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# Meloxicam may induce nephrotoxicity in California sea lions (*Zalophus californianus*) with chronic domoic acid toxicosis

Mattison Peters, DVM, MPH[1]*; Pádraig Duignan, DVM, PhD[1]; Margaret E. Martinez, DVM, DACVP, PhD[1]; Emily Trumbull, DVM[1]; Mark G. Papich, DVM, DACVCP[2]; Cara L. Field, DVM, PhD, DACZM[1]

[1]The Marine Mammal Center, Sausalito, CA
[2]College of Veterinary Medicine, North Carolina State University, Raleigh, NC

*Corresponding author: Dr. Peters (mpeters@marinemammalcare.org)

**Objective**
To describe biochemical and pathologic findings associated with meloxicam-associated nephrotoxicity in California sea lions (CSLs) with concurrent evidence of chronic domoic acid toxicosis.

**Animals**
Stranded free-ranging CSLs treated with meloxicam while undergoing rehabilitation in California between 2017 and 2023.

**Clinical Presentation**
6 adult females and 1 male pup were included in this case series due to postmortem evidence of meloxicam-associated nephrotoxicity. Five patients were in treatment for suspected domoic acid toxicosis, and 2 were in treatment for malnutrition. Three individuals showed evidence of antemortem renal impairment (azotemia, electrolyte derangements, and clinical decline), while the other 4 were diagnosed postmortem.

**Results**
6 of the 7 CSLs for which serum was available before and after treatment with meloxicam developed a significant increase in serum creatinine, BUN, phosphorous, and potassium following treatment. At necropsy, renal papillary necrosis was evident on gross and histologic examination consistent with meloxicam-associated nephrotoxicity for all 7 cases. These individuals also had hippocampal atrophy supportive of chronic domoic acid toxicosis.

**Clinical Relevance**
This is the first report of meloxicam-associated nephrotoxicity in pinnipeds. While multiple factors such as dehydration, nutrition, other medication, and/or compromised immune function may have contributed to the development of nephrotoxicity, further evaluation of renal effects of domoic acid as well as effects of NSAIDs is warranted in CSLs.

**Keywords:** meloxicam, NSAID, nephrotoxicity, California sea lion, domoic acid

Meloxicam is an NSAID that inhibits cyclooxygenase (COX)-mediated prostaglandin synthesis and is widely used in veterinary medicine. The enzyme COX has 2 main isoforms, COX-1 and COX-2. Prostaglandins synthesized by COX-1 are primarily constitutively expressed and are involved in maintenance of normal gastrointestinal and renal function, while those synthesized by COX-2 (primarily inducible expression) more commonly mediate pain and inflammation, although crossover can occur. Cyclooxygenase 2–preferential or –selective NSAIDS are considered to have fewer detrimental side effects, and although meloxicam inhibits COX-2 more than COX-1, adverse effects of meloxicam administration, including gastrointestinal ulceration, hepatotoxicity, and nephrotoxicity, have been documented in multiple species.[1–3]

Nephrotoxicity associated with NSAID administration is more likely to occur at higher dosages, in dehydrated patients, and/or in patients with preexisting renal disease.[4] The pathophysiology of kidney injury associated with NSAID administration in humans and domestic mammals is attributed to inhibition of prostaglandins that maintain renal perfusion and glomerular filtration rate as well as sodium and water retention, culminating in renal vasoconstriction and ischemia.[5–7] Within the kidney, renal papillae are particularly susceptible to ischemic damage due to relatively low perfusion. Renal ischemia due to NSAID toxicity can thus result in acute or chronic renal papillary necrosis.[6,8,9]

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Meloxicam is commonly administered to pinnipeds in managed care and rehabilitation, and associated toxicity has not been reported in these species.[10] Pharmacokinetic data suggest that presumptive therapeutic blood concentrations of meloxicam are achieved with a single dose of 0.1 mg/kg in California sea lions (CSLs) following oral administration. Although the half-life after a single dose in CSLs is approximately 33 hours, meloxicam was still detectable in plasma 96 hours after oral administration.[11] There are no data on plasma concentrations in CSLs following repeated dosing.

