Staphylococcal Pyelonephritis and Cystitis in a California Sea Lion (Zalophus Californianus)

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Abstract
An adult, female California sea lion (Zalophus californianus) was stranded in Laguna Beach, California (33°32'43.96"N x 117°47'51.10"W) on the afternoon of January 13, 2007. Clinical signs included marked lethargy, head-bobbing and 'wet-dog shaking'. A presumptive diagnosis of Domoic Acid intoxication was made and supportive therapy including anti-seizure treatment was instituted. About thirty hours later, seizure activity had abated but the animal continued to deteriorate to a semi-comatose state and euthanasia was elected. Postmortem examination revealed severe, bilateral pyelonephritis and cystitis from which a pure culture of Staphylococcus aureus was isolated. With the exception of Leptospira sp interstitial nephritis, bacterial urinary tract infections are very rare in marine mammals. To the author's knowledge this is the first report of Staphylococcus aureus pyleonephritis and cystitis in the California sea lion. [JMATE. 2008;1(1):12-14]

Keywords: California sea lion, bacterial nephritis, Staphylococcus aureus

Introduction
An adult, female California sea lion (Zalophus californianus) stranded on the beach at Shaw's Cove, Laguna Beach, California (33°32'43.96"N x 117°47'51.10"W) on January 13, 2007. A rescue team from the Pacific Marine Mammal Center (PMMC) was dispatched to the scene and noted the sea lion to be very lethargic, reluctant to move and exhibiting periodic head-bobbing and generalized 'wet-dog shaking'. Domoic acid (algae toxin) intoxication was suspected and the animal was impounded and transported to PMMC.

Physical examination revealed moderate, generalized body wasting and intermittent low-grade tonic-clonic seizure activity with head-bobbing. Heart rate was 86 bpm, respirations ~30/minute and temperature 38°C (100.4°F). Rehydration was instituted by subcutaneous administration of lactated Ringers. Diazepam (5 mg/ml Benzodiazepine) was given intramuscularly (dosage = 5 mg) to control seizures. Attempts at blood collection via the caudal gluteal and jugular veins were unsuccessful. Twenty four hours later, the sea lion's condition had stabilized with no obvious seizure activity. However, the following morning she had deteriorated to a semi-comatose state with very sluggish to non-existent reflexes and moderate bradycardia and dyspnea. Because of a poor prognosis, euthanasia and postmortem examination was elected. Following euthanasia, blood samples were collected for pick-up and analysis (CBC and serum chemistry) by a local, commercial veterinary laboratory. Unfortunately, the samples were accidentally destroyed in-route to the laboratory.

Postmortem examination revealed a marked generalized muscular wasting and depletion of subcutaneous and visceral adipose stores. Gross and histologic examination of the brain, as well as analysis of blood and cerebrospinal fluid for Domoic acid by a specific ELISA (Dr. Astrid Schnetzer, Caron Laboratory, University of California, Los Angeles) failed to substantiate a diagnosis of Domoic Acid poisoning.

Visceral gross and microscopic pathology was restricted to the urinary system. Kidneys were discolored yellow and exhibited a marked, bulging accentuation of the reniculi (Figure 1a). Transverse sections of kidneys revealed multifocal clusters of multiple, 'ragged' reniculi with scattered, relatively normal renal tissue (Figure 1b). Wright's Giemsa and gram-stained impression smears contained necrotic material with intermixed masses of neutrophils and mononuclear phagocytic cells, containing large numbers of gram-positive cocci, singly, as pairs or in clusters (Figure 2).

Histologically, there was moderate to severe, multifocal to diffuse tubulogglomerular necrosis and loss, with moderate to severe, tubulointerstitial neutrophil infiltration. Mononuclear cells and macrophage and some lymphoid infiltration. Multifocal, varying sized colonies of gram-positive cocci with surrounding Splendore-Hoeppli material (radiating eosinophilic deposits) were scattered irregularly, throughout the renal parenchyma (Figure 3a). Large masses of degenerating neutrophils and other necrotic debris filled the pelvis of many reniculi (Figure 3b). The bladder was markedly distended with dark-colored urine. A cystocentesis revealed a
urine specific gravity 1.2, pH 7.5, moderate to marked levels of blood, ketones, glucose, protein, urobilinogen and bilirubin. The urine sediment contained large numbers of neutrophils, many macrophages containing gram-positive cocci and some plasma cells and lymphocytes. The bladder wall contained moderate to severe, multifocal areas of denuding necrotic urothelium with moderate to marked, submucosal infiltration by moderate to marked numbers of neutrophils and macrophages with occasional scattered colonies of gram-positive cocci.

