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What is This?

Clinical and Clinical Laboratory Correlates in Sea Otters Dying Unexpectedly in Rehabilitation Centers Following the *Exxon Valdez* Oil Spill

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Abstract. Following the *Exxon Valdez* oil spill, 347 oiled sea otters (*Enhydra lutris*) were treated in rehabilitation centers. Of these, 116 died, 94 within 10 days of presentation. Clinical records of 21 otters dying during the first 10 days of rehabilitation were reviewed to define the laboratory abnormalities and clinical syndromes associated with these unexpected deaths. The most common terminal syndrome was shock characterized by hypothermia, lethargy, and often hemorrhagic diarrhea. In heavily and moderately oiled otters, shock developed within 48 hours of initial presentation, whereas in lightly oiled otters shock generally occurred during the second week of captivity. Accompanying laboratory abnormalities included leukopenia with increased numbers of immature neutrophils (degenerative left shift), lymphopenia, anemia, azotemia (primarily prerenal), hyperkalemia, hypoproteinemia/hypoalbuminemia, elevations of serum transaminases, and hypoglycemia. Shock associated with hemorrhagic diarrhea probably occurred either as a direct primary effect of oiling or as an indirect effect secondary to confinement and handling in the rehabilitation centers. Lightly oiled otters were less likely to die from shock than were heavily oiled otters (22% vs. 72%, respectively). Heavily oiled otters developed shock more rapidly and had greater numbers of laboratory abnormalities, suggesting that exposure to oil was an important contributing factor.

Key words: Exxon Valdez; oil; sea otters.

Following the oil spill caused by the grounding of the *Exxon Valdez* oil tanker in Alaska's Prince William Sound, a major effort was made to treat oiled sea otters (*Enhydra lutris*) in rehabilitation centers in an attempt to return them to the wild. A total of 347 sea otters were presented to the rehabilitation centers for treatment. Of these 347 sea otters, 116 died while at the centers, 94 (81%) within the first 10 days of presentation. The gross pathologic and histopathologic alterations in otters that died unexpectedly in the centers have been described.[4,5] In this report, we present the clinical findings and principal laboratory changes in otters that died within the first 10 days after entering the rehabilitation centers regardless of degree of oiling or approach to therapy.

Materials and Methods

Data for 21 sea otters were selected for inclusion in this report based upon completeness of clinical records and availability of detailed gross and histopathologic necropsy reports. Otters were divided into three groups on the basis of degree of oiling. Otters with greater than 60% of their bodies covered by oil were classified as heavily oiled, those with 30–60% coverage were considered moderately oiled, and those with evidence of oiling but less than 30% coverage were classified

as lightly oiled. Arbitrary grading of external oiling did not necessarily correlate with measured tissue or blood levels of hydrocarbons.[6]

Clinical laboratory data for these 21 sea otters varied in terms of completeness and reliability more than did either clinical records or pathology reports. Laboratory data were collected primarily to aid the clinician in clinical assessment and treatment; consequently, there was considerable variation in the amount and type of laboratory data available for each otter. Logistic problems in sample management also affected the consistency of the laboratory data. Inclement weather hindered the transport of some samples to the laboratory. In some cases, laboratory data were totally lost as a result of these delays. In other instances, results were reported but were not included in this summary because the problems in sample management were considered so great that laboratory data were regarded as invalid. Every effort was made to include only those data resulting from acceptably collected, transported, and processed samples. Samples were processed through the Physicians Medlab Laboratories, Portland, Oregon.

Results

Sea otter hematology and clinical chemistry reference ranges were established for PML Laboratories (Tables 1, 2) through the analysis of hematology samples collected from eight and serum samples collected

346

Table 1. Hematology reference ranges for sea otters in southeastern Alaska (samples analyzed at PML Laboratories, Seattle, WA). All otters were at least 1 year of age.