Domoic acid (DA) toxicosis is a condition caused by exposure to the neurotoxin DA produced by diatoms of the genus *Pseudo-nitzschia*. Domoic acid binds to glutamate receptors in the brain and heart of mammalian species and keeps these receptors activated, with subsequent development of debilitating and often lethal neurologic and/or cardiac disease in numerous species. Over the past 25 years, thousands of CSLs have stranded along the eastern Pacific coast suffering from the toxic effects of DA, also referred to in humans as amnesic shellfish poisoning.[12] Domoic acid toxicosis can present as an acute or chronic syndrome in CSLs, and those with chronic disease may have comorbidities that contribute to their stranding.[13] Characteristic findings in CSLs with acute DA toxicosis include neuronal necrosis that progresses to hippocampal atrophy in the chronic form.[13,14] Degenerative cardiomyopathy has also been documented with both syndromes.[13] Nephrotoxicity associated with DA toxicosis has not been identified in CSLs, although DA has been documented to be excreted by the kidneys of multiple species, including CSLs,[1,12,15] glutamate receptors have been documented in rodent and human kidneys,[16–18] and studies in laboratory rodents have documented kidney injury when they are exposed to high levels of DA.[1]

The present case series documents the first report of meloxicam-associated nephrotoxicity in stranded CSLs undergoing rehabilitation. While only some individuals were diagnosed with suspected DA toxicosis while in care, all had histopathologic changes consistent with DA exposure. Our aim is to raise clinical awareness of the potential for nephrotoxicity associated with meloxicam use in CSLs, as well as to highlight the need for further research on renal effects of DA as a potentially significant comorbidity.

## Methods

Records for CSLs that were treated with at least 1 dose of meloxicam either orally or parenterally while undergoing rehabilitation at The Marine Mammal Center (TMMC) in Sausalito, California, between 2017 and 2023 were identified by a search of the TMMC internal patient database. Every patient admitted into rehabilitation at TMMC is assigned a unique identifier within the database, and full medical records, including all medications prescribed and delivered, are recorded and searchable, yielding a very high standard of data collection for each individual sea lion. All sexes, age classes, weights, outcomes, and reasons for meloxicam treatment were included in the initial search. Only cases that had renal histology available were considered for inclusion in this study, and records were reviewed for gross necropsy and/or histological findings of renal papillary necrosis. Cases that included gastrointestinal ulceration without parasitic etiology were also evaluated for concurrent nephropathy. For each case in which renal papillary necrosis was identified, clinical pathology, gross necropsy, and histopathology data were compiled for review.

All sea lions were housed and cared for following rescue as previously described.[11] Blood samples were collected from individuals during admission examination and as deemed medically necessary by the attending veterinarian. Serum biochemical values (creatinine, BUN, sodium, phosphorous, and potassium) that were not within internally established reference intervals after meloxicam administration were compared to values obtained on admission (pretreatment) using paired *t* tests. In the cases for which banked serum collected during the course of meloxicam treatment was available (saved under TMMC IACUC protocol 2021-1-1; stored at –80 °C), serum was evaluated for meloxicam concentrations with high-pressure liquid chromatography at the Clinical Pharmacology Laboratory at the College of Veterinary Medicine at North Carolina State University, as previously described.[11]

Postmortem, bodies were chilled (4 °C) and necropsied within 24 hours of death following standard protocols and included documentation of all significant gross lesions by photography. Tissue samples from all organs were sampled for histopathology following standard TMMC protocols, fixed in 10% formalin, trimmed in-house, and processed for H&E staining by the Veterinary Medical Teaching Hospital histopathology laboratory at the University of California-Davis. Serum was submitted to the California Animal Health and Food Safety Laboratory for leptospirosis testing.

### *Statistical analysis*

Serum creatinine, BUN, sodium, phosphorous, and potassium were compared before and after meloxicam treatment via paired *t* tests with Prism, version 10.1.1 (GraphPad Software).

