Steatitis in a Wild Common Loon (*Gavia immer*) and Review of the Literature

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Abstract: Steatitis was diagnosed at necropsy in a debilitated, adult female common loon (*Gavia immer*), constituting the first known case of the disease in loons. Steatitis has been described most commonly in a variety of domestic and captive species. In these cases, the cause of steatitis is most often vitamin E deficiency in a diet comprised mainly of fish. Until roughly 20 years ago, the occurrence of steatitis in wildlife was relatively rare. However, the condition is being seen increasingly in herons in Japan and both coasts of the US. While several environmental factors have been correlated with the outbreaks, the causes remain unclear and may be multifactorial. Rehabilitators are in a unique position to recognize and report the event of steatitis in wild birds such as loons, allowing researchers to better understand the extent and cause of steatitis in wild birds.

Key words: steatitis, fat, common loon, *Gavia immer*, vitamin E

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Case Presentation

In June of 2004, an adult female common loon (*Gavia immer*) was retrieved from Togus Pond in Augusta, Maine and brought to Avian Haven, a wild bird rehabilitation center. The loon was observed to be quiet, weak, lethargic, thin, and unconcerned with human presence. The loon died a short time later and was shipped to Tufts Cummings School of Veterinary Medicine as a part of an ongoing regional mortality study. The body was weighed, radiographed, and a necropsy was performed. The bird was thin (2182 g, normal weight 3380–4650 g, Dunning 2008). Radiographs revealed no significant findings. At necropsy there were good fat stores throughout the body, including abdominal fat and coronary fat; however, the general muscle condition was poor. The subcutaneous fat in the abdominal area was hardened and yellow–brown, while all other fat within the body appeared normal. The gastrointestinal tract was distended with gas and a small portion of the right liver lobe appeared necrotic. Numerous fungal plaques were noted on the outside of the trachea, major vessels, pericardium, within the lungs, and on airsac walls. These plaques appeared cream colored with a gray/green center, suggesting infection with Aspergillus sp.

Tissues from the necropsy were sent for histological examination (Northwest Zoopath, Monroe, WA) and a diagnosis of severe necrotizing steatitis with intralesional bacteria was made. Adipose tissue was found with severe, multifocal to coalescing, granulomatous or heterophilic inflammation and necrosis was associated with large numbers of extracellular and intracellular bacteria. Based on previous incidences of steatitis involving cyanobacteria (blue–green bacteria) producing the toxin microcystin, samples from the loon were tested for the presence of microcystin and were found to be negative. Water from Togus Pond and neighboring regions was not tested.

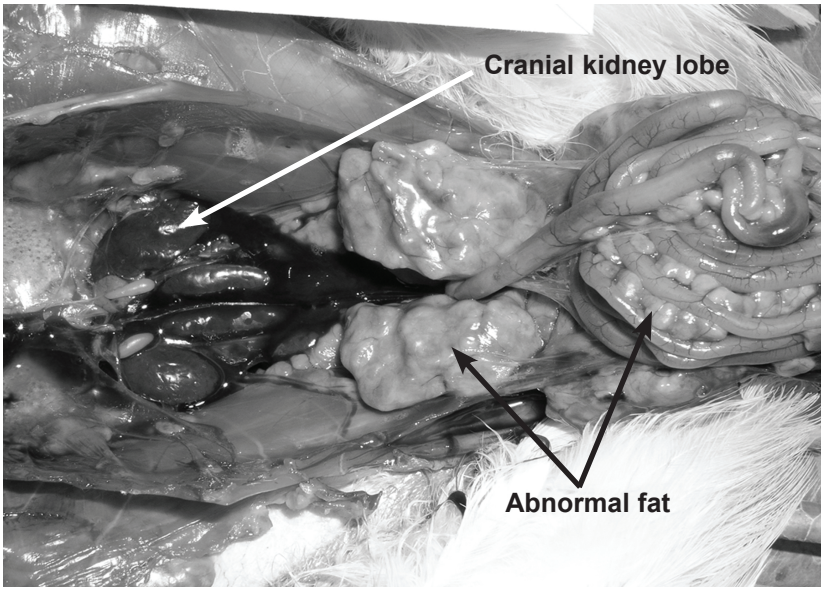


*Figure 1. Necropsied common loon (*Gavia immer*) showing abnormal subcutaneous fat deposits in the abdominal cavity.*

This is the first known case of steatitis reported in a common loon. The diet of the loon prior to its death is unknown. As adults, loons occupy freshwater environments during the breeding season and marine environments during the non–breeding season, and are primarily fish eaters. However, they will eat what they can see easily and capture readily, including invertebrates and some plant material.

