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Leptospirosis in Rehabilitated Pacific Harbor Seals from California

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ABSTRACT: Renal disease was observed in two rehabilitated Pacific harbor seals (*Phoca vitulina richardsii*) from a facility in California (USA). The seals had leukocytosis and high serum phosphorus, blood urea nitrogen and creatinine concentrations. A retrospective study of leptospiral antibody serum titers indicated both seals had elevated titers to *Leptospira interrogans* serovar *grippotyphosa*. A third seal, which died about the time when the index cases occurred, also had elevated titers to *L. interrogans* serovar *grippotyphosa*. Post mortem histopathologic examination of all three seals showed tubular necrosis consistent with interstitial nephritis; spirochetes were seen within the kidney parenchyma of the third seal. Sea lions (*Zalophus californianus*) or elephant seals (*Mirounga angustirostris*) housed near the harbor seals were possible sources of exposure, but local wildlife also could have been responsible.

Key words: Case report, harbor seal, *Leptospira interrogans* serovar *grippotyphosa*, leptospirosis, *Phoca vitulina richardsii*, renal disease.

Leptospirosis has been documented in many wild and domestic terrestrial species of mammals. However, leptospirosis among marine mammals, specifically *Leptospira interrogans* serovar *pomona*, has been reported only in two otariids, California sea lions (*Zalophus californianus*) and northern fur seals (*Callorhinus ursinus*) (Smith et al., 1974; Dierauf et al., 1985; Gulland et al., 1996). Despite a sympatric distribution of sea lions and harbor seals (*Phoca vitulina richardsii*), leptospirosis has not been identified in the harbor seal population along the coast of California (USA).

Each year hundreds of sick and injured pinnipeds are treated at rehabilitation centers along the coast of California. Within these centers, debilitated sea lions, elephant seals (*Mirounga angustirostris*), harbor seals, and other species are managed in close proximity, potentially allowing interspecies disease transmission. This paper describes clinical leptospirosis and associated lesions caused by *Leptospira interrogans* serovar *grippotyphosa* in Pacific harbor seals housed in a rehabilitation facility.

In June of 1996, two debilitated harbor seals, Pv1035 and Pv1047, housed at a marine mammal rehabilitation center in central California (USA; 37°49′N, 122°32′W) for 54 days and 71 days respectively, became depressed and anorexic within 24 hr of each other. The approximately 3-mo-old seals were housed together in a 5 × 5 m pen enclosed by chain-link fence with cement flooring. The pen contained an above-ground pool (2.5 m in diameter by 1.0 m deep) connected to similar pools in adjacent pens through a closed-circuit recycling filter system. Water disinfection with sodium hypochlorite occurred during filtration.

Clinical signs in the affected harbor seals included anorexia, depression and dehydration, with Pv1035 also having oral ulcers and halitosis. Blood collected from the extradural intravertebral sinus (Bossart and Dierauf, 1990) showed similar hematological and biochemical changes in the two affected seals. These included leukocytosis and elevations in blood urea nitrogen, serum creatinine, total protein, globulins, calcium and a marked elevation in phosphorous (Table 1). Urine collected from Pv1035 by cystocentesis had a specific gravity of 1.018 and contained many granular casts, red blood cells and rare white blood cells. Hematological and serum biochemical changes and urine abnormalities were similar to those observed

TABLE 1. Hematological values of harbor seals clinically affected by leptospirosis.

Diagnostic test	Pv1035	Pv1047	Reference values[a]
White blood cell × $10^3/mm^3$	27.7	26.3	5.5–14.6
Sodium mEq/l	203.0	172.0	145–56
Potassium mEq/l	5.5	4.3	4.0–6.0
Chloride mEq/l	156.0	121.7	97–111
Blood urea nitrogen mg/dl	186.0	>130.0	25–97
Creatinine mg/dl	4.0	0.9	0.4–1.4
Total protein mg/dl	10.4	9.5	6.1–9.7
Globulin mg/dl	6.5	6.4	2.1–5.6
Calcium mg/dl	12.2	11.3	8.8–10.9
Phosphorus mg/dl	21.6	10.7	4.3–7.9

[a] From Bossart and Dierauf (1990).

in sea lions with renal disease caused by leptospirosis. A presumptive diagnosis of dehydration and possible leptospirosis was made (Dierauf et al., 1985). Therapy was initiated with enteral tetracycline (Pfizer, New York, New York, USA), oral phosphorous binders (Amphogel®, Wyeth-Ayerst, Philadelphia, Pennsylvania USA) and electrolyte solution (Benfital®, Boehringer Ingelheim Animal Health, St. Joseph, Missouri, USA) given via stomach intubation. Harbor seal Pv 1047 recovered from illness and regained normal appetite, weight, hematological and serum biochemical parameters. Harbor seal Pv1035 deteriorated 10 days from the onset of clinical signs and was euthanized by intravenous administration of 1900 mg pentobarbitone (Buthanasia-6®, Anthony Products, Arcadia, California, USA).