## Results

### *Clinical presentation*

There were 256 CSLs treated with meloxicam between 2017 and 2023 at TMMC, and 33 of them (12.9%) had renal histology available for review. Seven cases (2.7%; 6 adult females and 1 male pup [approximately 10 months old]) were identified as having renal papillary necrosis consistent with NSAID-induced nephropathy. Five of the 6 adult females stranded with suspected DA toxicosis based on seizure activity or other neurologic abnormalities and concurrent spatiotemporal stranding during a known harmful algal bloom. One adult female was diagnosed with malnutrition, traumatic wounds, and

abnormal mammary tissue, and the pup was diagnosed with malnutrition following maternal separation and with pneumonia. Three of the 7 patients were treated with meloxicam due to the development of keratopathy in care (1 for rectal prolapse, 2 for traumatic wounds, and 1 for inflammation associated with verminous pneumonia). Three individuals were given an initial single dose of approximately 0.2 mg/kg of meloxicam (2 IM and 1 PO) followed by repeated doses of 0.1 mg/kg, PO, every 24 hours. Treatment was initiated between 5 and 21 days following stranding, and duration of treatment ranged from 3 to 16 days **(Figure 1)**. All animals were voluntarily eating frozen thawed herring (*Clupea* spp) at the time treatment was initiated. Five of the 7 cases were euthanized via IV sodium pentobarbital and phenytoin following a decline in health status. For 1 of these 5, renal ultrasonography was performed immediately prior to euthanasia, and a markedly increased echogenicity of the renicular cortices with concurrent hypoechoic medullae was noted **(Figure 2)**. Two animals died in treatment with nephropathy as the primary cause of death and DA toxicosis as a contributing factor as determined from necropsy and histopathology. Asphyxiation (drowning) was also a proximate cause of death for 1 case.

*Clinical pathology*

Of the 7 animals that developed nephrotoxicity, 6 had serum biochemical results available for review before and after meloxicam treatment. None of the individuals were azotemic on admission to rehabilitation, and serum creatinine, BUN, phosphorous, and potassium values were significantly different pretreatment compared to posttreatment **(Figure 3)**. Serum sodium, GGT, AST, and ALT did not differ **(Supplementary Table S1)**. Four animals had serum meloxicam concentrations evaluated, 3 at 96 hours following treatment (0.246 µg/mL, 0.263 µg/mL, and 0.074 µg/mL) and 1 at 16 hours following treatment (0.294 µg/mL). Serology for *Leptospira interrogans* serovar Pomona was negative in all 7 individuals. While only 2 individuals had urinalyses performed both before and after meloxicam treatment, 3 urinalyses were performed on admission (pretreatment) and were considered unremarkable, whereas 4 urinalyses were performed on animals posttreatment and were all minimally concentrated (urine specific gravity, 1.014 to 1.024), with 1 containing granular casts and 1 containing renal tubular epithelial cells indicative of tubular damage. Blood was identified on 2 pretreatment and 2 posttreatment urinalyses, though this finding may have been associated with cystocentesis **(Supplementary Table S2)**.

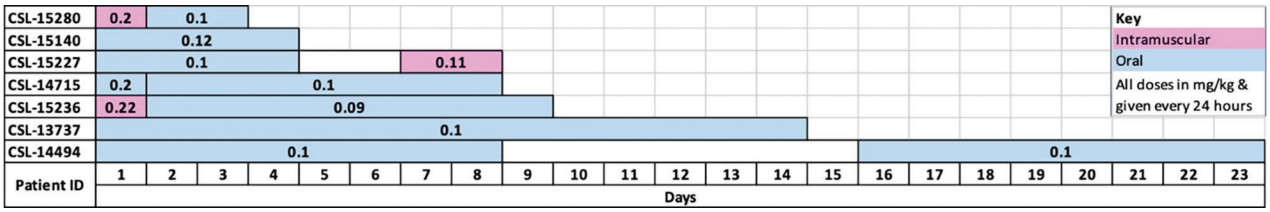


**Figure 1**—Dose and duration of meloxicam treatment for each California sea lion included in this series. Pink bars represent IM administration, and blue bars represent oral administration. Doses are provided in milligrams per kilogram within each bar.