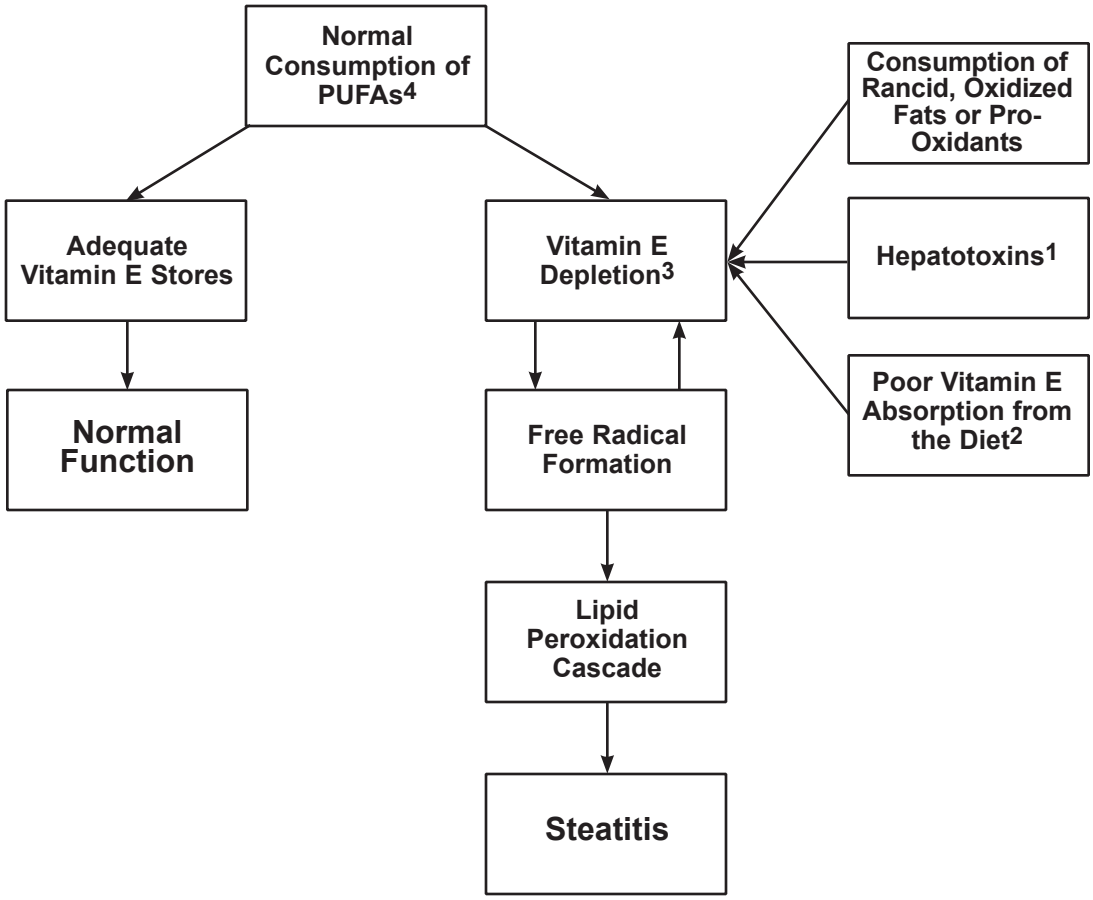
Review

Steatitis is an inflammatory disease of adipose tissue (fat) with accompanying deposition of ceroid pigment (a form of oxidized fats) within the cells (Kahn 2005; Niza et al 2003). This condition results from excessive lipid peroxidation and has been described previously in cats (Niza et al 2003), mustelids (Brooks et al 1985), equids (Menzies–Gow et al 2002), swine (Kirby 1981), primates (Juan–Salles et al 2003), kangaroos (Kabak et al 2011), fish (Begg et al 2000; Huchzermeyer et al 2011), reptiles (Ladds et al 1995), crocodiles (Huchzermeyer et al 2011), and birds (Pollock et al 1999; Wong et al 1999; Neagari et al 2011). In the western United States (US), steatitis was first recorded in wild great blue herons (*Ardea herodias*) over 20 years ago. Since then, an increasing number and frequency of cases have been seen in both this species and black–crowned night herons (*Nycticorax nycticorax*), to the point that in the San Diego area it is difficult to find young great blue herons that do not manifest the syndrome (Dr. Judith St. Leger, SeaWorld of California, San Diego, CA, personal communication). In 2008, additional steatitis outbreaks in wild populations were reported in herons and egrets in a reservoir in Japan (Neagari et al 2011), and in crocodiles and catfish in Kruger National Park, South Africa (Huchzermeyer et al 2011).

