Fatal Necrotizing Fasciitis and Myositis in a Captive Common Bottlenose Dolphin (*Tursiops Truncatus*) Associated with *Streptococcus Agalactiae*

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J VET Diagn Invest 2005 17: 617
DOI: 10.1177/104063870501700620

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J Vet Diagn Invest 17:617–622 (2005)

Fatal necrotizing fasciitis and myositis in a captive common bottlenose dolphin (*Tursiops truncatus*) associated with *Streptococcus agalactiae*

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Abstract. A common bottlenose dolphin (*Tursiops truncatus*) was presented for necropsy after acute onset of gastrointestinal signs and cutaneous lesions that rapidly progressed to death. Gross and microscopic findings were characterized by locally extensive severe necrohemorrhagic fasciitis and cellulitis, and severe necrotizing myositis in the head and dorsocranial thorax, with numerous disseminated gram-positive cocci. *Streptococcus agalactiae* was isolated from the lesions and from visceral organs (liver and lung), and it was identified by standard microbiology techniques. This communication is the first report of necrotizing fasciitis in a marine mammal associated with *S. agalactiae.*

Key words: Marine mammal; necrotizing fasciitis; *Streptococcus agalactiae*; *Tursiops truncatus.*

Necrotizing fasciitis (NF) is a term applied in human medicine to describe a rare but life-threatening, aggressive, toxin-mediated soft-tissue infection, primarily affecting the superficial fascial planes, but often associated with cellulitis, myositis, and possibly accompanied by early development of systemic toxicity.[9,22] Frequently, NF in adults is secondary to minor wounds or traumatic fractures and injuries or it may develop as a complication of surgical wounds. Associated conditions and risk factors are immunological disorders, old age, poorly controlled diabetes mellitus, and concomitant organ dysfunction.[16,22] Severe and diffuse necrosis of the overlying skin and toxic shock syndrome (TSS) occur, with up to 60–70% mortality rate when proper treatment is delayed.[13] The treatment of choice is deep surgical debridement and intravenous antibiotics to avoid septicemia.[9,16] Several microorganisms have been isolated from NF cases, primarily clostridia and invasive streptococci.[20] Although Lancefield group A streptococci (*Streptococcus pyogenes*) are frequently associated with NF, more recently, group G, and particularly, group B (*Streptococcus agalactiae*) infections have been documented as potential emerging disease.[7,10,11,20,23] The virulence factors for group A invasiveness are well established and probably shared by groups G and C, while pathogenesis of increased invasive capability of group B streptococci is not completely understood.[11,23] Rare cases of cellulitis and myofasciitis have been reported in association with group C streptococci.[5] Mixed bacterial infections, gram-negative enteric pathogens, and rarely, primary fungal infec-

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Table 1. Blood examination results of the bottlenose dolphin necropsied in the present study and reference ranges.*

Parameter		Reference range†	Captive‡	Free-ranging‡
RBC (10^6/mm^3)	4.19	2.9–5.4	3.0–3.7	3.1–4.0
WBC (10^3/mm^3)	**0.96**	4.1–10.8	5–9	5.6–12.4
Hb (g/dl)	**19.1**	12.7–18.1	13.5–15.5	12.7–15.5
MCV (fl)	**148.9**	101–143	115–135	111–127
HCT (%)	**62.4**	36.2–51.0	38–44	37–47
Platelets (10^3/mm^3)	**53**	100–200	80–150	92–127
Serum protein (g/dl)	**6**	6.2–8.3	6.0–7.8	6.4–8.8
Albumin (%)	64.9	48–75	71.6–88.3	45.3–61.6
Glucose (mg/dl)	99	87–150	90–170	62–139
Creatinine (mg/dl)	1.76	1.1–2.5	1.0–2.0	1.0–2.1
Bilirubin tot. (mg/dl)	0.11	0.0–0.2	0.1–0.2	0.1–0.4
Bilirubin dir. (mg/dl)	0.05	0.0–0.1	ND	ND
Cholesterol (mg/dl)	291	87–380	150–260	137–235
ALP (IU/liter)	**1,837**	123–1,050	300–1,300	51–610
AST (IU/liter)	510	184–516	190–300	133–318
ALT (IU/liter)	**95**	8–47	28–60	9–33
GGT (IU/liter)	31	7–41	30–50	17–31
CK (IU/liter)	**6,304**	14–486	100–250	ND
Na (mEq/liter)	157	141–168	153–158	151–158
K (mEq/liter)	**6.18**	3.5–5.7	3.2–4.2	3.2–4.4