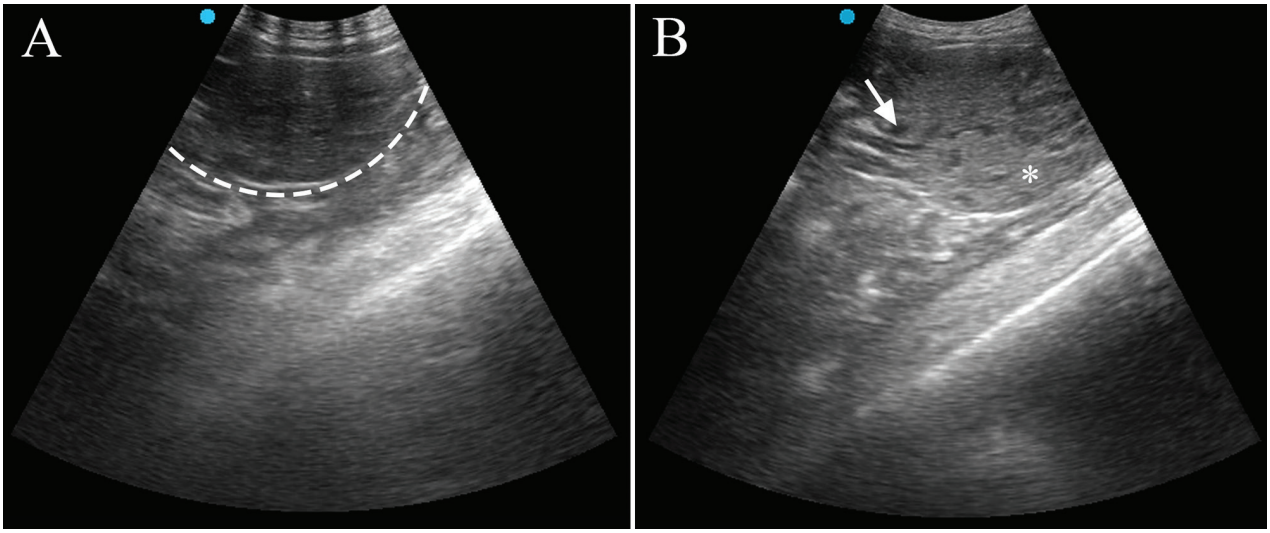


**Figure 2**—Transverse ultrasound images of (A) normal California sea lion kidney with ventral capsular surface demarcated by dashed line and (B) abnormal California sea lion kidney obtained from a California sea lion (designated *CSL-15236*) following meloxicam administration at the time of euthanasia with markedly increased echogenicity of the renicular cortices (asterisk) with concurrent hypoechoic medullae (arrow).

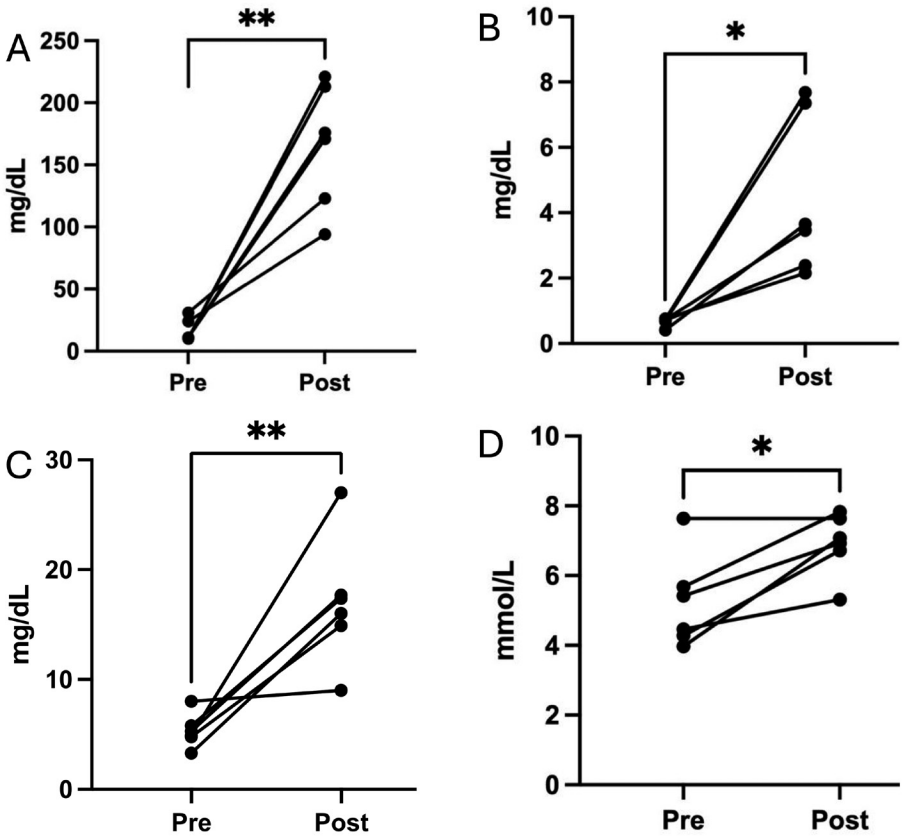


**Figure 3**—Serum biochemical changes in (A) BUN, (B) creatinine, (C) phosphorous, and (D) potassium pre- and postmeloxicam administration for 6 California sea lions. The asterisk indicates paired *t* test results with *P* value < .05, and the double asterisks indicate paired *t* test results with *P* value < .01.