Test	Mean (n = 8)	2 SD	Reference Range*
White blood cell count (WBC) (cells/μl)	9,175	4,507	4,668–13,682
Red blood cell count (cells × 10^6/μl)	5.08	0.67	4.41–5.75
Hemoglobin (g/dl)	19	2	17–21
Hematocrit (HCT) (%)	62	10	52–72
Mean cell volume (fl)	122	12	110–134
Mean cell hemoglobin (pg)	37	3	34–40
Mean cell hemoglobin concentration (g/dl)	31	3	28–34
Platelet count (cells × 10^3/μl)	295	129	166–424
Segmented neutrophils (Seg) (cells/μl)	4,136	2,199	1,937–6,335
Lymphocytes (Lymph) (cells/μl)	3,838	3,750	88–7,588
Monocytes (cells/μl)	208	100	108–308
Eosinophils (cells/μl)	942	719	223–1,661
Basophils (cells/μl)	51	102	0–153

Table 2. Serum chemistry reference ranges for sea otters in southeastern Alaska (samples analyzed at PML Laboratories, Seattle, WA). All otters were at least 1 year of age.

Test	Mean*	2 SD	Reference Range†
Glucose (Glu) (mg/dl)	156	92	64–248
Total protein (TP) (g/dl)	6	1	5–7
Creatine (mg/dl)	0.7	0.3	0.4–1.0
Cholesterol (mg/dl)	130	42	88–172
Triglycerides (mg/dl)	72	60	12–132
Alkaline phosphatase (ALP) (U/liter)	96	55	41–151
Aspartate aminotransferase (U/liter)	203	281	0–484
Alanine aminotransferase (ALT, SGPT) (U/liter)	181	147	34–328
Lactate dehydrogenase (U/liter)	374	320	54–694
Total bilirubin (mg/dl)	0.5	0.4	0.1–0.9
Sodium (Na) (meq/liter)	148	21	127–169
Potassium (K) (meq/liter)	3.9	0.7	3.2–4.6
Chloride (meq/liter)	111	15	96–126
Calcium (mg/dl)	8.6	1.4	7.2–10.0
Phosphorus (mg/dl)	4.5	1.8	2.7–6.3
Albumin (ALB) (g/dl)	2.7	0.5	2.2–3.2
Urea nitrogen (BUN) (mg/dl)	50	23	27–73
Creatine phosphokinase (U/liter)	1,643	4,555	0–6,198

* Sample size is 26 (19 females and nine males) except for CK, where sample size is 18.
† Mean ± 2 SD.

from 26 clinically normal adult sea otters from southeastern Alaska. These ranges are similar to those previously reported for other sea otters.[9,10] The reference range was considered to be the mean ± 2 standard deviations with one exception. The reference range for lymphocytes was too large to be clinically useful. Consequently, lymphocyte counts of less than 1,000/μl were regarded as lymphopenia and those greater than 6,000/μl were regarded as lymphocytosis (standard for most nonruminant mammals).[1] All data in this report have been compared with these reference ranges.

Tables 3–5 summarize the laboratory findings for the 21 heavily, moderately, and lightly oiled otters included in this report. Table 6 summarizes the principal syndromes that the laboratory data and clinical comments delineate.

From a laboratory data perspective, the most common hematologic abnormalities included lymphopenia and leukopenia, usually accompanied by increased numbers of immature neutrophils (Tables 3–6). Among heavily oiled animals, six of six had lymphopenia and four of six had leukopenia with increased immature neutrophilis. Of the moderately oiled otters, four of five had lymphopenia and two of five had leukopenia with increased immature neutrophils. Of the lightly oiled otters, four of seven had lymphopenia and two of seven had leukopenia with increased immature neutrophils.

Anemia was also a relatively common hematologic abnormality. Altogether, nine of 18 otters had anemia

(Tables 3–6): three of six heavily oiled otters, one of five moderately oiled otters, and five of seven lightly oiled otters. Reticulocyte counts were not done, and blood films were not available for retrospective study; as a result, the anemias could not be further classified.

The most prevalent abnormalities identified by clinical chemistry included azotemia, hyperkalemia, hypoglycemia, hepatocellular leakage, and hypoproteinemia/hypoalbuminemia (Tables 3–6).

