Necrotizing fasciitis is a rapidly spreading, bacterial, soft-tissue infection reported in both humans and dogs. A review of the pathophysiology, clinical findings, diagnosis, and treatment of necrotizing fasciitis is presented, with the goal of familiarizing veterinarians with this uncommon but potentially fatal condition. A case report highlighting the fulminant course of this disease is also included. J Am Anim Hosp Assoc 2005;41:104-109.

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Introduction
Necrotizing fasciitis is a fulminant, rapidly progressive, potentially life-threatening, bacterial infection of the fascial and subcutaneous tissues. In addition to extensive and severe local tissue damage, many animals with necrotizing fasciitis have concurrent systemic signs of shock. Although the etiology of necrotizing fasciitis in humans is commonly polymicrobial, group A streptococcus is believed to be the single most widely recognized causative species and is present in up to 71% of all human cases. In nine reported cases of canine necrotizing fasciitis, beta-hemolytic streptococcus was consistently isolated. Further serological classification of the bacteria as Lancefield group G was reported in a series of eight cases from Canada, and the isolates had characteristics typical of \textit{Streptococcus canis}. Although group G streptococcus is a rare cause of necrotizing fasciitis in humans, it appears to be the major etiological agent of this clinical entity in dogs.

Necrotizing fasciitis in people has been called the “flesh-eating disease” and has been the focus of much publicity and media attention in recent years, thus raising awareness of the disease among health-care professionals and the general population. In contrast, veterinarians may be less aware of this condition in animals, as there are few reports of necrotizing fasciitis in the veterinary literature. Recognition of necrotizing fasciitis in a clinical setting is further impaired by the lack of pathognomonic physical examination findings. As immediate surgical therapy is paramount in the successful treatment of necrotizing fasciitis in both humans and dogs, knowledge of the clinicopathological findings of necrotizing fasciitis in animals is essential for early diagnosis, expeditious surgical debridement, and improved outcomes. The purpose of this review is to better acquaint veterinarians with the pathophysiology, clinical signs, diagnosis, and treatment of necrotizing fasciitis in dogs. An illustrative case report is also included.
Pathophysiology

Group G streptococci are commensal organisms in dogs, and they may infect the fascial tissues when the normal skin barrier is compromised. Most affected humans and dogs have a history of mild injury to the skin in the days preceding the onset of clinical signs. Blunt trauma, without disruption to the skin, may also predispose to the development of necrotizing fasciitis in humans. The potential inciting injuries reported in previous cases of canine necrotizing fasciitis include minor dog bites, minor trauma, and, in one case, a skin infection. In some dogs, there was no history of a previous injury. People with diabetes mellitus, other immunosuppressive conditions, or peripheral vascular disease may be at greater risk for the development of necrotizing fasciitis. A similar association has not been made in dogs.

Necrotizing fasciitis had evidence of hypotension from either STSS or septic shock. The pathogenesis of STSS is not fully understood, but it is thought to involve massive cytokine release secondary to toxin-induced vasoconstriction or thrombosis of the nourishing vessels. Systemic signs of illness and shock, including fever, tachycardia, and peripheral hypoperfusion, are common in humans and dogs with necrotizing fasciitis. The term streptococcal toxic shock syndrome (STSS) describes the rapidly progressive, hypotensive shock and multiorgan failure that can accompany severe streptococcal infections in humans and dogs. Streptococcal toxic shock syndrome can occur secondary to streptococcal necrotizing fasciitis, or it may occur independently of necrotizing fasciitis as a consequence of streptococcal infections associated with other organs. Half of all human cases of necrotizing fasciitis have concurrent STSS. Most of the previously reported dogs with necrotizing fasciitis had evidence of hypotension from either STSS or septic shock. The pathogenesis of STSS is not fully understood, but it is thought to involve massive cytokine release secondary to bacterial exotoxin production, resulting in shock and organ failure. The presence of STSS in humans with necrotizing fasciitis greatly increases mortality. The mortality rate was also high in a series of six dogs with STSS associated mostly with streptococcal bronchopneumonia.

Diagnosis

The diagnosis of necrotizing fasciitis is based on a constellation of clinical signs, surgical findings, and histopathological results, combined with the positive culture of a recognized etiological agent. Because of the rapidly progressive nature of the disease, a positive outcome often hinges on initiation of therapy based on a presumptive diagnosis of necrotizing fasciitis, before culture and histopathology results are available.

