Pacific Marine Mammal Center

Case Reports in Marine Mammal Pathology
Volume 1, Number 1, November 2012

Fatal outcome of conspecific trauma in a young California sea lion (*Zalophus californianus*)

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Conspecific trauma often has serious consequences in marine mammals. This report describes what appears to be a fatal outcome of such an event. A juvenile female California sea lion (*Zalophus californianus*, Z-12-03-27-017, Ella) was found March 27, 2012, at Fisherman's Cove, Laguna Beach, CA. She was hiding in a small cavern among beach rock formations. A rescue team was dispatched to acquire the seal pup and transport it to the Pacific Marine Mammal Center (PMMC).

Physical examination at entry to PMMC revealed marked cachexia, characterized by moderate muscle wasting, dehydration, and no evidence of subcutaneous fat. Ella did not respond to human movements near her head, but she did respond to human touch. Rehydration and nutritional support were initiated. However, she was found dead early the following morning of March 28.

Postmortem Examination

Gross necropsy revealed moderate, systemic depletion of muscle mass. No adipose tissue was present in usual subcutaneous or visceral fat deposition sites. A conspecific cervical bite wound was suggested by a dark, 1-cm diameter cutaneous lesion, with connecting subcutaneous involvement indicating pressure necrosis.

Bilateral, ventral (gravitational) pneumonic congestion was observed in the thoracic cavity, with multifocal areas of evident pulmonary consolidation. The abdominal cavity contained about 400 ml of reddish, opaque, thick, fetid peritoneal effusion. (Figure 1) The greater omentum was diffusely darkened with moderate-severe vascular hyperemia-congestion. (Figure 2) The liver was moderately enlarged and mustard-colored. (Figure 2) A 20-25 cm portion of the mid-jejunum under the greater omentum was darkened with vascular congestion and grey discolor-ation on the anti-mesenteric side. (Figure 3) This lesion was transmural, necrotic, and sloughing, resulting in numerous red, ragged, full thickness mucosal deficits. The anterior jejunum and colon mucosa were diffusely thickened.

