BioGenex®, San Ramon, California, U.S.A.) and *Sarcocystis cruzi* (antiserum a

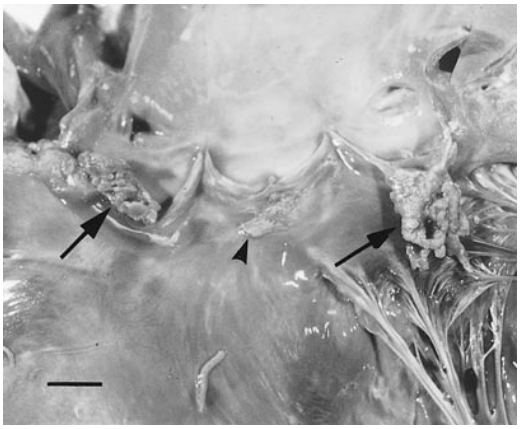

FIGURE 1. Aortic valve of a black bear, showing deposits of fibrin on the free edges of the right and left semilunar valvulae (arrows) and a small area of endocarditis beneath the dorsal valvula (arrowhead). Bar = 1 cm.

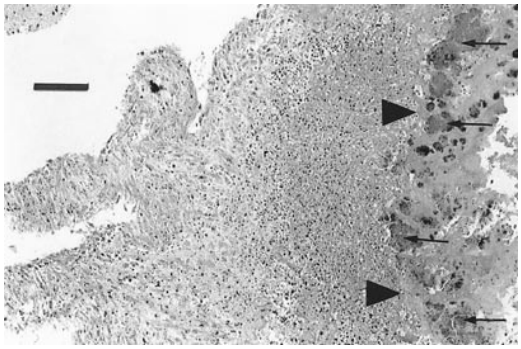

FIGURE 2. Low magnification of aortic valvula with an exudate of fibrin (arrowheads) enmeshed with inflammatory cells and bacterial colonies (arrows) attached to its ulcerated endocardial surface. H&E. Bar = 100 μm.

gift from M. Geffery, Central Veterinary Laboratory, Waybridge, U.K.) and a monoclonal antiserum against *Neospora caninum* (antiserum clone number 6G7 a gift from D. Lindsay, Auburn University, College of Veterinary Medicine, Alabama U.S.A.). Samples of lung, kidney, and aortic valve were cultured at 35 C in 5% CO_2 on 5% sheep blood and MacConkey agars (Oxoid Inc., Nepean, Ontario, Canada), and bacterial isolates were identified to species (Quinn et al., 1994).

Significant microscopic lesions were found in the heart, kidneys, brain, and adrenals. Abundant fibrin was attached to the aortic valve, particularly near the free edges of the semilunar valvulae where there was complete destruction of the normal architecture. Degenerate and necrotic inflammatory cells, primarily neutrophils, were enmeshed in the fibrin together with a large number of Gram-positive coccoid bacteria forming small colonies (Fig. 2). The underlying tissue was infiltrated by moderate numbers of neutrophils and macrophages. Similar lesions involved the adjacent region of the endocardium. Some sections of kidney had locally extensive areas of infarction containing large colonies of Gram-positive cocci and surrounded by a thick zone of neutrophils. The uriniferous spaces of many glomeruli were infiltrated by large numbers of neutrophils, and their glomerular tufts were occluded by multiple fibrin thrombi. A few large vessels at the corticomedullary junction also were partially occluded by fibrin thrombi, and there was partial or complete fibrinoid necrosis of their walls. The brain contained several small aggregates of neutrophils and macrophages, primarily within the perivascular spaces of small blood vessels, but also scattered throughout the neuropil. A few of the affected blood vessels contained fibrin thrombi. Large colonies of Gram-positive cocci were present within the adrenal cortex but were not associated with an inflammatory reaction and, therefore, were assumed to have resulted from terminal septicemia.

Incidental microscopic findings included several protozoal cysts within the cerebral neuropil and a large number of nematode larvae encysted within the skeletal muscle. These parasites were not associated with an inflammatory response. The nematode larvae resembled *Trichinella* sp., but they were not further identified. Trichinosis is a zoonotic disease occasionally reported in black bears from Labrador (Butler and Khan, 1992). Immunohistochemical staining of the cerebral protozoal cysts was negative for *T. gondii* and *N. caninum,* but positive for *Sarcocystis* sp. Sar-

cocystosis is commonly reported in wildlife, and sarcocysts are frequently identified incidentally in skeletal muscle and brain of a wide variety of mammals, birds and cold-blooded animals (Dubey et al., 1998).

