Staphylococcus intermedius cellulitis and toxic shock in a dog

C. Girard, R. Higgins

Abstract — A Labrador retriever was examined for sudden lameness and cellulitis of the right forelimb. Bacterial culture of the dermis yielded a large number of *Staphylococcus intermedius*. The association of this bacterium with toxic shock is discussed.

Résumé — Cellulite à *Staphylococcus intermedius* et choc toxique chez un chien. Un Labrador a été examiné pour une boiterie soudaine du membre antérieur droit associée à une cellulite. *Staphylococcus intermedius* a été isolé du derme. L'association entre cette bactérie et un choc toxique est discutée.

(Traduit par les auteurs)

Can Vet J 1999; 40: 501–502

A 3-year-old, Labrador retriever was examined for sudden lameness. The right forelimb was hot, painful, swollen, and nonweight-bearing. A cutaneous serosanguineous exudate was noted at the level of the metacarpus. The mucous membranes were pale and the capillary refill time was prolonged. The animal had a fever (40.5°C) and was depressed. Hematology and clinical chemistry were not performed. Cefazolin (Ancef, Smithkline Beecham Pharma, Mississauga, Ontario) and ketoprofen (Anafen, Rhône Merieux, Mississauga, Ontario) were given, IV, without success. The dog became comatose, developed convulsions, and died 48 h after the onset of clinical signs. He was submitted for necropsy at the Diagnostic Service of the Faculté de médecine vétérinaire of the Université de Montréal.

On postmortem examination, moderate autolytic changes were present. The right forelimb was markedly swollen and there was an ulcerated suppurative area craniomedial to the carpus (Figure 1). The subcutaneous tissues of the limb and the adjacent thorax were thickened by serosanguineous fluid. The underlying fascia and skeletal muscles appeared normal. A small amount of mucohemorrhagic material, which seemed to be digested blood, was present in the trachea. The lungs were congested and edematous. Mucohemorrhagic material was also present in the stomach. Tissue samples were fixed in formalin and routinely processed for microscopic examination by using hematoxylin-phloxin-saffron (HPS) and Gram stains.

On microscopic examination, there was severe diffuse inflammation of the deep dermis and subcutaneous tissues at the level of the carpus (Figure 2). Degenerate inflammatory cells were associated with numerous gram-positive cocci. Many veins of the dermis exhibited vasculitis, characterized by necrotic inflammatory cells within the intima and the media, and thrombosis. There were diffuse hemorrhages and edema in the dermis and subcutis. Proximal to the carpus, changes were mainly vascular, with hemorrhages and edema of the dermis and subcutaneous tissues, and fibrinous thrombi in some vessels of the deep dermis. Fibrinous thrombi were also observed in trabecular veins and venous sinuses of the red pulp of the spleen, in portal veins, and in the reticularis of the adrenal glands. Severe generalized pulmonary congestion and edema were present. The liver and spleen were congested. Keratinized epithelial cells and necrotic material were observed in some bronchi and alveoli.

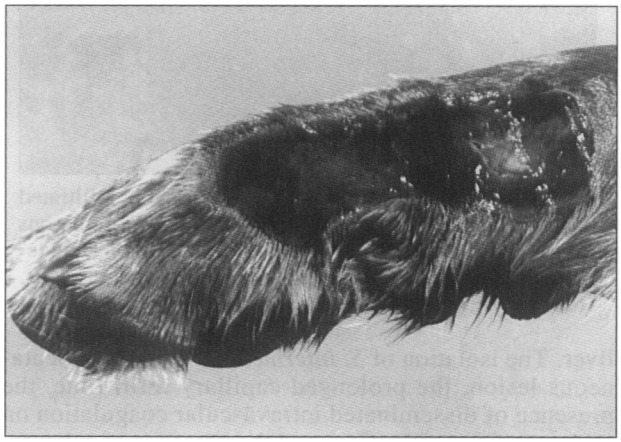

Figure 1. Severe cellulitis caused by *Staphylococcus intermedius* in right forelimb of a dog; the skin has been incised to demonstrate the thickening of the subcutaneous tissues by a serosanguineous exudate.

Bacteriological examination of tissues revealed the presence of a large number of *Staphylococcus intermedius* in the dermis; there was no evidence of *Streptococcus canis*. Strict anaerobes were not found in the dermis. Only a few contaminants were isolated from lungs and liver. Since the animal was dead and no other animal was involved, antimicrobic sensitivity testing of the *S. intermedius* isolate was not carried out.

The present case involving *S. intermedius* is very similar to those associated with Lancefield group G *Streptococcus canis*, which is known to produce many virulence factors similar to those of group A streptococci (1,2). In humans, *Staphylococcus aureus* has been associated with toxic shock syndrome for many years (3,4). Trauma was not documented in this case. Septic shock is usually associated with gram-negative septicemia, but some gram-positive septicemias have similar consequences (5). *Staphylococcus* spp. have already been associated with mastitis, septicemia, and fatal endotoxemia in a dog (6). However, septicemia could not be confirmed in the present case by culture of lungs and

Department of Pathology and Microbiology, Faculté de médecine vétérinaire, Université de Montréal, P.O. Box 5000, St. Hyacinthe, Quebec J2S 7C6.

Address correspondence and reprint requests to Dr. Christiane Girard.