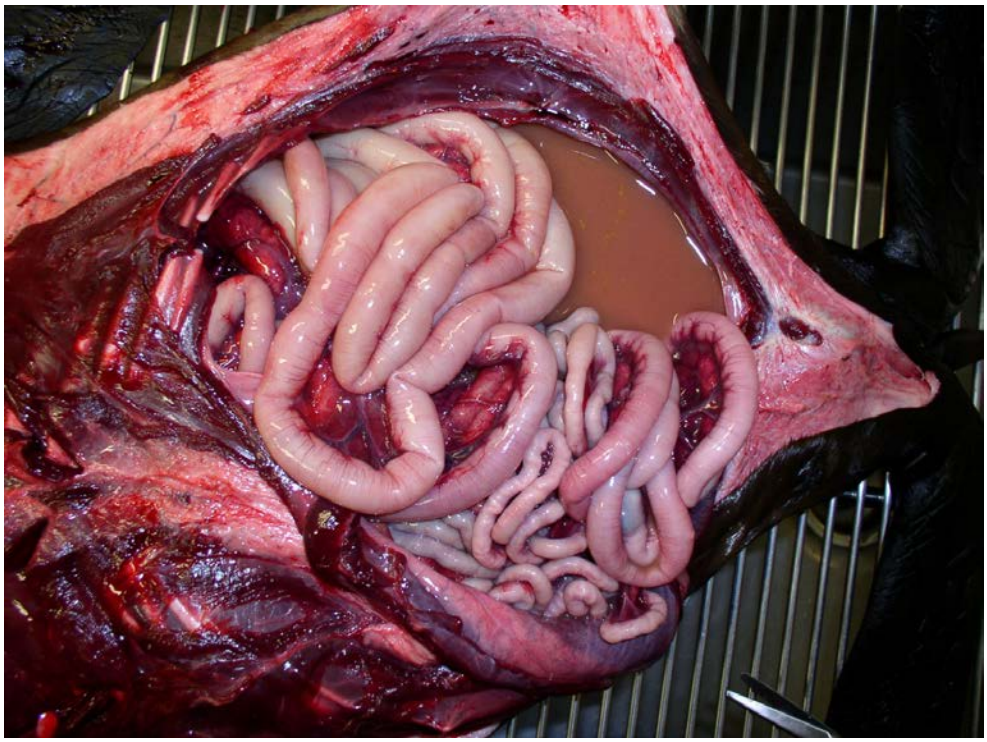

Figure 1: Peritoneal effusion

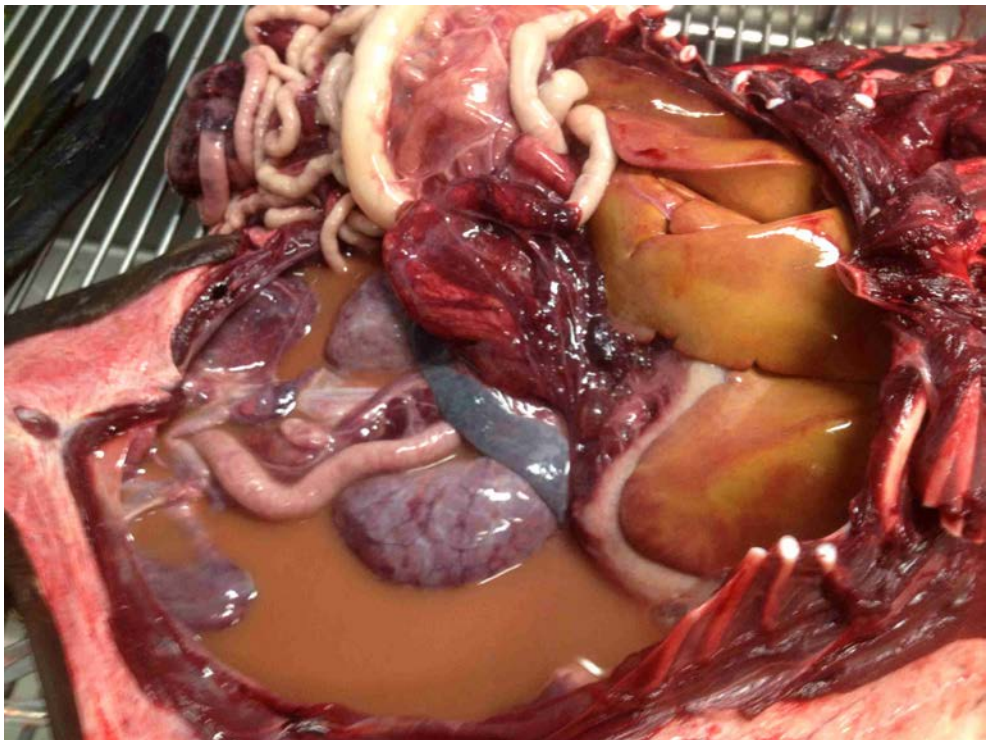

Figure 2:

Dark, congested mesenteric vasculature, fatty-appearing liver
Gross necropsy diagnoses included: a) adipose depletion, subcutaneous and visceral, severe, diffuse; b) skeletal muscle depletion, severe, diffuse; c) abscessation, subcutaneous, right mid-cervical, moderate-severe, with probable pressure necrosis suggesting conspecific bite wound; d) pneumonopathy, multifocal, moderate-severe, consolidating; e) peritonitis, diffuse, severe, chronic-active, probably secondary to multifocal small intestine mural deficits; f) seropathy-mesenteropathy, mild-moderate, jejunal to colonic, with congestion and hemorrhage, multifocal, moderate-severe; g) hepatopathy, mustard discoloration, diffuse, severe, suspicious for toxic fatty change; h) jejunopathy, necrotizing, hemorrhagic, multifocal, moderate-severe, with multiple full-thickness deficits; i) colonopathy, Acanthocephaliasis, multifocal-to-disseminated, and *Corynosomiasis*.