Initial findings on physical examination include localized erythema, edema, and pain at the affected site, as well as systemic signs of fever and tachycardia. The majority of human cases involve an extremity, although the perineum and trunk are other common sites of involvement. Seven of nine dogs previously reported with necrotizing fasciitis had involvement of a limb. The neck and ventral thorax were the other reported sites. The most consistent and outstanding sign in people is evidence of extreme pain that is disproportionate to the appearance of the affected area. Severe pain is also present in most affected dogs. The outward appearance of an affected limb is not pathognomonic. Localized heat and swelling of the site were consistently identified on initial presentation of dogs with necrotizing fasciitis. Subsequent sloughing of the skin has been occasionally reported in both dogs and people. Cutaneous bullae formation is a relatively specific but delayed sign in people. Although cutaneous bullae are not common in affected dogs, blisters were reported on the digits of the distal pelvic limb of one dog. The differentiation between necrotizing fasciitis and other soft-tissue infections is difficult to make on initial presentation, as the severe fascial necrosis associated with necrotizing fasciitis is not apparent from the exterior. In one retrospective study, 85% of people with necrotizing fasciitis were erroneously diagnosed as having cellulitis or subcutaneous abscesses on initial presentation.

Imaging studies may be useful adjunctive tools in the diagnosis of necrotizing fasciitis. Computed tomography (CT) and magnetic resonance imaging (MRI) in people have documented fascial involvement and delineated its extent. The use of CT and MRI for the detection of necrotizing fasciitis in dogs has not been described. Although poorly sensitive for detecting necrotizing fasciitis in people, plain radiographs may reveal the presence of gas within the soft tissues. The presence of soft-tissue gas may be an unlikely finding in affected dogs, as gas-producing anaerobes are not usually involved. In one previously reported dog, a fistulogram was used to demonstrate extension of the contrast agent along the fascial planes. Although diagnostic imaging may reveal findings supportive of necrotizing fasciitis, the absence of such findings should never exclude the diagnosis.

Results of biochemical and hematological tests are nonspecific and usually reflect severe systemic inflammation, vasculitis, sepsis, or organ dysfunction. In people, common findings include azotemia, hypoglycemia, hypoalbuminemia, hypocalcemia, leukocytosis or leukopenia, and thrombocytopenia. Evidence of organ dysfunction on initial presentation is associated with an increased risk of mortality in people.

A presumptive diagnosis of necrotizing fasciitis can be made on the basis of surgical findings. The hallmark surgical finding in humans and dogs is the ease of separation of fascia from other tissues by blunt dissection, owing to fascial and subcutaneous tissue necrosis. A positive “finger test,” implying easy dissection of the subcutaneous tissue from the deep fascia by means of the surgeon’s index finger,
is a key finding of necrotizing fasciitis in people.\textsuperscript{2} The finger test may be performed prior to surgical exploration through the use of a local anesthetic and small scalpel-blade incision. A positive finger test indicates the need for immediate surgical intervention.\textsuperscript{2} Another frequent surgical finding is the presence of copious exudative fluid in the subcutaneous spaces of the affected area.\textsuperscript{1,2,12} The exudate is thin, malodorous, and resembles “murky dishwater.”\textsuperscript{2,14} Cytological evaluation of this fluid may reveal the presence of bacteria and leukocytes.\textsuperscript{1}

Although supportive clinical findings, a positive finger test, and the presence of copious subcutaneous exudate are suggestive enough of necrotizing fasciitis to warrant immediate action, culture and histopathological examination are needed to establish a definitive diagnosis.\textsuperscript{1} Tissue for culture should be taken from the leading edge of the affected region, as organisms identified at that location are likely responsible for the ongoing tissue destruction, and samples from the center of the wound are more apt to contain secondary invaders.\textsuperscript{3} Gram staining of impression smears or fine-needle aspirates may reveal chains of Gram-positive cocci, suggesting the presence of streptococci. Histopathology of tissue biopsies reveals coagulation necrosis of the fascia and subcutaneous fat, neutrophilic infiltration of the dermis and fascia, and thrombosis of the adjacent vessels.\textsuperscript{11,12} In humans, a bedside incisional biopsy with frozen-section analysis and culture is recommended for early diagnosis.\textsuperscript{1,2} In patients with clinical findings highly suggestive of necrotizing fasciitis, any substantial delay in surgical intervention until after culture and histopathology results become available is not recommended and may contribute to mortality.\textsuperscript{12}