Coagulase-positive *Staphylococcus aureus* was isolated in large numbers from the aortic valve and kidneys and in moderate numbers from the lungs. *Escherichia coli* was isolated in small numbers from the kidneys, and *Proteus* sp. was isolated in small numbers from the lungs and kidneys. The latter two bacterial species were interpreted as postmortem contaminants.

Septicemia associated with severe, subacute, fibrinosuppurative, valvular endocarditis caused by a coagulase-positive *S. aureus* was diagnosed as the cause of the bear's death. The intra-abdominal accumulation of blood and the abundant left perirenal hemorrhage were thought to have resulted from rupture of a major renal blood vessel weakened by septic embolism, although this could not be confirmed grossly or microscopically. Infectious valvular endocarditis is a well documented disease in humans (Silver, 1983; Anderson and Becker, 1992) and domestic animals, including dogs and cats (Drazner, 1979; Taboada and Palmer, 1989; Elmwood et al., 1993; O'Grady, 1995), rabbits (Francioli and Freedman, 1979), and cattle, horses, and swine (Kasari and Roussel, 1989; Roussel and Kasari, 1989; Maxon and Reef, 1997). However, according to O'Grady (1995), this condition is a rare cause of clinical heart disease in dogs and cats, which suggests that these carnivores are less susceptible than other domestic species to infection of cardiac valves. Infectious valvular endocarditis also has been infrequently reported in free-ranging wild animals, including a raccoon (*Procyon lotor*) (Diters and Ryan, 1980), a bald eagle (*Haliaeetus leucocephalus*) (Jessup, 1980), and a Thern sea otter (*Enhydra lutris*) (Joseph et al., 1990). To our knowledge, this disease has not been previously documented in wild black bears. The signalment and etiopathogenesis of infectious valvular endocarditis in the domestic dog have some similarities to those in the black bear of this report. Male dogs are affected twice as often as females, and the incidence of the disease is higher in dogs greater than 4 yr of age, especially in large breeds (Taboada and Palmer, 1989; Elmwood et al., 1993; O'Grady, 1995). The left side of the heart is most commonly affected, and the mitral valve is involved at a slightly higher rate than the aortic valve, with either *S. aureus,* β-hemolytic *Streptococci* or *E. coli* being isolated from the lesions (O'Grady, 1995). However, in humans, when the cardiac valves are normal prior to infection, the aortic valve is the one most often involved (Silver, 1983). *S. aureus* can produce proteases capable of damaging endothelial surfaces (O'Grady, 1995), and, in humans, coagulase-positive *S. aureus* is reported to be a virulent organism, producing fulminant infections (Silver, 1983). These factors may help explain the distribution of cardiac lesions and the relatively rapid death of this black bear. Possible sources of bacteria include infections of the skin, respiratory tract, urogenital tract, and oral cavity (Drazner, 1979). Dental procedures, including tooth extractions, are another well documented route of infection (Drazner, 1979). However, determining the portal of entry can be difficult (Silver, 1983; Roussel and Kasari, 1989), and the origin of the infection, as in this case, is often not identified (Jessup, 1980; Anderson and Becker, 1992; Maxon and Reef, 1997). It was tempting to suggest the valvular infection in this bear was related to problems connected with its last capture (i.e., collar-associated dermatitis or contaminated injection site from an immobilization dart). However, the long time delay between the immobilization procedure and the bear's death (i.e., April to at least October), the absence of supporting lesions at necropsy (i.e., no dart-induced abscess or continued cervical dermatitis), and the valvular lesion's lack of chronicity (i.e., it was subacute not chronic

inflammation) made this association highly unlikely. The sequelae of infectious valvular endocarditis most often result from embolization. The kidneys and spleen are the organs most commonly affected by emboli in the dog; however, the left cardiac ventricle, brain, intestine, and other organs also may be affected (O'Grady, 1995). Infarcts are the most common renal lesion, and hemorrhage into the urinary tract as well as abnormal bleeding associated with thrombocytopenia, possibly related to disseminated intravascular coagulation, are reported (Taboada and Palmer, 1989; Elmwood et al., 1993). These factors could explain the marked perirenal hemorrhage observed in this black bear.