* ND = not determined; RBC = red blood cells; WBC = white blood cells; Hb = hemoglobin; MCV = mean corpuscular volume; HCT = hematocrit; ALP = alkaline phosphatase; AST = aspartate aminotransferase; CK = creatine kinase; Na = sodium; K = potassium. Boldface entries indicate altered parameters when compared to the reference ranges from the laboratory.

† Reference ranges from the laboratory where the exams were performed for this study.

‡ Reference ranges for captive and free-ranging bottlenose dolphins according to the literature.[3]

tion have also been described as causative agents of NF.[9] Although *Staphylococcus aureus* traditionally causes TSS, the syndrome is usually not associated with tissue necrosis.[2] The few cases that have been reported in domestic animals have mainly involved group G streptococci.[6,15]

This report is believed to describe the first case of NF in a marine mammal (*Tursiops truncatus*), and the first case of NF in animals associated with *S. agalactiae.*

A captive 15-year-old female bottlenose dolphin was presented for necropsy. The clinical history indicated occasional traumatic skin lesions due to fighting with a male dolphin in the same pool. The dolphin had presented an acute onset of abdominal contractions; vomiting; acute, severe green-yellow diarrhea; and bilateral locally extensive moderate subcutaneous swelling, just caudal to the blowhole. Slow, weak swimming was noted. Blood examination was performed and results were compared also with literature reference ranges (see Table 1).[3] Slightly increased hematocrit (62.4%), hemoglobin (Hb) (19.1 g/dl), and mean corpuscular volume (MCV) (148.9 fl) and decreased white blood cell (WBC) count (0.96×10^3/mm^3) were noted. Thrombocytopenia (53×10^3/mm^3) was also apparently present. Creatine kinase value was very high (6,304 IU/liter), and alkaline phosphatase (ALP) (1837 IU/liter) and alanine aminotransferase (ALT) (95 IU/liter) were also moderately increased. Potassium (6.18 mEq/liter) was only slightly raised. Creatinine, bilirubin, other enzymes, and electrolytes were within reference ranges, with the exception of aspatate aminotransferase (AST), which was increased if referred to literature values. Erythrocytes appeared minimally higher than reported in the literature, but a slight variation might be due to calibration of the instruments to red blood cell size.[3] Minimal hypoproteinemia (6 g/dl) was also detected, with decreased albumin percentage when compared with the literature reference range for captive bottlenose dolphins. Septicemia and endotoxic shock, possibly due to clostridial infection, were suspected and intravenous antibiotics (ceftriaxone), cortisone (dexamethasone), and fluid therapy were administered. The dolphin died 2 hours after the onset of clinical signs and it was necropsied within 24 hours.

At necropsy, a bilateral, extensive (approximately 70×50 cm), moderate to severe, subcutaneous swelling was observed caudal to the blowhole (Fig. 1A). Throughout this swollen area, there was severe, diffuse subcutaneous edema, emphysema, hemorrhage, and necrosis that tracked deep along fascial planes and involved the epaxial skeletal musculature, with much less severe involvement of the blubber (Fig. 1B, 1C). Mild to moderate enlargement and hyperemia of the subcutaneous lymph nodes were noted. Bilateral mucopurulent conjunctivitis and severe retrobulbar subcutaneous hemorrhage were detected. Visceral organs showed diffuse moderate to severe hyperemia. In addition, there were multiple hyperemic gastric erosions and diffuse hyperemia of small intestinal mucosa. The mesenteric lymph nodes were moderately enlarged and diffusely hyperemic. Moderate bilateral diffuse pulmonary emphysema, parenchimal edema, hyperemia, and enlargement of the right cardiac chambers were observed.