**Treatment and Prognosis**

Successful treatment of necrotizing fasciitis hinges on early and complete surgical debridement of the necrotic tissue, often requiring multiple surgical procedures.\textsuperscript{1,2,12} Medical therapy in the absence of surgical debridement is fruitless because of poor antibiotic delivery to the affected area and the continued production of bacterial toxins.\textsuperscript{12} In humans, surgical exploration is often performed every 24 to 48 hours, and debridement is continued until the infection is completely halted.\textsuperscript{1} Amputation of a limb is sometimes required in order to achieve complete excision of affected tissue.\textsuperscript{2,10} The extent of the initial debridement influences survival, and it is considered preferable to remove too much tissue rather than risk leaving necrotic material behind.\textsuperscript{1}

Hemodynamic support, appropriate antibiotics, wound care, nutritional support, and analgesia are other essential components of therapy.\textsuperscript{2} Initial broad-spectrum antibiotic therapy is recommended while awaiting culture results.\textsuperscript{1,2} A triple combination of penicillin, an aminoglycoside, and metronidazole or clindamycin comprise the most frequently advocated antibiotic regime in humans with necrotizing fasciitis.\textsuperscript{1,2} Streptococci causing necrotizing fasciitis are generally sensitive to penicillin; however, if large numbers of slow-growing organisms are present, the antibiotic may be less effective, owing to decreased bacterial expression of penicillin-binding proteins in the stationary phase of the cell cycle.\textsuperscript{1,11} Clindamycin may be preferable because of its direct inhibitory effect on protein and exotoxin synthesis, which potentially slows toxin-mediated tissue destruction.\textsuperscript{1,11} One retrospective study of humans with necrotizing fasciitis showed a trend toward better survival with therapy that included clindamycin.\textsuperscript{15} The efficacy of enrofloxacin in dogs with necrotizing fasciitis has been questioned in the veterinary literature based on reports of poor clinical response to enrofloxacin alone, regardless of in vitro bacterial sensitivity.\textsuperscript{4,5} It has been hypothesized that the use of enrofloxacin in dogs with *Streptococcus canis* infections may contribute to the emergence of necrotizing fasciitis and STSS, as fluoroquinolones may induce bacteriophages encoding superantigen genes, thus potentially enhancing bacterial virulence.\textsuperscript{16} Monotherapy with fluoroquinolones is currently not recommended.\textsuperscript{4}

The potential role of nonsteroidal antiinflammatory drugs (NSAIDs) in the development of necrotizing fasciitis in people has been scrutinized. It has been hypothesized that NSAIDs may contribute to the rapidity with which necrotizing fasciitis progresses, secondary to NSAID-induced inhibition of neutrophil chemotaxis, phagocytosis, and bacterial activity.\textsuperscript{11} Data from retrospective studies of affected humans suggest that NSAIDs may also mask initial clinical signs, leading to a delay in the diagnosis.\textsuperscript{17} Prospective studies in humans have failed, however, to demonstrate an association between NSAID therapy and increased susceptibility to necrotizing fasciitis or a worsening of the disease.\textsuperscript{18}

The administration of oxygen at increased atmospheric pressure, known as hyperbaric oxygen therapy (HBOT), has been advocated for the postsurgical management of humans with necrotizing fasciitis. Theoretically, HBOT promotes wound healing by increasing tissue oxygenation, decreasing edema, enhancing angiogenesis, stimulating fibroblast activity, and improving leukocyte function.\textsuperscript{14} Retrospective studies have reported conflicting results, however, on the impact of HBOT on survival in people with necrotizing fasciitis.\textsuperscript{10,19} The use of HBOT has been reported in one canine case of necrotizing fasciitis.\textsuperscript{3} Although the dog survived, it was difficult to ascertain whether HBOT had an influence on the outcome.\textsuperscript{3}