*Gross necropsy*

Three of the case animals had moderately enlarged (approx 20% to 25%) kidneys that were diffusely pale on the capsular surface, while 2 others were pale but not appreciably enlarged. One had congested capsular blood vessels. On section, 5 had variably expanded pale tan to yellow renicular cortices, and 4 had marked corticomedullary congestion **(Figure 4)**. For all 7, the renicular papillae were variably swollen (edematous), were streaked or stippled white (dystrophic mineralization), and had a yellow cast (sloughed necrotic tubular epithelium). An incidental finding in 1 kidney from 1 adult female was several thin-walled fluid-filled cysts consistent with tubular ectasia from nonspecific tubular injury.

On serial coronal sectioning of formalin-fixed brains, the 6 adult females had grossly apparent lesions, including symmetric or asymmetric atrophy and distortion of hippocampal complexes (amygdaloid body, cranioventral hippocampus, and parahippocampal gyrus) and cerebral blood vessel congestion. Other gross findings included hemorrhagic gastric ulcers (2 cases), mild periacinar hepatic pallor (4 cases), and a firm and discolored mammary gland (1 case). For 1 animal found dead in its pool, the stomach contained at least 2 L of pool water and the lungs were diffusely congested, consistent with asphyxiation. This animal had marked atrophy of the ventral hippocampal complex bilaterally and was suspected to have had a seizure in its pool. Parasite burdens and associated gross findings in the gastrointestinal and respiratory tracts for all cases were within expected ranges for the species.

*Histopathology*

The kidneys of all 7 individuals had marked renal papillary coagulation necrosis with varying degrees of renal papillary mineralization and subsequent reflux nephropathy characterized by tubular ectasia, degeneration, and necrosis (Figure 4). One also had mild proximal convoluted tubule mineralization, and 1 had mild renal cortical amyloidosis.

All 7 brains had lesions consistent with DA encephalopathy. Two had moderate chronic changes characterized by hippocampal atrophy (1 was left sided, and 1 was symmetrical). The other 5 showed marked acute-on-chronic changes characterized by unilateral or bilateral acute neuronal necrosis superimposed on preexisting hippocampal atrophy (Figure 4). One adult female with acute-on-chronic DA encephalopathy also had myocardial necrosis and interstitial fibrosis consistent with that etiology. Amyloidosis involving cerebral choroid plexus and pituitary was present in 3 adult animals, including the aforementioned case with renal amyloid. Two adults had invasive urogenital carcinoma limited to the cervix (1 case) or to the cervix and vagina (2 cases) with no evidence of urinary tract involvement. Chronic mastitis was confirmed for 1 animal.

Six out of 7 cases had liver available for histopathologic examination. Liver changes comprised mild periacinar pallor, hepatocellular atrophy, and bile retention. Two individuals had mild to moderate biliary ductular hyperplasia. Stomach was available for histopathologic examination in 6 out of 7 cases, and in 2 individuals, gastric ulceration and hemorrhage were observed.