Cytological preparations of the subcutaneous hemorrhagic effusion were stained by May Grumwald Giemsa and examined. The sample consisted of low numbers of inflammatory cells, mainly neutrophils and reactive macrophages, and rare fibroblasts, admixed with abundant proteinaceous fluid and lipid vacuoles with many cocci. The cocci were

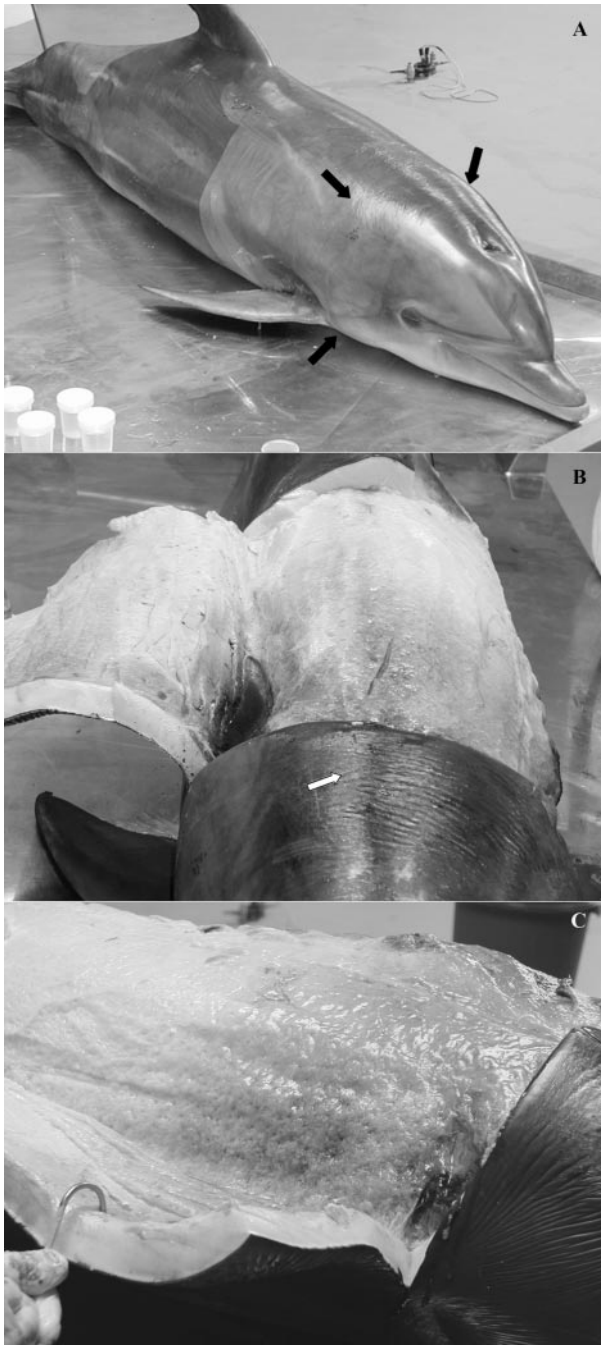

Figure 1. Common bottlenose dolphin, lateral and cranio-dorsal thorax: bilateral, locally extensive, moderate to severe, subcutaneous swelling **A** (arrows), with moderate stretching of the skin, **A** and **B** (arrows); severe subcutaneous edema, emphysema, and hemorrhage, **B** and **C**.

occasionally organized in short chains and often within degenerate and necrotic neutrophils (Fig. 2). Swabs for bacterial culture were obtained from the affected superficial and deep subcutis and skeletal muscle, and from lung, liver, and intestine.