Nutritional support is a crucial aspect of therapy. Large amounts of fluid and protein are lost from the exudative wounds after debridement, and there is an increased demand for protein and calories to support active tissue healing.\textsuperscript{20} Feeding of twice the basal caloric requirements has been advocated, which may improve morbidity and mortality in affected people.\textsuperscript{20}

Despite the therapeutic advances, mortality rates in humans have changed little in the past century and range from 17% to 28%.\textsuperscript{2,10,12,13} A delay in initial debridement has been repeatedly implicated as a major predictor of mortality in affected people.\textsuperscript{2,10,12} Other factors affecting outcome include extent of the initial debridement, evidence of
systemic hypoperfusion on initial presentation, and increased age. In contrast, the mortality rate in dogs with necrotizing fasciitis appears to be lower, with 88% (8/9) of reported dogs surviving to discharge.

**Illustrative Case Report**

A 4-year-old, castrated male, 57-kg Irish wolfhound was presented to the Atlantic Veterinary College with a 3-day history of right forelimb lameness. Twenty-four hours prior to the onset of lameness, the dog had fallen from a stationary vehicle onto his right side. At the onset of lameness, the referring veterinarian was able to localize pain to the right shoulder. Meloxicam (0.1 mg/kg PO q 24 hours) was prescribed. The dog showed no improvement after 2 days of therapy. Rapid development of swelling over the right shoulder and the onset of hemorrhagic diarrhea prompted the dog’s referral.

Physical examination revealed a mentally dull, thin (body condition 2/5), nonambulatory dog. The dog was febrile (40.7°C), tachycardic (120 beats per minute), and approximately 5% dehydrated. Pulse quality, mucous membrane color, and capillary refill time (CRT) were within normal limits. Diffuse, soft-tissue swelling extended from the proximal aspect of the right humerus to the carpus, and the right forelimb was markedly painful on manipulation. Digital rectal examination revealed frank blood.

Initial complete blood cell count and serum biochemical profile results showed evidence of acute inflammation, cholestasis, increased muscle enzyme leakage, mild electrolyte changes, and hypoproteinemia [see Table]. Activated clotting time and indirect systolic blood pressure measurements were normal. No abnormalities were found on thoracic and abdominal radiographs or abdominal ultrasonography. Radiographs of the right forelimb showed soft-tissue swelling and mild degenerative changes associated with the scapulohumeral joint.

A tentative diagnosis was made of cellulitis, systemic inflammatory response, and potential drug-induced gastrointestinal ulceration. Initial therapy consisted of intravenous (IV) fluids supplemented with 20 mEq/L potassium chloride, ampicillin (20 mg/kg IV q 8 hours), metronidazole (10 mg/kg IV q 12 hours), and enrofloxacin (2.5 mg/kg IV q 12 hours). Ranitidine (1 mg/kg IV q 12 hours) and sucralfate (1 g PO q 8 hours) were administered for gastric mucosal protection. Morphine sulfate (0.5 mg/kg intramuscularly [IM] q 4 hours) was provided for pain relief.

Despite antibiotic therapy, within 20 hours of admission, the soft-tissue swelling over the right forelimb had progressed to include the ventral cervical region. The skin over the affected area was taut, erythematous, and warm to the touch. Fluctuant, subcutaneous pockets of fluid were palpable over the humerus and antebrachium. A fine-needle aspirate of one of the fluid pockets yielded a moderate quantity of foul-smelling, opaque, blood-tinged fluid. On cytological examination of the fluid, degenerate neutrophils, with many containing Gram-positive cocci, were observed. The fluid was submitted for aerobic and anaerobic culture. Mild serum hypoglycemia (3.1 mmol/L; reference range 3.3 to 5.6 mmol/L) was documented at that time and presumed to be secondary to sepsis. Dextrose (50% i) was subsequently added to the IV fluids to make a final concentration of 5% dextrose. Indirect arterial blood pressure, mucous membrane color, and CRT remained adequate. A fresh-frozen plasma transfusion (13 mL/kg IV once) and heparin therapy (75 units/kg subcutaneously [SC] q 8 hours) were begun in an attempt to prevent the development of disseminated intravascular coagulation. Surgical drainage of the limb was achieved by making two incisions, each 15 cm in length, over the antebrachium and humerus [see Figure]. The fascia was easily separated from the underlying muscle by digital dissection, and a moderate amount of murky fluid was drained. A wet-to-dry bandage was applied. Analgesia was provided by a local anesthetic block with lidocaine hydrochloride (50 mg SC) and a constant-rate infusion of morphine sulfate (1 mg/kg per hour IV).

