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Evaluation and treatment of lead toxicosis in rehabilitated avian species: 95 cases (2014–2023)

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Objective
To evaluate avian patients presented to a wildlife rehabilitation facility with confirmed lead toxicity for clinical signs associated with blood lead level groups, efficacy of subcutaneous chelation therapy with calcium disodium EDTA, and case outcome based on presenting blood lead levels.

Methods
A retrospective record review of 95 avian cases representing 19 species treated for lead toxicosis from a wildlife rehabilitation center in Wisconsin between 2014 and 2023 was conducted. Data were evaluated for presenting complaints, clinical signs, radiographic findings, chelation therapy protocol, clinical pathology data, and case outcome.

Results
A low lead level of < 20 µg/dL was the most commonly diagnosed level in avian patients. The presence of neurologic disease was found most commonly in birds with higher blood lead levels, whereas poor body condition, trauma, and anemia were most common in birds with low lead levels. Bald eagles (*Haliaeetus leucocephalus*) and tundra swans (*Cygnus columbianus*) were the most prevalent species presented. All methods of chelation therapy evaluated resulted in a reduction of blood lead levels. Patients with intake blood lead levels > 60 µg/dL were more likely to die or be euthanized while in care.

Conclusions
A bird with a blood lead level > 60 µg/dL is more likely to present with neurologic disease and have a poor case outcome. Diluted calcium disodium EDTA was clinically effective when administered SC.

Clinical Relevance
Initial blood lead level, in conjunction with species, clinical signs, and radiographic changes, can help guide clinical decision-making for avian patients with lead toxicosis.

Keywords: avian, lead, toxicity, chelation, wildlife

Lead is a pervasive and relatively common environmental contaminant without biological function.[1] Environmental lead contamination has caused documented toxicosis in wildlife, including avian, mammalian, amphibian, and reptile species.[2–8] Common sources of environmental lead include urban exposure (paint, fuel, mining, batteries), contaminated soil, tackle from fishing gear, and lead shot from hunting.[4,6,9] Avian scavenger species, including birds of prey, are particularly prone to lead toxicosis via incidental ingestion of spent ammunition in the form of fragmented lead shot left in the environment from hunted carcasses or in wounded animals that are unable to be collected by hunters.[4,7,9] Waterfowl ingest spent ammunition or lead tackle that is left behind by fishermen when it is mistaken for grit in water sediment.[4,9] Previously established avian blood lead toxicosis categories include background (blood lead levels are < 20 µg/dL), subclinical (20 to 60 µg/dL), clinical (> 60 µg/dL), and severe (> 100 µg/dL).[7,10]

The amount of lead absorbed by wild birds and subsequent systemic intoxication is highly variable depending on several factors, including the route of exposure, location, and size of the lead particles, as well as the acidity of the gastrointestinal (GI) tract.[7,10,11] Lead is absorbed in the small intestine, where it then enters systemic circulation. Approximately 6% of systemic lead is stored in soft tissues, including blood, muscle, parenchymal tissues, and

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the nervous system, and the remaining 94% is stored in bone.[11] There may be considerable individual and species variability of systemic effects from lead toxicosis, and the threshold for intoxication for some species may be lower than previously believed.[4,7,9,10] Bald eagles (*Haliaeetus leucocephalus*) have repeatedly been shown to be more sensitive to lead toxicity than other wild avian species, including red-tailed hawks (*Buteo jamaicensis*), swans (*Cygnus* spp), and turkey vultures (*Cathartes aura*).[4,7,10]

Commonly described clinical signs or systemic changes caused by lead toxicosis in birds can be divided into several categories, including neurologic, GI, and hematologic signs.[7,11] The duration of lead exposure can affect the level of toxicity seen in birds.[7,11] Those with acute lead toxicity are usually in good body condition but may have clinical signs such as hind limb paresis, depressed mentation, visual impairment, labored breathing, and bright green feces, with blood lead levels often severely elevated at > 100 µg/dL.[7,11] Wild birds with chronic lead toxicity are often in poor body condition, weak, anemic, and depressed, with blood lead levels usually between 60 and 100 µg/dL.[7,11]

Antemortem blood lead concentration testing can be accomplished using several different methods, with gold-standard methods encompassing graphite-furnace atomic absorption spectrometry and inductively coupled plasma mass spectrometry.[4,11,12] Antemortem testing can also be performed on whole blood with the use of a portable point-of-care lead analyzer (LeadCare, LeadCare II; Magellan Diagnostics), which was originally created to assess humans for lead exposure but has been used to evaluate lead in wildlife in field and rehabilitation settings due to its convenience and low cost.[4,12,13]

There are several reported treatments for avian lead toxicosis, and treatment should be tailored to the individual patient on the basis of signalment, blood lead levels, lead location in the body, and severity of clinical signs.[11] Reported treatment methods include removal of metallic foreign bodies in the GI tract if present (via endoscopy, forced casting, or gastrotomy) and chelation therapy, which most commonly includes injectable calcium disodium EDTA (CaEDTA) and oral dimercaptosuccinic acid (DMSA).[7,9,11] Lead accumulation in bone and soft tissues can make treatment difficult and lead to fluctuating blood lead levels, as redistribution of tissue-stored lead has been noted following the initiation of chelation therapy with both CaEDTA and DMSA.[7,11] While other chelators are available, they are not commonly used in wildlife medicine and thus are not included in this study.