Representative tissues were sampled for histology, fixed in 10% formalin, routinely processed, and stained with he-

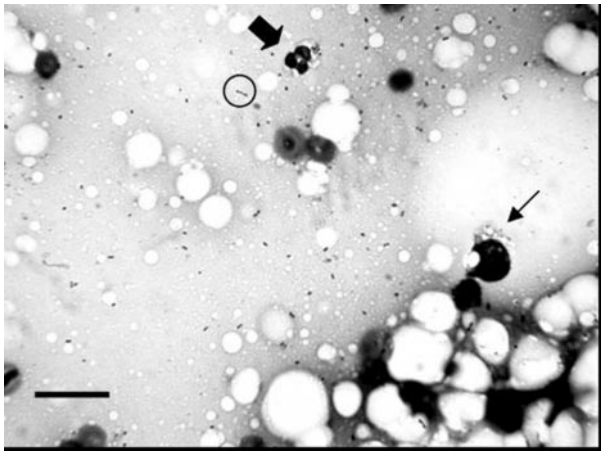

Figure 2. Common bottlenose dolphin, cytological smear from subcutaneous effusion. Many bacterial cocci, occasionally organized in short chains (circle), are admixed with abundant proteinaceous fluid, numerous lipid vacuoles, and rare inflammatory cells. A neutrophil containing a few intracytoplasmic bacteria (thick arrow) and a foamy macrophage (thin arrow) are present. May Grunwald Giemsa. Bar = 25 μm.

matoxylin, eosin, and tissue special stains. Histopathologically, some autolysis of visceral tissues was recognized. The skin and the skeletal muscle of the head and cranial thorax showed severe diffuse subacute hemorrhagic necrotizing cellulitis, fasciitis, and myositis, with septic thrombi, diffuse edema, congestion, and disseminated bacterial colonies (Fig. 3). Muscle fibers had large, pale areas lacking sarcoplasmic detail and clear demarcation (Fig. 3B). Subcutaneous and muscle-associated nerves frequently showed necrosis with abundant neutrophilic infiltration. Tissue Gram-staining revealed florid intralesional gram-positive cocci. Other special stains (periodic acid–Schiff [PAS] and Giemsa) didn't allow identification of any other etiological agent (yeasts, fungi, parasites). No epithelial inclusion bodies were detected. Superficial lymph nodes were hyperplastic, severely hemorrhagic, congested, and mildly edematous. Moderate superficial diffuse chronic gastritis of the forestomach was noted. The intestine exhibited a chronic, diffuse, moderate lymphoplasmacytic enteritis with hyperplasia and congestion of the gut-associated lymphoid tissue. The mesenteric lymph nodes had diffuse lymphoid hyperplasia with moderate follicular fibrin deposition, edema, hyperemia, and moderate sinus histiocytosis. Within the liver parenchyma, there was moderate to severe centrilobular congestion, with atrophy of hepatocytes presenting frequent intracellular hemosiderosis and mild cholestasis. Special stains failed to reveal PAS-positive or copper residues. A diffuse, moderate lytic change was also observed, partially consistent with a postmortem process. In the kidney, occasional tubular epithelial cells had intracytoplasmic granules (hemosiderin and bile pigments) and diffuse moderate congestion was noted. The lungs featured severe diffuse congestion, moderate diffuse emphysema, and scant intralveolar protein residues (edema). Interstitial, and more rarely, intralveolar hyaline fibrillar deposits, consistent with collagen at the Masson trichrome staining, and diffuse moderate degeneration of alveolar epithelium