Thirty-six hours after admission, the skin over the humerus and antebrachium became dark red, and the fascia visible through the incisions was purple and edematous. Aggressive surgical debridement with limb amputation was recommended to the owners but was declined. Within 46 hours of admission, soft-tissue swelling encompassed the entire right limb and neck, the proximal half of the left forelimb, and the ventral aspect of the thorax from the xiphoid process to the thoracic inlet. The skin in these regions was

![Figure](image_url)
mottled purple, and serosanguineous fluid could be aspirated from the subcutaneous tissues of the newly affected areas. Despite a constant-rate infusion of fentanyl (6 µg/kg per hour IV) and ketamine (1 mg/kg per hour IV), adequate pain control was not achieved. The owners elected to euthanize the dog and permitted a full postmortem examination.

The necropsy revealed dark purple, edematous fascial tissue along the right forelimb. A cloudy, brown fluid was present between the fascial planes of the right forelimb and thorax, extending caudally to the xiphoid process and cranially to the ventral aspect of the neck. The proximal half of the left forelimb was similarly affected to the level of the left elbow. The right cephalic vein was thrombosed.

Histologically, the panniculus, fascial planes, and endomysial connective tissue were expanded by protein-rich fluid, neutrophils, macrophages, erythrocytes, and cellular debris. Systemically, the lungs, liver, spleen, kidneys, and colon were congested, and the spleen contained multiple acute infarcts. Erosions of the gastric and colonic mucosa were present. Aerobic culture of the subcutaneous fluid yielded growth of Lancefield group G beta-hemolytic streptococcus, consistent with *Streptococcus canis*. The final pathological diagnosis was necrotizing fasciitis, panniculitis, and cellulitis secondary to a group G streptococcal infection.

Despite supportive care, broad-spectrum antibiotic therapy, and open wound drainage, the fasciitis in this dog rapidly progressed, emphasizing the importance of early and aggressive surgical debridement for successful management of necrotizing fasciitis. Antibiotic therapy in the absence of necrotic tissue debridement was ineffective, probably because of continued bacterial growth and toxin production as a result of poor antibiotic penetrance into the affected areas. It is unknown if early high amputation of the right forelimb would have changed this dog’s outcome.

The hematochezia seen in this dog is not a typical finding in cases of necrotizing fasciitis, although it may occur as a consequence of shock. In this case, the hematochezia was thought to be secondary to meloxicam-induced gastrointestinal erosions.

### Table

**Initial Laboratory Data From an Irish Wolfhound With Necrotizing Fasciitis**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Reference Range</th>
<th>Patient Data</th>
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<tr>
<td>White blood cell count (× 10^3 cells/μL)</td>
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<tr>
<td>Segmented neutrophils (× 10^3 cells/μL)</td>
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<td>Band neutrophils (× 10^3 cells/μL)</td>
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<td>Neutrophil morphology</td>
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<td>Packed cell volume (%)</td>
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<td>54.1</td>
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<td>Platelet count (× 10^3 cells/μL)</td>
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<td>Estimated as normal</td>
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<tr>
<td>Glucose (mmol/L)</td>
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<tr>
<td>Blood urea nitrogen (mmol/L)</td>
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<td>Creatinine (μmol/L)</td>
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<td>Activated clotting time (sec)</td>
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</table>
Conclusion

Necrotizing fasciitis should be suspected in animals that are presented with local erythema, edema, and disproportionately severe pain, especially in the presence of fever or shock. The surgical findings of easily parted fascial planes, copious exudate, and necrotic fascial and subcutaneous tissues are supportive of a diagnosis of necrotizing fasciitis. The diagnosis can be confirmed through histopathology of affected tissues and growth of group G streptococcus on culture of the affected site. Early diagnosis and aggressive surgical debridement are essential for the successful treatment of this disease. A broad-spectrum antibiotic regimen that includes clindamycin and appropriate supportive care are also important components of therapy.

References