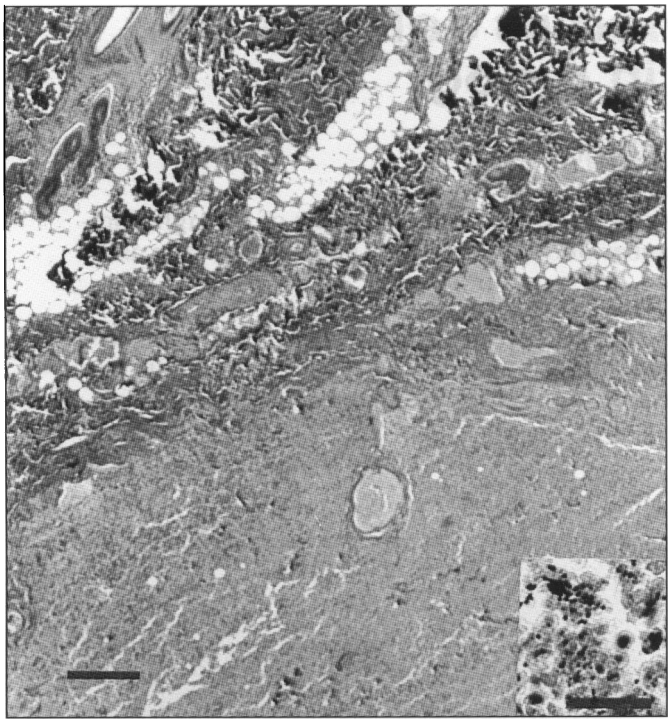

Figure 2. The subcutaneous tissue is massively infiltrated by necrotic inflammatory cells associated with numerous cocci (Inset: bar = 50 μm). The overlying dermis is edematous, and numerous hemorrhages are present. Hematoxylin-phloxin-saffron (HPS) stain. bar = 100 μm.

liver. The isolation of *S. intermedius* from the subcutaneous lesion, the prolonged capillary refill time, the presence of disseminated intravascular coagulation on microscopic examination, and the presence of subcutaneous necrosis are suggestive of toxic shock, as already defined for streptococcal toxic shock syndrome (7).

Staphylococcus aureus and group A streptococci have been shown to produce exotoxins associated with toxic shock syndrome that are known as T cell superantigens (3,4). Superantigens are able to activate a larger number of T cells compared with conventional antigens, because their binding to V5B regions does not involve the antigen recognition site of the T cell. Therefore, the interaction between the antigen-presenting cells and T cells occurs without regard for antigen specificity (3,4). Stimulated T cells release massive quantities of cytokines that cause the changes associated with toxic shock syndrome, such as fever, hypotension, and multiorgan failure (4). *Staphylococcus aureus* produces several different superantigens, such as enterotoxins A, B, C, D, and E, and toxic shock syndrome toxin-1 (3). Each of these enterotoxins, for the most part, binds to a unique subset of T cells (3).

In previous reports, a large proportion of the *S. intermedius* strains isolated from dogs were shown to produce one or more enterotoxins, comprising toxic shock syndrome toxin (8–10). Production of enterotoxins was not examined for this strain.

This is, to our knowledge, the first case of toxic shock associated with *S. intermedius* cellulitis reported in a dog. Aggressive surgical debridement, supportive medical care and antibiotics are recommended for treatment of streptococcal shock and fasciitis in dog (2), and the same approach should be taken by veterinarians who encounter *S. intermedius* cellulitis and toxic shock in the dog.

Acknowledgments

We thank Drs. Annabelle Cyr and Patrice Duchesne for submission of this case. CVJ

References

1. Prescott JF, Mathews K, Gyles CL, et al. Canine streptococcal toxic shock syndrome in Ontario: An emerging disease? Can Vet J 1995; 36: 486–487.
2. Miller CW, Prescott JF, Mathews K, et al. Streptococcal toxic shock syndrome in dogs. J Am Vet Med Assoc 1996; 209: 1421–1426.
3. Goodglick L, Braun J. Revenge of the microbes: superantigens of the T and B lineage. Am J Pathol 1994; 144: 623–636.
4. Kiska DL. Staphylococcal and streptococcal toxic shock syndrome. Clin Microbiol Newsletter 1997; 19: 33–37.
5. Cotran RS, Kumar V, Robbins SL. Hemodynamic disorders, thrombosis, and shock. In: Cotran RS, Kumar V, Robbins SL, Schoen FJ, eds. Pathologic Basis of Disease. 5th ed. Philadelphia: WB Saunders, 1994: 93–123.
6. Dernell WS, Kreeger J. Peracute, necrotizing mastitis as a cause of fatal septicemia and endotoxemia in a dog. Canine Pract 1992; 17: 25–29.
7. US Department of Health and Human Services, Centers for Disease Control and Prevention, Working Group on Severe Streptococcal Infections. Defining the group A streptococcal toxic shock syndrome; rationale and consensus definition. J Am Med Assoc 1993; 269: 390–391.
8. Fukuda S, Tokuno H, Ogawa H, et al. Enterotoxigenicity of *Staphylococcus intermedius* strains isolated from dogs. Zbl Bakt Hyg (A) 1984; 258: 360–367.
9. Almazan J, de la Fuente R, Gomez LE, Suarez G. Enterotoxin production by strains of *Staphylococcus intermedius* and *Staphylococcus aureus* isolated from dog infections. Zbl Bakt Mikrobiol Hyg (A) 1987; 264: 29–32.
10. Hirooka EY, Müller EE, Freitas JC, Vicente E, Yoshimoto Y, Bergdoll MS. Enterotoxigenicity of *Staphylococcus intermedius* of canine origin. Int J Food Microbiol 1988; 7: 185–191.