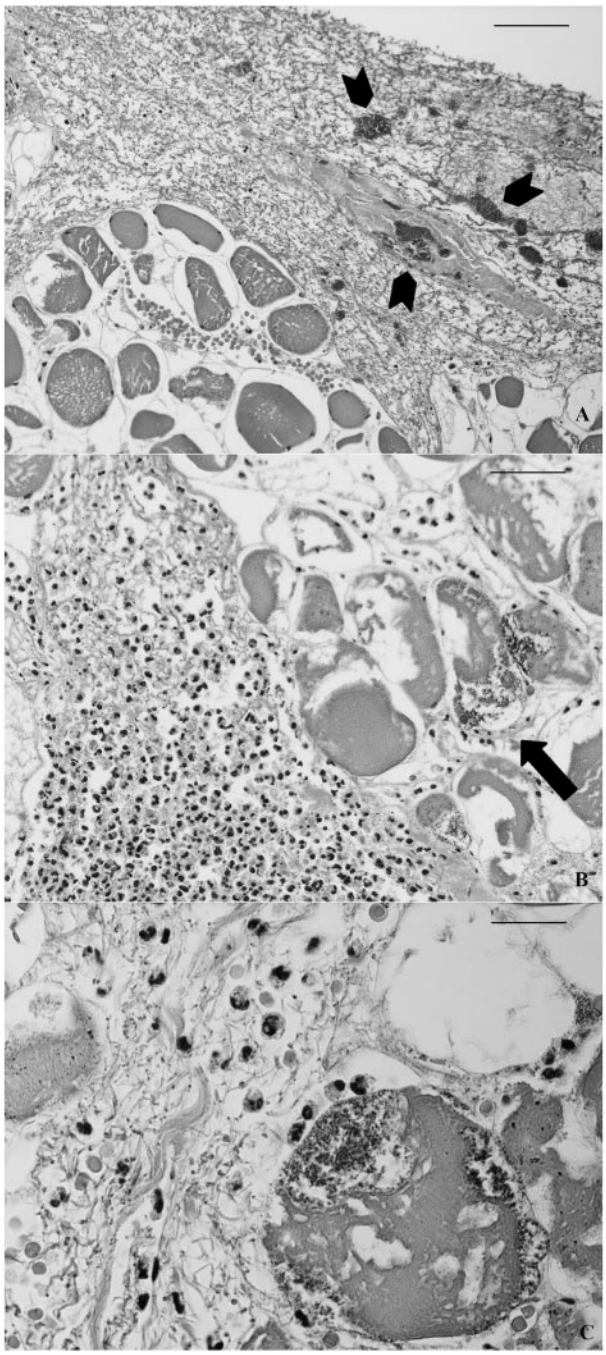

Figure 3. Common bottlenose dolphin, fascia and skeletal muscle of cranio-dorsal thorax. **A**, severe, diffuse necrotizing fasciitis, with many disseminated bacterial colonies (arrow heads); **B** and **C**, severe, diffuse necrotizing myositis. Note degeneration and necrosis of muscle fibers **B** (thin arrows), multifocally associated with bacterial colonies **B** and **C** (thick arrows). The inflammatory infiltrate is mainly composed by neutrophils and macrophages, **C**. Hematoxylin and eosin. Bars: **A** = 100 μm; **B** = 150 μm; **C** = 25 μm.

were also noted. Minor changes included diffuse bronchiolar epithelial erosion, with scattered disseminated dystrophic calcifications.

Bacteriology testing for aerobic and anaerobic microrganisms yielded pure growth of *Streptococcus agalactiae*, identified by standard biochemical assays from subcutis, skeletal muscle, liver, and lung. No anaerobe isolates were detected. The yeast *Rhodotorula* spp. was also isolated from the superficial subcutis. No virology tests were performed because no evidence of virus infection was noted and no reports regarding morbillivirus infections in captive marine mammals have been described. The sensitivity of the *S. agalactiae* strain to a panel of antibiotics was assessed and the results are shown in Table 2.

Table 2. Antibiogram results on the *Streptococcus agalactiae* strain isolated in this study.