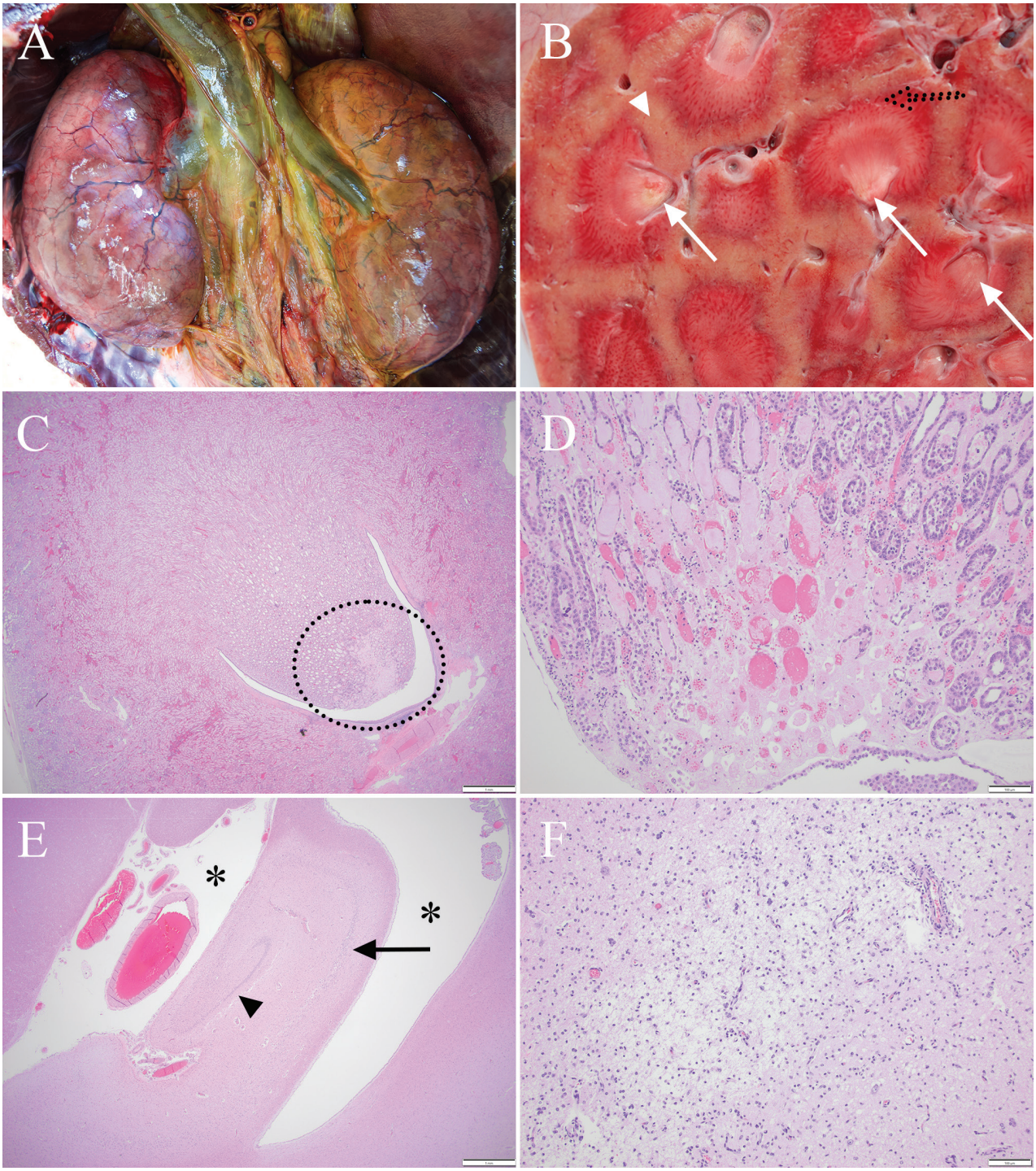


**Figure 4**—A—Enlarged and diffusely pale kidneys in situ of CSL-15236, an adult female California sea lion. B—California sea lion kidney as in (A) removed and sectioned to show its renicular architecture and expanded pale tan cortices (arrowhead), congestion of blood vessels at the corticomedullary junctions and pale white-streaked papillae (white arrows). C—Histology of the same sea lion kidney as in A and B showing a complete reniculus with focal pallor (necrosis) of the distal papilla (dashed circle). 12.5X magnification; H&E stain; scale bar = 1 mm. D—Higher-power (100X) view of a distal papilla showing loss of tubules, interstitial edema, and focal hemorrhage. H&E stain; scale bar = 100 µm. E—Right ventral hippocampus of CSL-15236 showing marked atrophy of the hippocampal complex (chronic lesion) and relative expansion of the ventral horn of the lateral ventricle surrounding it (stars); the neuron-depleted cornu ammonis (black arrow) and dentate gyrus (arrowhead) are indicated. H&E stain; 12.5X magnification; scale bar = 1 mm. F—Higher-power view of the same hippocampus as in E showing focal malacia (area of pallor) with perivascular and interstitial edema and loss of neuropil (acute lesion). H&E stain; 100X magnification; scale bar = 100 µm.