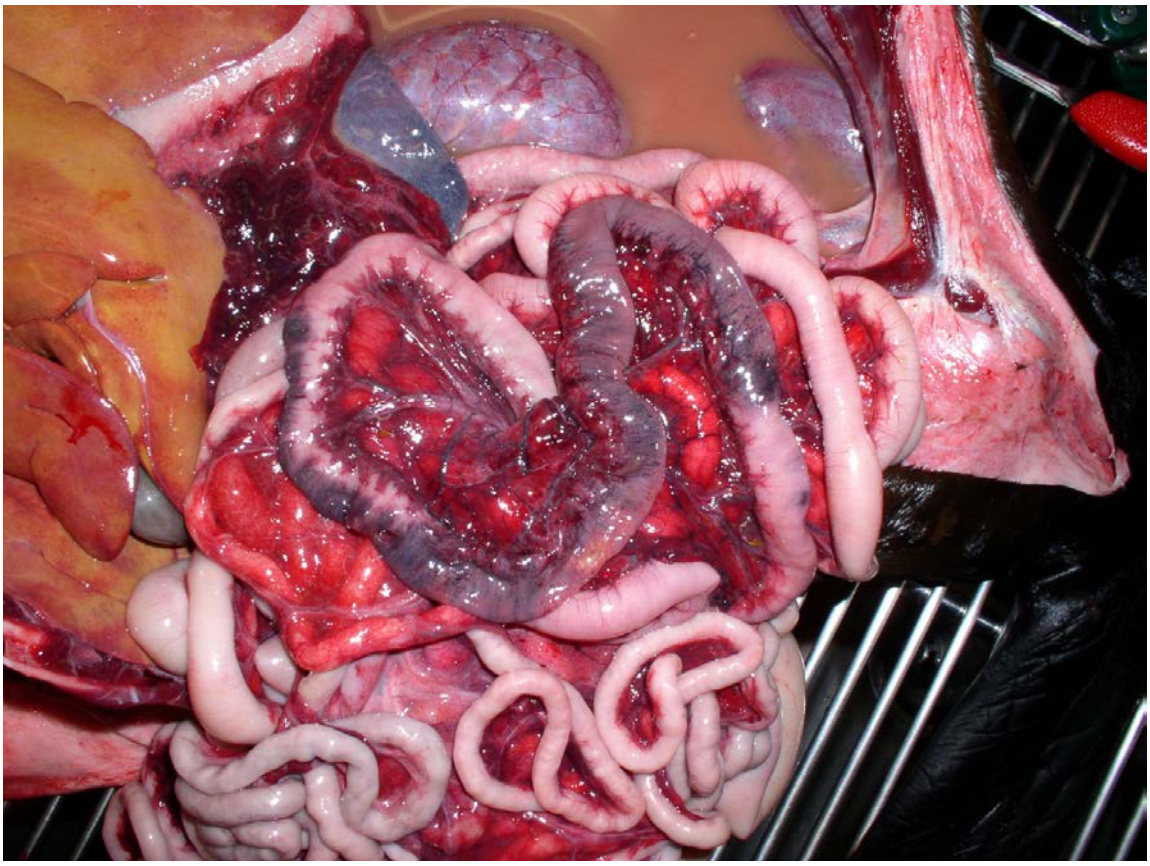

Figure 3. Anti-mesenteric border of the jejunum

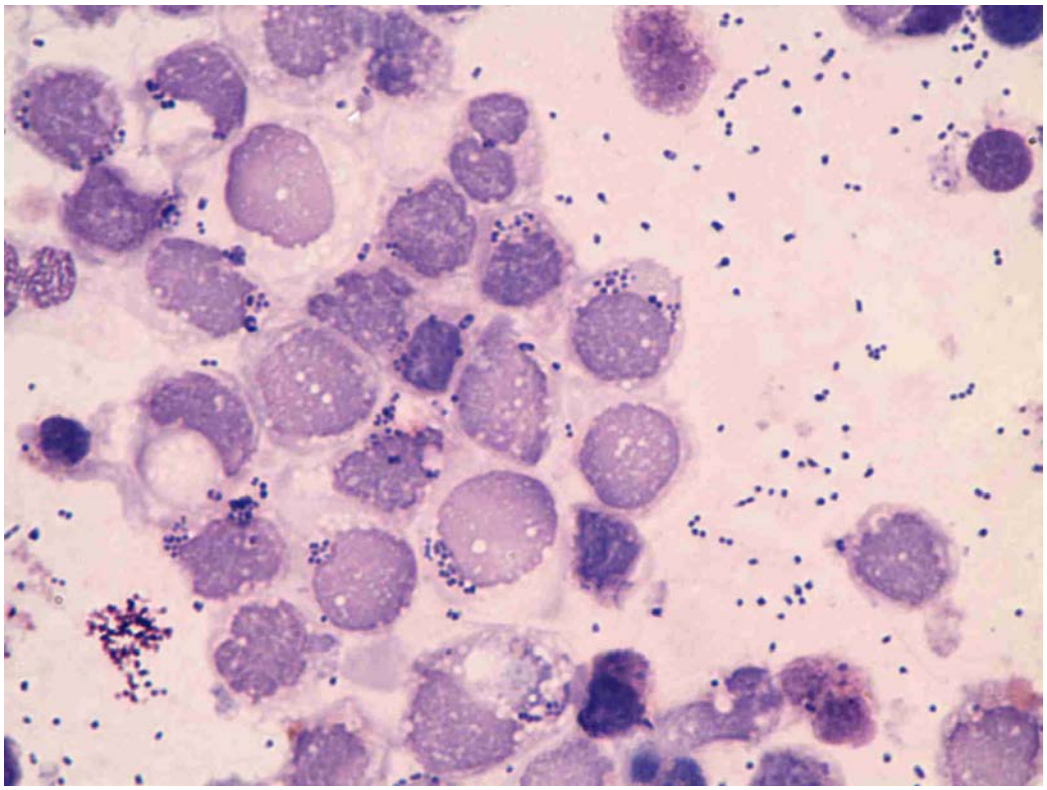

Figure 4. Cytology of peritoneal effusion

The primary observation was a large number of mononuclear cells with large, round nuclei, undergoing chromatin vacuolar degeneration. Also visible were narrow rims of frequently vacuolated and faintly basophilic cytoplasm. Cocci and coccobacilli frequently were found in mononuclear cytoplasm. Moderate neutrophilia also was observed, with degenerative chromatin and occasional small clusters of diplococci or coccobacilli.

Impression smear of a mesenteric lymph node revealed broad sheets of normal small lymphocytes, with scattered medium-to-large lymphoid cells having thin rims of densely basophilic cytoplasm.

Cytologic diagnoses included a) peritonitis, subacute-to-chronic-active, moderate-to-severe, mixed, coccoid and coccobacilli bacterial population; b) lymphadenopathy, mesenteric, moderate (probable reactive hyperplasia, secondary to peritonitis).

Vitreous humor analysis

Vitreous humor from the right eye was colorless and hazy, containing <2.5gm/dl protein. Cellular evaluation revealed <100,000 total cells/ul , and 948 nucleated cells/ul.

Microbiology

Microbiological evaluation of peritoneal effusion revealed: 2+ *Escherichia coli* (2 strains); 3+ *Enterococcus sp*; 1+ S*hewanella algae.*

Histopathology

In the lungs there were areas of gross pneumonic consolidation revealed multilobular foci of marked (80-90%) neutrophilia, along with 10-15% monocytes and macrophages actively phagocytizing cells and debris. A few of these foci contained suspected *Parafilaroides sp.* larvae that also were observed in occasional bordering and non-border-ordering airways that were otherwise near-normal. Diagnosis: 1) Pneumonia, multilobular, multifocal, moderate-to-marked, chronic-active, mixed and 2) *Parafilaroidiasis*, multifocal-to-disseminated, moderate and occurring with and without associated inflammatory exudate.

Cervical Skin and Subcutis: Marked, diffuse hyperemia and hemorrhage; moderate-to-marked, widely scattered inflammatory foci of neutrophils and mononuclear cells in subcutaneous tissue surrounding the abscess. The abscess cavity revealed numerous septae tags having marked hyperemia and moderate edema, with minor hemorrhage and leukocytosis. Diagnosis: Cellulitis, cervical, subcutaneous, focal, mixed, probable conspecific trauma.

