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Source: Journal of Wildlife Diseases, 11(1) : 8-12

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-11.1.8

MORTALITY FACTORS IN WHISTLING SWANS AT LAKE ST. CLAIR, ONTARIO[1]

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Abstract: Post mortem examinations of 31 whistling swans (*Olor columbianus*) collected from the Lake St. Clair marshes in the spring of 1972 indicated that lead toxicosis and filariasis (*Sarconema eurycerca*) were the main causes of mortality.

INTRODUCTION

Whistling swans breed across the coastal Arctic region of North America from Baffin Island to Alaska and winter in two separate groups; the eastern division winters along the Atlantic coast of the United States, principally in Chesapeake Bay, and the western division winters in California.[9]

Mortality has been reported in both the eastern[1,2,12,13] and western divisions.[1,8,10] Causes of death have been attributed to trauma and avian tuberculosis,[8,13] coccidiosis and idiopathic impaction of the proventriculus,[10] botulism and aspergillosis,[8] filariasis,[2,8] and lead toxicosis.[1,5,8,10,12,13] The latter has been involved in the greatest losses.

Lake St. Clair is one of the stop-over points used by the swans in the first stage of their spring migration from their Atlantic wintering grounds.[9]

In the spring of 1972, a large number of the swans were observed in the marshes and fields east of Lake St. Clair in Ontario between Mitchell's Bay and the mouth of the Thames River. In April, weekly searches were made throughout this area for waterfowl with lead poisoning. Twenty-six dead and six moribund swans were collected.

CLINICAL FINDINGS

Two of the six moribund birds died shortly after arrival at the laboratory. The other four birds were suspected of having lead poisoning because they were asthenic atactic, emaciated and had green diarrhea. Radiographs showed that two of these swans had lead pellets in their gizzards. These pellets were removed using a stomach pump designed by the author. The four swans were treated intravenously with CaEDTA (calcium disodium ethylene diamine tetra-acetate) daily as described by Rosen and Bankowski.[10] Three of the birds (swans nos. 11, 16 and 31) remained anorectic and died within 5 days after initial treatment. The other bird regained its appetite and muscular strength on the first day after treatment and was released after 2 weeks. Since this bird did not have lead pellets in its gizzard, treatment was given on a presumptive diagnosis of lead poisoning based on asthenia, emaciation and green diarrhea.

PATHOLOGY

Diagnoses were based on necropsy, and histopathologic, microbiologic and toxicologic findings. At necropsy, selected tissues were fixed in neutral buffered 10% formalin for histopathological examination and liver tissue was frozen for

[1] This study was supported by the Canadian Wildlife Service.

lead analysis. The gizzard contents were washed and agitated in a flat pan in order to find lead pellets.

Histopathological examinations were made on the fixed tissues which were embedded in paraffin, sectioned at 5 μm, and stained with hematoxylin and eosin. Sections of kidney were also routinely stained by the Ziehl-Neelsen method for acid-fast intranuclear inclusion bodies, and some liver and spleen sections were stained by the Prussian-blue method to confirm the presence of iron pigments.[7] Tissues were cultured routinely for pathogenic organisms on blood agar and MacConkey's agar and incubated aerobically for 24 hours at 37.5 C. Lung tissue from one bird was cultured on Sabouraud's dextrose agar for fungi. Liver lead concentrations were determined by ashing 5 g of tissue in a low temperature Plasma Asher,[2] collecting the lead residue with 10 ml of 1.0 N nitric acid and measuring the lead content by atomic absorption spectrophotometry.[3]

The pathological agents found in the swans are outlined in Table 1. Complete examinations on 8 of the 31 swan carcasses were prevented by decomposition and freeze damage. Data collected on these birds were limited to body condition, the contents of the proventriculus and gizzard including lead pellets, and liver lead concentrations. Five of these swans (swans nos. 20, 25, 26, 27, 29) had liver lead concentrations higher than 10 ppm (Table 1), a level considered toxic in whistling swans.[13] Other pathological agents were not noted in these birds. Of the other 23 swans, a diagnosis of uncomplicated lead toxicosis was made on seven birds. Four swans had filariasis caused by *S. eurycerca,* and one had aspergillosis. Seven swans had combined lead toxicosis and filariasis and one had lead toxicosis, filariasis and a staphylococcus infection. The cause of death of two swans (Nos. 2 and 8) was not determined.

Nineteen of the 31 swans had lead shot in their gizzards (Table 1). The number of lead shot ranged from 1 to 53 pellets per bird and averaged 14 pellets per bird. These swans plus swans nos. 3 and 18 had liver lead concentrations higher than 10 ppm (Table 1). The liver lead concentrations in these toxic birds ranged from 11.6 ppm to 44.3 ppm and averaged 21.8 ppm. There was no correlation between the numbers of pellets in the gizzard and the concentrations of lead in liver tissue.

The gross lesions found in the lead-poisoned swans included emaciation, green-stained feathers around the vent, impaction of the proventriculus, hydropericardium, myocardial infarction, eroded gizzard lining, green-stained gizzard lining, gall bladder distended with bile, epicardial and endocardial hemorrhages, flaccid heart, and infarction of gizzard and skeletal muscle (Table 2). Microscopically, myocardial degeneration was found in 12 of the 16 toxic birds examined. Degenerative changes which may have been solely due to lead[4] were complicated by the presence of filaria in seven of these swans. Splenic and hepatic hemosiderosis occurred in 14 (89%) and 13 (81%) of these swans, respectively. Three (19%) had hepatic degeneration and another three had necrosis of renal tubules. Acid-fast intranuclear inclusion bodies in renal tubules, which have been reported in lead-poisoned mallards,[3,6] were not found.