Symptoms of steatitis typically include fever, anorexia, depression, hyperesthesia (abnormal sensitivity to stimuli), reluctance to move, pain, and subcutaneous, irregular nodular masses (Koutinas et al 1993). Muscle degeneration also has been seen to occur in combination with nutritional steatitis (Brooks et al 1985) and affected birds have been found to present with the steatitis of several fat stores, weakness, lethargy, muscle atrophy, inability to fly, and aspergillosis infection.

Lipid peroxidation leading to steatitis can result from an imbalance between pro–oxidant compounds and antioxidant defenses, specifically the balance between the potent antioxidant vitamin E and polyunsaturated fatty acids (PUFAs). These fatty acids are particularly susceptible to peroxidation by free radicals and pro–oxidants (Webb and Twedt 2008; Niza et al 2003). The imbalance between vitamin E and PUFAs can be triggered by inadequate intake of vitamin E in the diet, consumption of high levels of PUFAs that deplete vitamin E, or a combination of the two (Niza et al 2003). Additionally, omega–3 fatty acids are more vulnerable to peroxidation than omega–6 fatty acids (Surai et al 2001). PUFAs from marine fish are predominantly omega–3 fatty acids (Brooks et al 1985; Li et al 2011). Therefore, adequate consumption of vitamin E is invaluable to species that consume diets composed primarily of marine fish, as their requirements for the vitamin are higher (Dierenfield 1989, also personal communication 2012, Novus International, St. Louis, MO). Indeed, dietary steatitis has been induced in a number of captive species fed diets high in marine fish but deficient in vitamin E (Brooks et al 1985; Niza et al, 2003; Larsen et al 1983; Tocidlowski et al 1997; Manawatthana and Kasorndorkbua 2005).

Lipids are naturally occurring fats obtained in the diet and necessary for normal functions. Lipid peroxidation is a process whereby lipids that are part of cell membranes become chemically modified leading to cell and tissue damage. Polyunsaturated fats (PUFAs) are a type of lipid that is particularly susceptible to peroxidation.



[1]Hepatotoxins can include compounds such as organic poisons (eg: microcystin), heavy metals, or certain drugs.
[2]Poor vitamin E absorption can be caused by the absence of hepatic or biliary secretions, or any disorder that causes fat malabsorption.
[3]Other factors that contribute to vitamin E depletion include the depletion of antioxidants (eg: vitamin C or selenium), depletion of enzyme co-factors (eg: Zn, Cu or Mn), or excess heat.
[4]Balance of omega-3 to omega-6 ratio is also an important factor.

Thus, factors that prevent adequate consumption or absorption of vitamin E or any factor that depletes the body's vitamin E may lead to steatitis in species that regularly consume high levels of PUFAs. The presence of heavy metals can accelerate the peroxidation of PUFAs (Geraci and St. Aubin 1980; Van Vleet 1982) and anything toxic to the liver (hepatotoxins) can contribute to liver oxidative stress via a number of pathways (Jaeschke et al 2002; Jaeschke 2012). Vitamin E has been shown to decrease the effects of these hepatotoxins (Sokol et al 1998) and thus it is plausible that the presence of certain hepatotoxins also can lead to the depletion of vitamin E.

Metabolism and excretion of vitamin E are important to its abundance and availability in the body (Traber 2005). Vitamin E is absorbed and delivered to the liver before being metabolized and excreted into the bile or urine (Traber 2005). Absorption requires biliary and pancreatic secretions and undergoes similar processes as those used to digest and metabolize fatty acids (Traber 2005). In the absence of pancreatic and/or biliary secretions, vitamin E absorption and secretion into the lymphatic system are poor or negligable (Traber 2005). Thus, any disorder that causes fat malabsorption or disrupts biliary and pancreatic secretions can lead to vitamin E deficiency (Traber 2005; Ohno et al 2003; Outas et al 2004; Potts et al 1975). Other factors that may increase the dietary requirements of vitamin E include increased vitamin A consumption, decreased consumption of other antioxidants or cofactors such as selenium, consumption of rancid (oxidized) fats, and high levels of pro–oxidants such as microcystin or aluminum (Klasing 2000; Brown 2011).

The authors suggest two broad pathways that may place wild species (piscivorous populations in particular) at an increased risk for steatitis:

1. Ecological changes such as overfishing (Jennings and Kaiser 1998; Coll et al 2008) or climate change (Walther et al 2002) that alter marine trophic structure and may shift prey items to those with higher PUFA concentration than the predator species are capable of metabolizing and detoxifying, thus depleting the available vitamin E and leading to steatitis.
2. Environmental contaminants such as microcystin, heavy metals, or other pollutants may be depleting the animal's vitamin E or disrupting its absorption and metabolism, thus leaving the piscivore with little or no protection against lipid peroxidation in conjunction with their naturally high–PUFA diet.