Liver: Moderate-to-marked, diffuse cytoplasmic vacuolation, compatible with fatty change. Diagnosis: Hepatocellular vacuolative change (fatty liver), diffuse, moderate.

Mesenteric Lymph Node: Moderate-to-marked follicular lymphoid hyperplasia.

Anti-mesenteric Jejunum (from blanched areas): Moderate-to-marked, mural vascular congestion and hemorrhage; diffuse mucosal necrosis and sloughing; moderate-to-marked, mainly mononuclear, mucosal and muscularis inflammatory infiltrate. Diagnosis: Jejunitis, mixed, middle-to-distal, multifocal, moderate- to-severe, necro-hemorrhagic with multiple full thickness mural deficits;

Ileum: Mild-to-moderate, mixed infiltrate of neutrophils, monocytes, lymphoid cells, and some eosinophils, with moderate embedded *Corynosoma sp*. Colon: Moderate numbers of *Corynosoma sp*. embedded in mucosa; submucosa distended by multiple moderate-to-large, proliferating lymphoid follicles. Some lymphoid follicles intimately abutted embedded *Corynosoma sp*. Diagnosis: Ileitis and colitis, diffuse, subacute, mixed

Greater Mesentery (level of lower jejunum and colon): Cuboidal, mesothelial hyperplasia; diffuse, mild-to-moderate edema; marked vascular and lymphatic congestion; moderate-to marked-infiltration by mixed mononuclear cells (85%) and small numbers of neutrophils (15%). Several small arteriolar cross-sections revealed moderate-to-marked neutrophilia, probably resulting from sedimentation.

Discussion

Skeletal muscle and adipose depletion are consistent with starvation, and gross findings of starvation, peritonitis, and sudden death, are common among young marine mammals. In this case, the extreme degree of starvation may have resulted from, or contributed to, multiple intestinal perforations and subsequent peritonitis. A conclusion of starvation is further supported by the histopathologic finding of diffuse hepatocellular vacuolation. The small thin and purplish blue appearance of the spleen likely reflected starvation and hypovolvemia. The kidneys were histologically normal, suggesting that their gross appearance (Figures 2 & 3) may have resulted from being surrounded by peritoneal effusion.

The growth of enteric bacteria from the culture of peritoneal effusion indicates that the proximate cause of the bacterial peritonitis was the multiple intestinal perforations. What is not clear is the proximate cause of the intestinal segmental discoloration, necrosis, and perforations. Among possible causes are: a) mucosal damage and ulceration caused by parasites; b) foreign substances in the bowel; c) bacterial infection following toxic or bacterial ulceration; d) blunt force external trauma.

Comments

Malnutrition results from failure to properly take in or assimilate nutrients and energy. Starvation is the extreme outcome. Starvation is common among orphaned sea lion pups, as it is in other young marine mammals. Orphaning can result from early weaning secondary to separation from the dam during stormy weather, purposeful abandonment, or inadvertent orphaning following severe injury to, or death of, the dam. Young sea lions also may be victims of competition with larger or older individuals, especially in areas where prey are sparse. In these situations, injury or disease may limit or prevent hunting.

Complications of starvation can include immune compromise and aberrant feeding behaviors (ingestion of foreign bodies or unusual prey items). Multi-organ dysfunction may follow these events sequentially, but ingesting non-food objects or prey can lead to complicating injuries, toxin exposure, or introduction of pathogens. Thus, the sequence and contributions multiple possible influences on the death trajectory are difficult to define for this sea lion.

Diagnostic challenges are magnified when dealing with non-domestic species. California sea lions resist physical examination, restraint, and diagnostics such as or blood collection. Wild animals may not respond to painful stimuli in the same way as domestic animals, and often compensate for abnormalities, making them difficult to detect. Additionally, wildlife veterinarians and animal care staff usually do not have the benefit of clinical history. As a result, differential diagnoses may be suspected or established at gross necropsy, and refined based on histopathology, cytology, and microbiology, but it is not unusual for definitive diagnoses to remain elusive.