Streptococcus agalactiae endocarditis with embolization in a dog

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An 8-year-old, neutered, mixed Labrador retriever was presented at the Hôpital des petits animaux, Faculté de médecine vétérinaire in Saint-Hyacinthe with a problem of partial anorexia and lethargy of about a week's duration.

The dog weighed 55 kg and had a bilateral mucopurulent ocular discharge. The rectal temperature was 41°C. Hematological examination revealed a leucocytosis with a mature neutrophilia. Biochemical analysis was compatible with a profile of metabolic acidosis. A blood culture done on the day of presentation was positive for *Streptococcus agalactiae* (Lancefield group B). The animal was given IV fluid therapy (Ringer's lactate), as well as antibiotics (Cefazoline, 1800 mg, IV, q8h). On radiography, the abdomen appeared normal, but changes compatible with a diagnosis of either osteomyelitis or neoplasia were observed at the level of the 3rd and 4th sternebrae.

Forty-eight hours after initiation of the treatment, the hyperthermia had resolved, but the overall clinical status of the animal was worsening. Another blood culture was done and was negative for *S. agalactiae*. The dog also had an episode of epistaxis. The platelet count had dropped to 114 000 platelets/L, and the one-stage prothrombin and partial thromboplastine times were prolonged. Disseminated intravascular coagulation was suspected. Because of the poor prognosis, the owner elected for euthanasia.

During postmortem examination, the following lesions were observed: visible infarcts in the kidneys, spleen, and heart; petechiae on the surface of the kidneys; suffusion and hemorrhages in the adipose and muscular tissue of the thorax, on the parietal pleura, and in the subcutaneous connective tissue; thrombotic valvular endocarditis (aortic valves) and cardiac valve vegetations (left auriculoventricular valves). A routine bacteriological examination of a sample collected at the level of the affected sternebrae was positive for *S. agalactiae*. Histological examination revealed the presence of fibrinous microthrombi in the liver, an old infarcts with recanalized thrombi in the kidney, heart, and spleen; a moderate multifocal glomerulonephritis; and recent microinfarcts in the brain. Sections of the aortic leaflets colored with Gram's stain showed numerous gram-positive cocci. Based on these findings, we made a diagnosis of cardiac valve vegetation due to *S. agalactiae,* with chronic thromboembolism and terminal disseminated intravascular coagulation.

A review of the veterinary literature of the last 10 y did not show any report on endocarditis linked specifically to *S. agalactiae* in animals. Endocarditis caused by Lancefield group B streptococci has been reported on a few occasions in humans (1–4). One paper dealing with 5 cases over a period of 13 y mentioned some interesting facts: in 4 of the 5 cases, the aortic valve was involved, whereas in the last case the mitral valve was infected (4). In the present report, the aortic and left auriculoventricular valves were affected. Systemic emboli occur quite frequently with group B streptococci endocarditis because of the friable nature of the cardia vegetations (1–3). We hypothesize that chronic embolization was occurring in the present case, which would explain the presence of recent thromboembolism along with old infarcts and recanalized thrombi.

Streptococcus agalactiae is a microorganism frequently isolated in bovine mammary gland infections (5). In companion animals, isolation of *S. agalactiae* has been documented on only a few occasions. Davies *et al* (6) isolated β-haemolytic streptococci from 22 of 138 dogs that died of fading-puppy syndrome ranging in age from 1 to 8 y. Two of the 22 isolates were Lancefield group B streptococci. The specific isolation site of these isolates was not specified. Kornblatt *et al* (7) reported the isolation of *S. agalactiae* from the internal organs of puppies from 2 different litters, which had died in the neonatal period, as well as from the vaginal discharge of 1 dame and from a vaginal swab taken from the other. In humans, Lancefield group B streptococci are considered to be pathogenic organisms responsible for severe neonatal infections, such as, meningitis, pneumonia, and septicemia; they have also been isolated form the vagina and cervix of women (8,9). In the cat, 2 isolations of *S. agalactiae* have been reported (10); one from the abdominal dialysate fluid of an adult male, and the other from the uterus of a queen that had delivered kittens a few days earlier.

Although the occurrence of *S. agalactiae* as a cause of endocarditis in the dog is a rare event, it is interesting to note that it presents numerous similarities to the conditions occurring in humans.

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References

1. Vilde JL, Roujeau JC, Bure A, Verliac F, Bastin R. Infections graves et septicémies à streptocoque du groupe B chez l'adulte: à propos de quatre observations, dont une endocardite tricuspidienne compliquée de glomérulonéphrite. Sem Hôp Paris 1974; 50: 355–362.
2. Lerner PI, Gopalakrishna KV, Wolinsky E, McHenry MC, Tan JS, Rosenthal M. Group B streptococcus *(S. agalactiae)* bacteremia in adults: analysis of 32 cases and review of the literature. Medicine 1977; 56: 457–473.
3. Hager WD, Speck EL, Mathew PK, Boger JN, Wallace WA. Endocarditis with myocardial abscesses and pericarditis in an adult: group B *Streptococcus* as a cause. Arch Intern Med 1977; 137: 1725–1728.
4. Backes RJ, Wilson WR, Geraci JE. Group B streptococcal infective endocarditis. Arch Intern Med 1985; 145: 693–696.
5. Erickson ED. Streptococcal disease. In: Howard JL, ed. Current Veterinary Therapy 3: Food Animal Practice. Philadelphia: WB Saunders, 1993: 575–578.
6. Davies ME, Skulski G. A study of beta-haemolytic streptococci in the fading puppy in relation to canine virus hepatitis infection in the dam. Br Vet J 1956; 112: 404–415.
7. Kornblatt AN, Adams RL, Barthold SW, Cameron GA. Canine neonatal deaths associated with group B streptococcal septicemia. J Am Vet Med Assoc 1983; 183: 700–701.
8. Baker CJ, Webb BJ, Barret FF. Antimicrobial susceptibility of group B streptococci isolated from a variety of clinical sources. Antimicrobial Agents Chemother 1976; 10: 128–131.
9. Persson KMS, Forsgren A. Antimicrobial susceptibility of group B streptococci. Eur J Clin Microbiol Infect Dis 1986; 5: 165–167.
10. Dow SW, Jones RL, Thomas TN, Linn KA, Hamilton HB. Group B streptococcal infection in two cats. J Am Vet Med Assoc 1987; 190: 71–72.