The presence of the cyanobacteria Microcystis in algal blooms has been linked to the development of steatitis, resulting in die–offs of great blue herons in the Chesapeake Bay, where in the fall of 2001 several great blue herons were diagnosed with steatitis (Rattner et al 2006). These birds presented with emaciation, lethargy, inability to fly, and an unusually hard abdomen. Concurrent to when the birds were diagnosed, nearby waters were found to contain large blooms of Microcystis, a cyanobacterium known to produce the hepatotoxin microcystin (MC). MC is known to cause oxidative stress to animal tissues including the liver (Campos and Vasconcelos 2010; Guzman and Solter 1999) and has been shown to be harmful to fish, mammals, and birds through skin contact, ingestion, and inhalation (Driscoll et al 2004). Tissue samples from the affected birds showed evidence of toxic levels of microcystin. Additionally, over the last 10 years, isolated cases of young great blue herons and black–crowned night herons with steatitis have been presented to Tufts Wildlife Clinic and to rehabilitators in the northeastern US, but no large–scale mortalities have been reported.

In August and October of 2008, over 70 egrets and herons also were found sick or dead in Japan (Neagari et al 2011). Weakness, lethargy, and inability to fly were reported prior to death, and significant amounts of firm subcutaneous and body cavity fat composed of necrotic adipose tissues and low levels of vitamin E were reported postmortem (Neagari et al 2011). While the reservoir where the birds were retrieved was found to exhibit high levels of cyanobacteria concurrent with the steatitis outbreak, no microcystin was detected in the reservoir water or from the livers of the egrets (Neagari et al 2011). However, the birds were observed feeding on Japanese anchovies from a nearby fish farm rather than feeding at the reservoir. Because certain types of farmed fish can carry a wide range of contaminants and toxins (Hastein et al 2006), further investigation into this observation may be of interest.

Finally, it has been suggested that steatitis may be to blame for a large number of dead and sick herons observed in 2007 at Massinger Dam, Mozambique (Myburgh 2009). While no necropsies were performed to conclusively diagnose steatitis in these birds, steatitis has been observed in crocodile and fish populations in the region, in concurrence with an observed decrease in heron populations, and in the presence of aquatic pollution and cyanobacteria (Myburgh 2009; Huchzermeyer et al 2011).

While the link between pollutants, cyanobacteria, and steatitis in birds is not well understood, it is known that vitamin E plays a role in protection from liver toxicity in mice (Gehringer et al 2003), rats (Sokol et al 1996), and tilapia (Prieto et al 2009), and that antioxidant depletion and subsequent lipid peroxidation in bivalves can be utilized as biomarkers for toxicity in contaminated aquatic ecosystems (Cossu et al 1997). It is plausible that factors such as pollution and microcystin deplete vitamin E stores, predisposing piscivorous birds to an increased risk of steatitis.

Discussion

To date, nearly all cases of steatitis in piscivores in the US have been reported from marine coastal areas, but to the authors' knowledge, none have been reported from inland lakes and rivers. It is impossible to know how many steatitis occurrences remain undocumented, as recognition and reporting of the disease may be low. As with many wildlife issues, wildlife rehabilitators represent an important resource for detection and documentation of steatitis in herons and other species. Although many rehabilitators do not have the resources to make definitive laboratory diagnoses, the clinical signs are fairly distinctive and a presumptive diagnosis can be made by anyone familiar with heron physical examinations.

Rehabilitators can recognize if the bird they have received may have steatitis by being aware of several factors. Most often steatitis cases are the young of the year and present as 'down and weak.' On physical exam, these birds are usually of good body weight, but when the abdomen and area over the caudal keel of the sternum are palpated, the abdomen appears swollen, firm, and 'lumpy–bumpy.' By wetting the ventral feathers with alcohol or soapy water, lumpy, yellowish

masses are usually apparent under the skin. Please note there are other conditions that can cause lumpy, swollen abdomens, including tumors and parasitic visceral larval migrans. If radiographs (x–rays) are available, the abdominal area often appears very dense, much whiter than expected.

The authors strongly encourage rehabilitators and biologists who handle waterbirds to look for this condition, document it via photographs and written records, work with their local veterinarians to preserve clinical samples and tissues for histopathology, and notify state and federal agencies of the findings. Only in this way are we able to document the extent of the problem, determine if (and how) it is spreading, and establish what may be the causes of steatitis in wild fish–eating birds.

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