Azotemia, indicated by elevated serum urea, was the most common serum chemistry finding, followed closely by hepatocellular leakage, as indicated by elevated serum transaminases. Six of six heavily oiled otters, three of four moderately oiled otters, and six of nine lightly oiled otters were azotemic. Urine specific gravities were not available to help differentiate prerenal from renal azotemia. However, using the conservative assumption that serum urea levels above 200 mg/dl indicate renal azotemia, at least one of four moderately oiled otters and three of nine lightly oiled otters were in renal failure when sampled near death. Evidence of hepatocellular leakage was present in five of five heavily oiled otters, three of four moderately oiled otters, and five of nine oiled otters.

Table 3. Principal laboratory findings* in heavily oiled sea otters that died within 10 days of presentation at rehabilitation centers in Alaska. Abbreviations are those given in Tables 1 and 2.

Otter No.	Hematology					Serum Chemistry							
	HCT	WBC	Seg	Band	Lymph	Glu	TP	ALB	BUN	ALT	ALP	Na	K
VZ111	↓	↓↓	↓↓	↑↑	↓↓	N	↓↓	↓	↑↑	↑↑	N		
VZ070						N	N	N	↑↑	↑↑	N	↓	↑↑
VZ006	N	↓↓	↓↓	↑↑	↓↓	↓↓	N	↓↓	↑↑	↑↑	N	N	N
VZ135	↓↓	↓↓	N	↑↑	↓↓	N	↓	↓	↑↑	↑↑	↑↑	N	N
VZ035	↑↑	N	N	N	↓↓								
VZ023	↓↓	↓↓	↓↓	↑↑	↓↓	↓	↓	↓	↑↑	↑↑	N	N	N
VZ013	N	N	N	↑↑	↓↓	↓↓	↓↓	↓↓	↑↑		N	N	↑↑
Totals													
↓	3/6	4/6	3/6	0/6	6/6	3/6	4/6	5/6	0/6	0/5	0/6	1/5	0/5
↑	1/6	0/6	0/6	5/6	0/6	0/6	0/6	0/6	6/6	5/5	1/6	0/5	2/5
N	2/6	2/6	3/6	1/6	0/6	3/6	2/6	1/6	0/6	0/5	5/6	4/5	3/5

N = normal; ↓ ↓; = moderate to marked decrease (>3 SD below the mean); ↓ = marginal decrease (2–3 SD below the mean); ↑ ↑ = moderate to marked increase (>3 SD above the mean); ↑ = marginal increase (2–3 SD above the mean).

Hypoglycemia, hypoproteinemia/hypoalbuminemia, and hyperkalemia were somewhat less frequent alterations and were found in nearly equal proportions in the three groups of otters.

The most common clinical syndrome seen regardless of degree of oiling was shock. Shock was usually characterized by hypothermia and lethargy and often by hemorrhagic diarrhea. Shock was occasionally observed at the time of presentation. In heavily and moderately oiled otters, if not evident at presentation, shock generally developed within 48 hours of initial examination at the centers. In the lightly oiled otters included in this report, shock generally occurred during the second week of captivity.

A high proportion of otters in all three groups died with convulsions (Table 6). Four of seven heavily oiled, four of five moderately oiled, and three of nine lightly oiled otters were having seizures at or near the time of death.

Anorexia was also a common clinical problem for otters in the rehabilitation centers (Table 6). Anorexia was reported in three of seven heavily oiled otters, two of five moderately oiled otters, and one of nine lightly oiled otters.

Discussion

The majority of sea otters that died within the first 10 days of confinement in the Valdez and Seward rehabilitation centers appear to have succumbed to shock. Terminal signs included lethargy, anorexia, and convulsions, often accompanied by hemorrhagic diarrhea.