Antibiotic	Results
Ampicillin	Sensitive
Amoxicillin + clavulanic acid	Sensitive
Cefoperazone	Sensitive
Oxacillin	Sensitive
Cefalotin	Sensitive
Cefuroxime	Sensitive
Tilosin	Sensitive
Spiramycin	Sensitive
Rifampicin	Sensitive
Tiamulin	Sensitive
Penicillin G	Intermediate
Cefalexin	Intermediate
Gentamicin	Resistant
Streptomycin	Resistant
Tetracycline	Resistant
Tilmicosin	Resistant
Sulfamethoxazole-trimethoprim	Resistant
Clindamycin	Resistant

On the basis of the pathological findings and the microbiology results, necrotizing fasciitis, cellulitis, and myositis due to *S. agalactiae* was diagnosed, and toxic shock syndrome was considered as the presumptive cause of death. *Rhodotorula* spp. is a common contaminant of air, soil, and water, and it may cause rare opportunistic mycosis in immunocompromised marine and terrestrial mammals.[4] In this case, it was not yielded from deep subcutis and from internal organs, and it was not detected by histology; therefore, it was not considered as relevant for the pathogenesis of the disease.

Streptococcus agalactiae is one of the most important causes of bovine mastitis, and its role in neonatal and adult sepsis and meningitis as well as invasive cutaneous infections is well documented in man.[8] Only a few cases of recognized human NF have been associated with *S. agalactiae*,[21] and NF has been fatal when associated with a toxic shock syndrome.[8,20] The association between NF and TSS remains unclear; primary traumatic lesions and concomitant chronic illnesses may have an important role in the development of the septic-toxic fatal consequences of the infection.[20,21] In veterinary medicine, uncommon severe dermal, fascial, and muscular necrosis, referred to as NF, have been recognized in dogs infected with *Streptococcus canis.*[6] Minor wounds are often noted 24–48 hours before the onset of clinical signs of NF and a few cases of fatal TSS development have been reported.[6,15] Apart from the well-known group A streptococci skin infection and TSS associated

events, the role of group G and B in the severe skin-necrotizing lesions is increasing in importance both in human and veterinary medicine.[6,10,11,15,23]

The clinical–pathological aspects described in this bottlenose dolphin illustrate a case of necrotizing fasciitis and myositis and a presumptive associated fulminant TSS due to *S. agalactiae*. Toxic shock syndrome is classically characterized by early hypotension with tissue edema, congestion, organ systems failure (lungs, liver, kidney), and, generally in the later phases, disseminated intravascular coagulopathy (DIC) and death.[19,20] Fever is normally present, particularly when pyrogenic toxin is produced in *S. pyogenes* infections.[20] Other clinical signs include confusion, vomiting, and diarrhea.[20] Tissue necrosis due to NF is generally superficial and associated with cutaneous rash and skin distension. Pain is present in the early stages, as it may disappear with disruption of nerve fibers.[12] Clinical pathology may demonstrate hemoconcentration, thrombocytopenia, increased prothrombin time, and activated partial thromboplastin time, possibly anemia, and impaired renal and liver function (increased serum creatinine values and liver enzymes, hypoalbuminemia).[20,21] Increased serum creatine kinase (CK) usually indicates a severe deep soft-tissue infection.[20] Both leukocytosis and leukopenia have been described in NF, the latter possibly due to depletion of circulating white cells sequestered in peripheral tissues.[1,12] Isolation of *Streptococcus* spp. from either a sterile or a nonsterile site of the body coupled with 2 or more clinical signs is required to confirm the diagnosis.[20]

