

Canine Streptococcal Toxic Shock Syndrome

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Streptococci are a family of gram-positive bacteria some of which can cause either localized or systemic infections in both humans and animals. Some strains rarely cause disease and are often considered to be commensal (normal) inhabitants of the skin and mucosal surfaces (oral, nasal, intestinal), while other strains are capable of causing serious or even life-threatening infections. In dogs, *Streptococci* (Strep) are known for their ability to occasionally cause septicemia (blood born infections) in puppies and a range of localized diseases in adults.

In the early 90's, *Streptococci* (Group A, β -hemolytic Strep) emerged as the cause of a previously unrecognized disease in humans. The clinical disease became known as Streptococcal Toxic Shock Syndrome (STSS) because it closely mimics the better known "Toxic Shock" in women caused by toxin producing strains of *Staphylococci* (Staph). Rapid onset, high fever, hypotension, and shock are prominent characteristics of STSS in humans.

At approximately the same time, a number of unusual cases of necrotizing fasciitis caused by *Streptococci* ("flesh eating Strep") were also reported in humans. This syndrome relates to a very aggressive and rapidly advancing infection of subcutaneous tissues with extensive tissue destruction and high mortality rates.

In 1996, Miller and Prescott reported on a series of seven dogs from southern Ontario that had severe systemic disease and shock associated with infection with β -hemolytic *Streptococcus canis* (Group G). In four of these dogs the infection was associated with necrotizing fasciitis. As a result of surgical debridement, supportive medical care, and treatment with antibiotics, all of these dogs survived. In contrast, all three dogs with streptococcal shock without associated necrotizing fasciitis died or were euthanized within 48 hours. The lungs were considered the primary site of infection in two of these dogs as their clinical signs were related to respiratory distress and shock. Historically, similar disease outbreaks have been reported by Garnett et al (1982) in a group of research dogs, in captive coyotes by Gates and Green (1979), and in racing Greyhounds by Sundberg et al (1981).

Multiple outbreaks of fatal STSS occurred in racing Greyhounds in 1992 and again in January/February of 1999. Cases of STSS have recently been reported in other dog breeds following dog shows or performance events. There are also occasional reports of STSS occurring in a single pet household with no apparent exposure to other dogs.

The reason for the emergence/re-emergence of canine STSS/NF is unclear and very little is known about transmission, prevention, or immunity following possible exposure. Dogs that develop STSS appear to be normal and healthy prior to being recognized as very sick only a short time later. The course of the disease, from initial recognition of illness to death, can be as short as 6 hours. It is not uncommon for the dog to appear normal at bedtime and to be found dead the next morning.

Typically, infected dogs are found in lateral recumbence, either being too weak to move or experiencing rigidity with mild convulsions. Rapid, uncontrolled fine muscle fasciculations are often noted. A consistent and important clinical finding is a very high temperature (105° F). Treatment at this point with injectable *Streptococci*-specific antibiotics (clindamycin or crystalline penicillin-G) is important in order to increase the likelihood of recovery. As the disease progresses, a deep, non-productive cough, typical of pulmonary edema, develops. Rapid, spontaneous hemorrhaging, typical of disseminated intravascular coagulation, develops. This can be associated with coughing up blood, bleeding from the nose, severe bruising of the skin, and in some cases bloody diarrhea. Profound hypotension and toxic cardiomyopathy may develop. Antibiotics, even in combination with aggressive shock therapy, are generally not sufficient to save these dogs. Characteristic necropsy findings include hemorrhagic pneumonia; however, successful isolation of the *Streptococci* organism from the live patient or necropsy tissues may be difficult.

Unfortunately, to date, advances in detection and prevention have been few. No vaccine has been developed, no medication has been found to be effective in preventing the infection, and no test has been beneficial at identifying those animals at risk.

References

- Garnett NL, Eydeloth RS, Swindle MM, Vonderfecht SL, Strandberg JD, Luzarraga MB. Hemorrhagic *streptococcal pneumonia* in newly procured research dogs. *J Am Vet Med Assoc*. 1982 Dec 1; 181(11): 1371-4.
- Gates NL, Green JS. *Epizootic streptococcal pneumonia* in captive coyotes. *J Wildl Dis*. 1979 Oct; 15(4): 497-8.
- Miller CW, Prescott JF, Mathews KA, Betschel SD, Yager JA, Guru V, DeWinter L, Low DE. Streptococcal toxic shock syndrome in dogs. *J Am Vet Med Assoc*. 1996 Oct 15; 209(8): 1421-6.
- Sundberg JP, Hill D, Wyand DS, Ryan MJ, Baldwin CH. *Streptococcus zooepidemicus* as the cause of septicemia in racing greyhounds. *Vet Med Small Anim Clin*. 1981 Jun; 76(6): 839-42.