Shock occurred either as a direct effect of oiling or as an indirect effect secondary to confinement and handling in the rehabilitation centers. Lightly oiled otters were less likely to die from shock than were heavily oiled otters (22% vs. 72%, respectively). More heavily oiled otters developed signs of shock more rapidly than did lightly oiled otters. In addition, heavily oiled otters

Table 4. Principal laboratory findings* in moderately oiled sea otters that died within 10 days of presentation at rehabilitation centers in Alaska. Abbreviations are those given in Tables 1 and 2.

Otter No.	Hematology					Serum Chemistry							
	HCT	WBC	Seg	Band	Lymph	Glu	TP	ALB	BUN	ALT	ALP	Na	K
VZ113	N	↓↓	↓↓	↑↑	↓↓	↑↑	N	↓↓	↑↑		N	N	↑↑
VZ100	N	↓↓	↓	N	↓↓		↓↓	↓		↑↑	↑↑	N	↑↑
VZ085	N	N	N	N	↓↓	↓↓	↓	N	N	↑↑	N	N	N
VZ080	N	↓↓	N	N	↓↓	N	N	↑↑	↑↑	N	N	N	↑↑
VZ190	↓↓	↓↓	↓↓	↑↑	N	↓↓	N	N	↑↑	↑↑	N	N	↑↑
Totals													
↓	1/5	4/5	3/5	0/5	4/5	2/4	2/5	2/5	0/4	0/4	0/5	0/5	0/5
↑	0/5	0/5	0/5	2/5	3/5	1/4	0/5	1/5	3/4	3/4	1/5	0/5	4/5
N	4/5	1/5	2/5	3/5	1/5	1/4	3/5	2/5	1/4	1/4	4/5	5/5	1/5

N = normal; ↓ ↓ = moderate to marked decrease (>3 SD below mean); ↓ = marginal decrease (2–3 SD below the mean); ↑ ↑ = moderate to marked increase (>3 SD above the mean); ↑ = marginal increase (2–3 SD above the mean).

Table 5. Principal laboratory findings in lightly oiled sea otters that died within 10 days of presentation at rehabilitation centers in Alaska. Abbreviations are those given in Tables 1 and 2.

Otter No.	Hematology					Serum Chemistry							
	HCT	WBC	Seg	Band	Lymph	Glu	TP	ALB	BUN	ALT	ALP	Na	K
VZ141	↓↓	N	N	↑↑	↓	↑↑	↓↓	N	↑↑	↑↑	↑↑	N	↑↑
SW127	N	N	N	N	↓↓	N	N	N	N	N	N	N	N
SW125	N	N	N	N	N	N	N	N	N	N	N	N	N
SW135						N	N	N	N	N	N	N	N
VZ106	↓↓	↓↓	↓↓	↑↑	↓	N	↓↓	↓	↑↑	↑↑	↑↑	N	↑↑
VZ011	↓↓	↓↓	↓↓	↑↑	↓↓	↓↓	↓↓	↓	↑↑	N	N	N	↑↑
VZ081	↓↓	N	N	↑↑	N	↓↓	N	N	↑↑	↑↑	N	N	↑↑
VZ136	↓↓	N	N	↑↑	N	↓↓	N	↓↓	↑↑	↑↑	N	N	↑↑
SW120						↓↓	↓↓	↓↓	↑↑	↑↑	N	N	↑↑
Totals													
↓	5/7	2/7	2/7	0/7	4/7	4/9	4/9	4/9	0/9	0/9	0/9	0/9	0/9
↑	0/7	0/7	0/7	5/7	0/7	1/9	0/9	0/9	6/9	5/9	2/9	0/9	6/9
N	2/7	5/7	5/7	2/7	3/7	4/9	5/9	5/9	3/9	4/9	7/9	9/9	3/9

N = normal; ↓↓ = moderate to marked decrease (>3 SD below mean); ↓ = marginal decrease (2–3 SD below the mean); ↑↑ = moderate to marked increase (>3 SD above the mean); ↑ = marginal increase (2–3 SD above the mean).

generally had a higher proportion of laboratory abnormalities than did lightly oiled otters. These findings suggest that exposure to oil was an important contributing factor to decline and death in these otters.