In this dolphin, NF was clearly detected at gross examination and by histopathology. Streptococcal isolation from the site of injury and from other viscera indicated systemic involvement. Some difficulties were encountered discussing the hematobiochemistry results because reference ranges are not well established for this species. Increased CK serum levels were in accordance with a deep, necrotizing muscle lesion. Hyperkaliemia may be present in tursiops with rabdomyolysis.[3] Abundant liquid is generally sequestered in these lesions, eventually leading to hemodynamic derangements, with potential for cardiovascular and respiratory failure.[14] In addition, dilation of the right heart chambers (generally due to decreased myocardial contractility), generalized severe edema and visceral congestion, and evidence of respiratory failure (alveolar damage, edema, and emphysema) suggested shock was the most likely cause of death. Furthermore, agonal vomiting and sudden onset of diarrhea with no evidence of underlying intestinal pathology may indicate systemic toxic involvement. Thrombi were only detected at the site of injury, where they may be more numerous and easier to detect after death. In other anatomical locations, thrombi may undergo postmortem lysis.[21] Centrilobular liver degeneration indicated hypoxic damage, as seen in shock. Increased serum ALT further confirmed antemortem hepatocellular damage, as generally described in TSS.[20] Besides liver diseases, increased ALT may be found in muscle trauma, neoplasia, parasitic infections, and after administration of antibiotics and corticosteroids.[3] Raised ALP serum level was also detected. ALP isoenzymes are located in a variety of tissues, including liver, muscle, bone, and kidney. Generally similar trends to what are described in terrestrial animals for ALP activity exist in marine mammals. In bottlenose dolphins, ALP activity elevation has been described as a liver damage-specific indicator, even though it may increase also in muscle, bone, and kidney pathologies.[3] A significant difference between ALP levels in captive dolphins with respect to those in free-ranging animals can be noted (Table 1), and despite no specific explanation for this data have been published, metabolic, nutritional, and hepatic factors should be considered as responsible for this variation.[3] AST value, which generally increases in both hepatic and muscular damage, was higher than literature maximum levels, but it was still within the wider laboratory reference range. It showed, however, a sudden increase when compared with previous blood exams in this dolphin (data not shown), suggesting a possible correlation with both the muscular and the hepatic lesions. Hypoalbuminemia is described in marine mammals with advanced hepatic disease, hemorrhage, gastrointestinal diseases, protein-loosing nephropathies, and extensive skin lesions with epidermal compromise.[3] According to the laboratory reference range, this dolphin was not hypoalbuminemic, even though albumin level was below the literature ranges. The loss of albumin was considered not significant in this dolphin, and this was not surprising because marine mammals, like terrestrial ones, have tremendous hepatic reserve capacity for albumin production. No clear acute tubular necrosis was detected in the kidney, where diffuse autolytic change was noted. In addition, no alteration in serum creatinine level was evidenced. Increased hematocrit, Hb, MCV, and leukopenia were observed before death, as reported in human NF-TSS cases.[1,12] In marine mammals, WBC count may increase during several bacterial infections and stress, while decreased values may be noted in chronic fatal infective diseases.[18] No anemia was registered. Thrombocytopenia, generally present in human NF-TSS, possibly suggests local/systemic thrombosis and DIC, despite the fact that a wider range of platelet levels ($58–178 \times 10^3/mm^3$) is considered normal by other authors.[17]

The history of cutaneous lesions and the stress of captivity cannot be ruled out as predisposing factors for NF-TSS in this dolphin. The ocular lesions might have represented the portal of entry for the pathogen, despite the fact that no bacterial cultures were performed from this site. No other pre-existing skin lesions were detected at tissue examinations in this dolphin. Frequently in humans and domestic animals with NF, bacterial infection spreads from local penetrating skin infection and/or trauma. Generally face/neck NF has been described as a consequence of dental, oral, or eye lesions.[12,14]

Antibiotic choice is a key factor in the treatment of NF.[9,16] Streptococci are normally highly sensitive to penicillin. This antibiotic is the treatment of choice in NF, though it may fail in the presence of a large microbial population in the necrotizing processes.[7] Furthermore, angiothrombotic liquefactive necrosis of fascia may compromise antibiotic delivery within the site of infection.[23] In this case, the bacterial strain showed only intermediate sensitivity to penicillin. Rare reports suggest the use of clindamycin in combination with penicillin in cases of group B *Streptococcus* spp. (GBS) NF because resistance to penicillin and clindamycin is generally

not an issue with GBS infections.[23] In this case, other penicillins and third-generation cyclosporins would have been more effective (see Table 2), even though, in severe NF cases, a deep surgical debridement is needed in association with the antibiotics treatment.[9,16]

This case emphasizes the pathogenic potential of *Streptococcus* spp., particularly group B strains, in severe skin infections and the potential for certain cetacean infections to be zoonotic or anthropozoonotic.

Acknowledgement. We are grateful to Dr. Alessandro Bortolotto (Zoönomia, Italy) for providing clinical information.

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