The kidneys of Pv1035 were pale and swollen with moderate loss of renule differentiation and subcapsular hemorrhages. Histological examination of the kidneys showed tubular necrosis with associated mononuclear infiltrates typical of severe interstitial nephritis. Warthin-Starry silver stains of kidney sections were inconclusive, however, organisms could have been obscured by the extensive degree of necrosis within the stained tissues (Luna, 1968).

The discovery of interstitial nephritis typical of leptospirosis initiated a retrospective serologic study to establish whether or not the seals were infected with one or more serovars of *Leptospira interrogans*, and when. Frozen serum samples collected from Pv1035 and 1047 on admission to the rehabilitation center in April, and 6 wk later during clinical illness, were analyzed for antibodies to *L. interrogans* serovars *pomona, grippotyphosa, bratislava, canicola, icterohemorrhagiae* and *hardio* by the microagglutination test (MAT) (Gulland et al., 1996). Both animals were seronegative to all serovars on admission, but seroconverted during clinical illness with titers to *Leptospira interrogans* serovar *grippotyphosa* >1:1,600 in Pv1047 and >1:3,200 in Pv1035. Serum titers >1:100 were considered positive (Bolin, 1996).

To determine possible exposure sources to *L. interrogans*, freezer-banked serum from a subset of resident yearling sea lions (n = 10), 4-mo-old elephant seals (n = 10), and 3-mo-old harbor seal pups (n = 8) were tested for serum antibody titers against *L. interrogans* serovars *pomona, grippotyphosa, bratislava, canicola, icterohemorrhagiae* and *hardjo* by MAT. Other than the two index cases, all animals tested were underweight but had normal hematological and serum biochemical parameters. Three of the tested sea lions had titers >1:1,600 against both *L. interrogans* serovars *pomona* and *hardjo*. Of these three, one animal also had titers of 1:800 against *L. interrogans* serovars *grippotyphosa* and *icterohemorrhagiae*. Three of the tested elephant seals had titers >1:3,200 to *L. interrogans* serovar *grippotyphosa* with one of these having a titer of 1:1,600 against *L. interrogans* serovar *bratislava*. Of the harbor seal pups sampled, one (Pv1090) had a titer of 1:3,200 to *L. grippotyphosa*. This animal unexpectedly died within 2 wk of the two index cases. No clinical pathology parameters were obtained prior to death in this third seal, but histological examination showed nephritis

with positive Warthin-Starry silver staining of the spirochetes in the kidneys.

Positive silver staining of spirochetes in the kidneys of harbor seal Pv1090 along with high serum antibody titers to *L. interrogans* serovar *grippotyphosa* in all three affected harbor seals strongly suggests that *L. interrogans* serovar *grippotyphosa* infection caused the renal disease. Viral infections could predispose animals to secondary bacterial infections such as leptospirosis and herpesvirus infections have been found in approximately 50% of the stranded harbor seal pups on the coast of California that die during rehabilitation (Gulland et al., 1997). However, histologic examination of the adrenals and liver did not show viral inclusions in the affected harbor seals.

The seroconversion of the harbor seals to *L. interrogans* serovar *grippotyphosa* during rehabilitation suggests exposure occurred at the center. On-site California sea lions were one possible source of infection but elevated antibody titers to *L. interrogans* serovar *pomona* and *hardjo* were not consistent with the serovar titers found in the harbor seals. Even though some of the sea lions had lower elevated titers to *L. interrogans* serovar *icterohemorrhagiae* and *grippotyphosa,* these are tentatively considered a consequence of cross reactivity in the microagglutination test (Gulland et al., 1996). Although sea lions cannot be discounted as the source of infection, this suggests they were not likely the source of leptospirosis for the affected harbor seals. Several of the resident elephant seals had elevated titers to *L. interrogans* serovar *grippotyphosa.* Although no signs of renal disease were noted in the elephant seals, the possibility that these animals could have been the source of leptospirosis should be investigated further.

Leptospira, interrogans serovar *grippotyphosa* has been documented in raccoons, foxes, skunks, opossums, and mice (Maghami et al., 1977; Reilly, 1970). Raccoons, skunks, mice, voles, and foxes are abundant in the area surrounding the rehabilitation center and should be investigated to determine whether any of these could have been a potential sources of this infection.

Leptospira interrogans serovar *grippotyphosa* has not been investigated in wild harbor seals populations on the coast of California. High densities of diseased animals of various species in rehabilitation centers and the difficulties in controlling small terrestrial mammal populations can facilitate transmission of pathogens between individuals and species. In conjunction with coordinated sampling of wild populations, routine screening for exposure to and infection with pathogens upon entry and prior to exit from rehabilitation centers should be performed. This allows baseline health data on wild populations to be collected and will reduce the risk of introduction of an infectious disease into a previously unexposed population.

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