Laboratory findings correlated for the most part with clinical observations. The most frequent hematologic finding was lymphopenia. The most common causes of lymphopenia in domestic animals are 1) systemic stress, which results in increased endogenous production of glucocorticoids and resultant sequestration and possible destruction of circulating lymphocytes, or 2) exogenous steroid administration.[1] Certainly the otters in rehabilitation centers were stressed. In addition, many received steroid therapy upon initial presentation.

Leukopenia characterized by neutropenia with increased numbers of immature neutrophils (a degenerative left shift) was also common. A degenerative left shift suggests severe inflammation.[1] It is frequently observed in animals suffering from diarrhea, gastrointestinal hemorrhage, and possible secondary gastroenter-

itis. Such a scenario is certainly possible in those otters suffering from hemorrhagic diarrhea.

Hyperkalemia and hypoproteinemia/hypoalbuminemia were probably also related to diarrhea and gastrointestinal hemorrhage. Hyperkalemia was at least partially the result of release of potassium from dying cells in the areas of hemorrhage.[1] Acidosis, a common accompaniment of diarrhea, can also be a cause of hyperkalemia.[1] Hypoproteinemia and hypoalbuminemia were probably the result of protein loss in the diarrhea fluid.

Azotemia was the most common syndrome identified from the laboratory data. Because urine specific gravity results were not available for these animals, prerenal azotemia could not be absolutely differentiated from renal azotemia. However, necropsies did not reveal significant renal lesions, although renal lipidosis was noted.[4] It is therefore most likely that shock or dehydration led to prerenal azotemia as a result of decreased renal perfusion. In the few animals that probably had true renal azotemia (those with serum

Table 6. Principal clinical and laboratory findings in sea otters that died in rehabilitation centers in Alaska.

Otters	Shock	Convulsions	Anorexia	Lymphopenia	Leukopenia, Increased Bands	Anemia	Azotemia	Hyperkalemia	Hypoglycemia	Hepatocellular Leakage	Hypoproteinemia/ Hypoalbuminemia
Heavily oiled	5/7	4/7	3/7	6/6	4/6	3/6	6/6	2/5	3/6	5/5	4/6
Moderately oiled	4/5	4/5	2/5	4/5	2/5	1/5	3/4	4/5	2/5	3/4	2/5
Lightly oiled	7/9	3/9	1/9	4/7	2/7	5/7	6/9	6/9	3/9	5/9	4/9
Total	16/21	11/21	6/21	14/18	8/18	9/18	1/19	12/19	8/20	13/18	10/20

urea nitrogen values greater than 200 mg/dl), it is likely that long-standing reduced renal perfusion eventually led to primary renal injury near the time of death.

Hypoglycemia probably resulted from anorexia. Sea otters have high metabolic rates and devour large quantities of food.[7] Anorexia would be expected to quickly deplete hepatic glycogen stores, resulting in hypoglycemia. Hypoglycemia was probably the cause of many of the terminal convulsions.

Hepatocellular leakage may have been a reflection of primary hepatotoxicity but may also have been a nonspecific change associated with anorexia. During fasting, decreased availability of carbohydrates as an energy source leads to mobilization of fat from tissue stores to the liver. Increases in fat within hepatocytes result in increased cell membrane permeability with leakage of transaminases into the blood. In these otters, elevated transaminases correlated most frequently with hepatic lipidosis histopathologically.[4]

The cause(s) of anemia in these otters is difficult to determine, especially because the anemias were not well characterized. Certainly in those animals with gastrointestinal hemorrhage, blood loss must be considered a contributing factor. Oil exposure has been reported to cause Heinz body hemolysis in birds.[2,3] The possibility of Heinz body hemolysis in the otters could neither be substantiated nor refuted, but laboratory reports gave no indication that Heinz bodies were ever observed in the 21 animals evaluated for this report. Heinz bodies were described in several otters transferred from Valdez to Vancouver Aquarium in April 1989.[8]

Acknowledgements

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