The sole food item found in the preventriculi and gizzards of most of the swans was grass. Corn, bean sprouts, and other seeds were found much less frequently and usually in combination with grass. The seven proventricular impactions found in the lead-poisoned birds consisted of one of grass, two of grass and corn, one of corn, one of grass and seeds and one of bean sprouts.

Heartworms, *S. eurycerca*, were found grossly or histologically in the hearts of 12 of the 23 swans examined. These

[2] Plasma Asher, Model PDS-504, manufactured by the Trapelo Division of LFE Corporation, Walton, Mass.

[3] Beckman, Model 440 spectrophotometer.

TABLE 1. Liver lead concentrations and prevalence of ingested lead pellets, heartworms and microbial pathogens in whistling swans from Lake St. Clair.

Swan No.	Age	Sex	No. of Ingested Lead Pellets	Liver Lead (ppm wet weight)	Other Pathogens
1	—[2]	—	5	16.6	Heartworms
2	A	F	0	0.7	
3	A	F	0	30.1	
4	A	F	0	0.4	Heartworms
5	A	F	0	0.8	Heartworms
6	I	M	0	0.6	*Aspergillus fumigatus*
7	—	—	0	1.0	Heartworms
8	—	—	0	0.8	
9	A	M	8	—	
10	A	M	2	13.9	Heartworms
11	I	F	6	15.1	Heartworms
12	A	F	15	24.1	
13	—	—	32	18.1	
14[1]	—	—	0	1.0	
15[1]	—	—	0	0.4	
16	A	M	32	24.7	Heartworms
17	I	M	8	16.1	Heartworms
18	A	F	0	11.7	
19	I	M	18	14.9	
20[1]	I	F	3	34.1	
21	I	F	10	11.6	
22	I	M	16	23.6	Heartworms and *Staphylococcus* sp.
23	I	F	14	13.1	Heartworms
24	I	F	9	25.9	
25[1]	—	—	4	24.9	
26[1]	—	—	24	44.3	
27[1]	—	—	53	33.5	
28[1]	—	—	.0	0.5	
29[1]	—	—	1	17.1	
30	I	F	1	20.2	Heartworms
31	—	F	0	1.2	Heartworms

[1] Carcass in advanced stage of autolysis. [2] Data not available.

TABLE 2. Prevalence of gross lesions in lead-poisoned whistling swans.

Lesion	Number of birds Examined	Prevalence of Lesion Number	Percent
Emaciation	21	15	71
Green-stained vent	21	11	52
Proventricular impaction	21	7	33
Hydropericardium	16	11	69
Myocardial infarction	16	4	25
Epicardial and endocardial hemorrhages	16	2	13
Flaccid heart	16	1	6
Infarcts in skeletal and gizzard muscle	16	2	13
Eroded gizzard-lining	16	11	69
Green-stained gizzard-lining	16	16	100
Gall bladded distended with bile	16	8	50

included all five of the moribund birds. Grossly, white worms, ranging from 1 to 3 cm in length, were found in the epicardium and throughout the myocardium. Histologically, adult *Sarconema* were found in the myocardium with little or no inflammatory response adjacent to the parasite. Lesions associated with the parasite such as necrotic tracts, focal hemorrhages, epicardial and endocardial fibrosis and widespread inflammatory and degenerative changes throughout the myocardium[5] were commonly found.

Staphylococci sp. were isolated from the liver and spleen of one bird which had concurrent filariasis and lead toxicosis.

Aspergillus fumigatus was cultured from the lungs of one bird with extensive pneumomycosis and fibrinous pericarditis.

DISCUSSION

The poor response of three of the four sick swans to the CaEDTA treatment was possibly due to filariasis and in some cases to advanced lead toxicosis.

The pathological findings indicated that lead toxicosis was the main cause of death and that cardiac filariasis was a significant contributor to the losses of whistling swans from the Lake St. Clair area. To the author's knowledge, this is the first report of whistling swan mortality of this magnitude in the Lake St. Clair area. In April of 1971, the author found only two swan carcasses and observed two other swans which were unable to sustain flight. Relatively fewer swans were seen in the area at that time, but according to Robin and Gunn,[9] large numbers of swans were observed further north near Wallaceburg, Ontario. The mortality in 1972 may have resulted from greater numbers of birds occupying these more southerly marshes which have been heavily hunted for waterfowl for several years and where lead shot would be readily available. Robin and Gunn[9] estimated the average stop-over time for the swans at Lake St. Clair in 1971 was 4.52 days. There is insufficient information on the toxicity of lead in swans to estimate whether this time period might allow birds to ingest shot and become immobilized by lead toxicosis. Spring mortality of swans due to lead poisoning has been recorded from 1942 to 1964 in Wisconsin[12,13] which is northwest along the migration corridor.

Trainer and Hunt[13] felt that because of the diverse geographic pattern of losses, the sparse number of birds usually involved at individual sites, and the short duration of their stay (2-3 weeks) in Wisconsin, the swans might have acquired the lead shot prior to their arrival in that State.

Since little is known of the incidence of ingestion or the toxicity of ingested lead shot in whistling swans, radiographic and blood-lead surveys might be useful to determine the prevalence of ingested shot and of elevated lead levels in free-flying swans. A survey of this nature could perhaps be carried out in cooperation with the current whistling swan research program.[11]

Sarconema eurycerca infections have been reported in swans on their eastern wintering grounds.[2] It is likely that swans with heart damage caused by these worms would be at a disadvantage during migration.

Acknowledgements

I wish to thank Drs. R. G. Thomson and H. Carlson, Department of Pathology, Ontario Veterinary College, University of Guelph, and Dr. J. Pettit, Ontario Ministry of Agriculture and Food, for their assistance in evaluating the histopathological material. The technical assistance of Mr. Andy Chan is appreciated.

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Received for publication 1 August 1973