In light of the information obtained from this case, prophylactic antibiotic therapy should be warranted in black bear research when animals are immobilized, collar-associated dermatitis is identified, and/or teeth are extracted. Because the bacterial species most likely to be associated with valvular endocarditis in black bears are unknown, treatment should be with a broad-spectrum, bacteriocidal antibiotic.

LITERATURE CITED

ANDERSON, R. H., AND A. E. BECKER. 1992. The heart—structure in health and disease. Gower Medical Publishing, New York, New York, 266 pp.

BUTLER, C. E., AND R. A. KHAN. 1992. Prevalence of *Trichinella spiralis* in black bears (*Ursus americanus*) from Newfoundland and Labrador, Canada. Journal of Wildlife Diseases 28: 474–475.

DITERS, R. W., AND M. J. RYAN. 1979. Vegetative endocarditis with generalized bacterial embolism in association with phlegmon induced by *Dracunculus insignis* in a raccoon. Cornell Veterinarian 70: 213–317.

DRAZNER, F. H. 1979. Bacterial endocarditis in the dog. Compendium on Continuing Education 1: 918–924.

DUBEY, J. P., M. J. TOPPER, AND F. B. NUTTER. 1998. Muscular *Sarcocystis* infection in a bear (*Ursus americanus*). The Journal of Parasitology 84: 452–454.

ELMWOOD, C. M., M. A. COBB, AND R. L. STEPIEN. 1993. Clinical and echocardiographic findings in ten dogs with vegetative bacterial endocarditis. Journal of Small Animal Practice 34: 420–427.

FRANCIOLI, P. B., AND L. R. FREEDMAN. 1979. Streptococcal infection of endocardial and other intravascular vegetations in rabbits: Natural history and effect of dexamethasone. Infection and Immunity 24: 483–491.

HAINES, D. M., AND B. J. CHELACK. 1991. Review article. Technical considerations for developing enzyme immunohistochemical staining procedures on formalinparaffin-embedded tissues for diagnostic pathology. Journal of Veterinary Diagnostic Investigation 3: 101–112.

JESSUP, D. A. 1980. Valvular endocarditis and bacteremia in a bald eagle. Modern Veterinary Practice 6: 49–51.

JOSEPH, B. E., T. R. SPRAKER, AND G. MIGAKI. 1990. Valvular endocarditis in a Thern sea otter (*Enhydra lutris*). Journal of Zoo and Wildlife Medicine 21: 88–91.

KASARI, T. R., AND A. J. ROUSSEL. 1989. Bacterial endocarditis. Part 1. Pathophysiologic, diagnostic and therapeutic considerations. The Compendium on Continuing Education 11: 655–671.

LUNA, L. G. (EDITOR). 1968. Manual of histologic staining methods of the Armed Forces Institute of Pathology. McGraw-Hill Company, New York, New York, 258 pp.

MAXON, A. D., AND V. B. REEF. 1997. Bacterial endocarditis in horses: Ten cases (1984–1995). Equine Veterinary Journal 29: 394–399.

O'GRADY, M. R. 1995. Infective endocarditis. *In* Textbook of veterinary internal medicine, 4th Edition. S. J. Ettinger and E. C. Feldman (eds.). W. B. Saunders Company, Toronto, Canada, pp. 954–959.

QUINN, P. J., M. E. CARTER, B. K. MARKEY, AND G. R. CARTER. 1994. Clinical veterinary microbiology. Wolfe Publishing, Mosby-Year Book Europe Ltd., London, UK, 648 pp.

ROUSSEL, A. J., AND T. R. KASARI. 1989. Bacterial endocarditis in large animals. Part II. Incidence, causes, clinical signs and pathologic findings. The Compendium of Continuing Education 11: 769–773.

SILVER, M. D. 1983. Infective endocarditis. *In* Cardiovascular pathology, 6th Edition. M. D. Silver (ed.). Churchill Livingstone, Inc., New York, New York, pp. 517–549.

TABOADA, J., AND G. H. PALMER. 1989. Renal failure associated with bacterial endocarditis in the dog. Journal of the American Animal Hospital Association 25: 243–251.

VEITCH, A. M., AND F. H. HARRINGTON. 1996. Brown bears, black bears, and humans in northern Labrador: An historical perspective and outlook to the future. Journal of Wildlife Research 1: 245–250.

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