Swabs of asceptically obtained, clotted blood and the deep parenchyma of the right kidney were taken and submitted to a local veterinary laboratory (IDEXX) for bacterial culturing. No bacteria were cultured from the clotted blood but a pure culture of oxacillin-sensitive *Staphylococcus sp.* was obtained from the kidney. Culture samples were submitted to the University of California at Davis, College of Veterinary Medicine, Clinical Laboratory for speciation. The organism was found to be a coagulase-positive *Staphylococcus*, phenotypically identified as *Staphylococcus aureus*.

*Staphylococcus aureus* is a gram-positive, coagulase-positive bacterium commonly found as a benign, commensal on the skin and mucous membranes of the respiratory, gastrointestinal and urogenital tracts of wild and domestic mammals and birds. However, when these membrane barriers are compromised by physical trauma and/or immunopathies, *Staphylococcus* may gain entry into the tissues, resulting in bacteremia and disseminated inflammatory disease. A classic example in humans and other animal species is lower urinary tract trauma resulting in ascending disease through the bladder and into the kidneys (5). In this case, the fact that *Staphylococcal* bacteraemia was not detected from an aseptically collected blood sample gives credence to an ascending disease etiology, i.e. the bacteria probably gained entrance through the vulvar area, up the urethra, into the bladder and finally the kidney.

The normal kidneys of a California sea lion are multilobed or reniculate, being composed of hundreds of lobes or ‘reniculi’ (LL, Dim., “Little Kidneys”), which are further divided into the standard morphologic subunits of metanephric kidneys, i.e. a distinct layer of cortical tissue completely surrounding a medullary pyramid within a single calyx (1,6,7). Unlike the *Phoca* in which each reniculus is distinctly separated by an external capsule, in *Zalophus*, only a very thin fibrous capsule is noted surrounding the reniculus which allows inflammatory disease to readily spread between reniculi as depicted in this case (Figure 1b) (1).

Howard’s 1983 study on the pathobiology of marine mammal diseases noted kidney infections to be “uncommon” in marine mammals, but referenced a case of *Leptospiral* nephritis in a northern fur seal (3). A review of the literature since this time, clearly shows *Leptospira* are now a common etiology for chronic interstitial nephritis in the California sea lion (2,11). However, only a few cases of *Staphylococcus*-associated renal disease have been reported in marine mammals. In a case published in 1973 involving attempts to rehabilitate a newborn Harbour seal stranded in Alaska, pustular dermatitis was noted at 12 days of age, followed by death at 21 days with the post-mortem isolation of a coagulase-positive, *Staphylococcus aureus* from the lung, liver and kidneys (10). In 1974, Ketterer and Rosenfeld reported a *Staphylococcus aureus* subcutaneous abscess in a dolphin (*Trusios truncatus*), resulting in fatal septicemia and septic embolic nephritis (4). In 2001 and 2002, *Staphylococcus aureus* septicemia and severe, suppurative pyleonephritis was found following postmortem examinations in Harbour porpoises (*Phocoena phocoena*) stranded in the German North and Baltic Seas (8,9). To the authors’ knowledge, this case appears to be the first report of urinary tract infection attributed to *Staphylococcus aureus* in the California sea lion.

In conclusion, there are several important points to made and lessons to be learned from this case: 1) Generalized body

**Figure 1b.** Transverse sections of formalin fixed right kidney. Multiple foci of contiguous, ‘ragged’, inflamed reniculi are apparent, however several adjacent reniculi are relatively unaffected (medullary pyramid lower right, top section). The large vacuolated portion in the bottom section represents pelvic dilation secondary to corticomedullary loss from necrotizing inflammation.

**Figure 2.** Cut-surface touch impression of right kidney. Note mononuclear, phagocytic cells containing proliferating bacterial cocci arranged in couples or varying sized clusters. Wrights-Giemsa stain, 40x
wasting, lethargy and neurologic signs are not etiologic specific findings, but rather they are found in a myriad of subacute to chronic organ-system based diseases, 2) It is necessary to conduct gross postmortem examinations on all mortalities occurring in a rehabilitation center. In this case, had such an examination not been done, an erroneous clinical diagnosis of Domino Acid intoxication might have been made, 3) Cytological examination of tissues is an easily performed, "low tech" procedure that has significant diagnostic utility. In this case, staining of impression smears of grossly abnormal kidneys with a Wright-Giemsa and a Gram's stain, easily revealed a severe, gram-positive coccoid bacterial associated, inflammatory disease process.

References