## Discussion

This case series is the first to document adverse effects of NSAID administration in CSLs. Histopathologic findings in these cases were consistent with lesions identified in other species with NSAID-induced nephropathy as characterized by renal papillary necrosis. Two animals also had deep chronic gastric ulceration with hemorrhage that may have been associated with meloxicam-induced COX inhibition or, given that these were all free-ranging animals, some other undetermined process.[19] Hepatotoxicity was not identified in any of the cases. Meloxicam has been used regularly in CSLs at TMMC for more than 7 years with an overall low incidence (2.7%) of documented adverse effects (presented here), all of which involved nephrotoxicity. This incidence, however, is higher than what has been reported in dogs and cats that developed renal insufficiency following injectable meloxicam administration (0.087%), and emesis is the most common adverse effect reported in dogs and cats.[20] Although incidence of emesis following meloxicam administration was not evaluated in this study, it was not documented in any of the 7 cases in this series. In general, adverse drug effects are thought to be underreported in animals.[21]

While each affected individual in this case series also had lesions consistent with DA toxicosis, not all CSLs with DA toxicosis that were treated with meloxicam showed evidence of nephrotoxicity (TMMC, unpublished data, 2024). However, only animals with available renal histopathology results were included; the possibility of subclinical adverse effects in a larger subset of these patients cannot be ruled out. Given findings of renal glutamate receptors in humans and rodents,[16–18] renal excretion of DA,[12,15] and potential for nephrotoxicity at high levels in rodents,[2] as well as the frequency of DA intoxication in CSLs,[12–14] further investigation into renal effects of DA in CSLs is warranted and is underway at TMMC.[22]

Although the majority of patients historically treated with meloxicam at TMMC showed no adverse effects with the previously canine-derived and recommended dose of 0.1 mg/kg, every 24 hours (often with an initial loading dose of 0.2 mg/kg),[23] recent pharmacokinetic data suggest that less frequent dosing is likely indicated in CSLs.[11] Pharmacokinetic data are only available after a single oral dose of 0.1 mg/kg meloxicam; thus, direct comparison with repeated dosing in both healthy and compromised animals is not available. The animals included in this series were given multiple doses of meloxicam every 24 hours administered orally and/or parenterally. However, 2 of the 4 cases had serum concentrations that exceeded the maximum serum concentration of meloxicam for CSLs in a single-dose pharmacokinetic study. Factors that may contribute to the high serum concentrations when compared to the maximum serum concentration obtained after a single dose in healthy young CSLs include repeated treatment, age class, dehydration, comorbidities, nutrition, and immune function. Serum concentrations considerably higher than these values have been documented in other species without associated adverse effects, though tolerance and dosing of meloxicam vary greatly by species.[4,24,25] Meloxicam metabolism in marine mammals has not been evaluated, though research in humans and rats has suggested that it is protein-bound and primarily undergoes hepatic metabolism with limited urine and fecal elimination of the native compound.[11]

In cases of impaired renal function, as described in cats and dogs with chronic renal disease, meloxicam dose reduction is recommended.[7,26] Impacts of renal impairment on drug handling are complex, but may include variation in gastrointestinal absorption, volume of distribution, and renal drug excretion.[7] Although the CSL cases described in this series did not have evidence of renal disease prior to meloxicam administration, more sensitive metrics of renal function, including glomerular filtration rate, urine specific gravity, and urinary protein-to-creatinine ratios, were not evaluated. Strikingly, each individual was diagnosed with chronic DA toxicosis postmortem. Impacts of DA on renal function in CSLs is unknown, though nephrotoxic effects including renal tubular and vascular damage have been documented in other species.[1] Further investigation of the potential impact of DA on CSL renal function is underway and may include direct or indirect vasoconstrictive effects resulting in renal ischemia and/or direct effects on renal papillary tubules.[22] Either mechanism may be exacerbated by the effects of NSAIDs. Although meloxicam has been used safely and effectively in many marine mammal patients, caution is recommended, particularly in patients with suspected exposure to DA, other gastrointestinal and renal comorbidities, or unknown clinical histories.


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### ORCID

Mattison Peters https://orcid.org/0000-0003-0645-4233


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## Supplementary Materials

Supplementary materials are posted online at the journal website: avmajournals.avma.org.