This retrospective study examined 95 wild avian cases across 19 native species that presented to a high-volume wildlife rehabilitation clinic in Wisconsin from 2014 to 2023 and were subsequently diagnosed with lead toxicosis on the basis of intake blood lead levels. This study aimed to evaluate lead intoxication in wild birds by examining species, blood lead levels, presenting clinical signs, radiologic and clinical pathology findings, treatment, and outcome.

Methods

Record review

Avian patient records from the Dane County Humane Society's Wildlife Center in Madison, Wisconsin, from January 2014 to August 2023, were reviewed. Records were kept electronically with the Wildlife Incident Log/Database and Online Network medical record (Wildlife Center of Virginia). The records of all avian patients that presented during the above time period were evaluated (n = 6,301). Inclusion criteria that warranted further record evaluation included avian patients that had blood lead testing performed with 1 blood lead level available prior to initiation of chelation therapy and subsequent treatment attempts with information on case outcome (released, died, or euthanized). Full records for avian patients that met the inclusion criteria were then reviewed for species, presenting complaint, clinical signs, diagnostics performed, metallic foreign body location (if applicable), blood lead level, treatments (specifically, type of chelation therapy, route administered, and dose), and clinical pathology data including PCV, RBC morphology and type, WBC count, WBC differential, and disposition status. Clinical signs at time of intake were assigned by licensed wildlife rehabilitators.

Blood lead level evaluation

Peripheral blood lead concentrations were evaluated immediately after obtaining venous whole blood with the use of a point-of-care analyzer (LeadCare or LeadCare II; Magellan Diagnostics). Blood lead levels were often verified with an alternative method if they were measured as high by the point-of-care lead analyzer, which indicates a blood lead level > 65 µg/dL. High blood lead level samples were sent to commercial laboratories for verification of blood lead level using graphite-furnace atomic absorption spectrometry (Wisconsin State Laboratory of Hygiene and Marshfield Labs). Out of 38 samples measuring high, 15 samples were not verified by a commercial laboratory. Samples were not submitted if a patient died or was euthanized prior to the sample being shipped to the outside laboratory. Categorization of avian blood lead levels for this manuscript was extrapolated from previously established blood lead level categories; however, different names were assigned to these groups to reflect that no amount of blood lead is normal and any amount can result in clinical signs. These groups included the following: low (< 20 µg/dL), moderate (20 to 60 µg/dL), and high (> 60 µg/dL).[7,10] Because not all high levels were verified, the severe (> 100 µg/dL) blood lead level category was not included in the final categorization. A point-of-care analyzer was not available for 13 months during the study period, so blood samples obtained during this time were sent to the Wisconsin State Lab of Hygiene, Marshfield Labs, or Idexx (Idexx Laboratories Inc) to have blood lead levels evaluated when lead toxicosis was suspected.

Presenting clinical signs

Presenting clinical signs in birds diagnosed with lead toxicosis were divided into several categories, including neurologic signs, respiratory signs, GI signs, trauma, and poor body condition. Neurologic signs included ataxia, paresis, stargazing, head tilt, tremors, recumbency, nystagmus, and depressed mentation. Respiratory signs used for analysis included respiratory distress, dyspnea, open-mouthed breathing, wheezing, or abnormal pulmonary auscultation. Gastrointestinal clinical signs used for analysis include regurgitation, diarrhea, full crop with poor GI motility, bright green feces, anorexia, hyporexia, and inappetence. Trauma included any mention of fractures, ocular injuries, abrasions, bleeding, fistulas, wing hike or droop, laceration, and limping. Poor body condition was defined as having a body condition score of ≤ 4/9 or having the intake evaluator use the term *thin* or *poor body condition score* to describe the patient.[14] Miscellaneous clinical signs included other problems that did not fit into one of the above categories, including identification of hemoparasites or GI parasites and dehydration.

Lead toxicosis treatment

Treatment for lead intoxication was initiated if blood lead levels were > 10 µg/dL per institutional protocol. A total of 10 patients with blood lead levels < 10 µg/dL were treated with chelation therapy due to concurrent clinical signs suggestive of lead toxicosis (n = 9) or having metallic fragments in their GI tract (1). Single-agent therapy with DMSA (25 to 35 mg/kg, PO, q 12 h) was initiated when blood lead levels were between 10 and 20 µg/dL and the GI tract was functioning. Injectable CaEDTA (100 mg/kg, IM or SC, once, then 50 mg/kg, IM or SC, q 12 h) was initiated when blood lead levels were > 20 µg/dL. When given SC, CaEDTA was administered in a fluid pocket if the patient was receiving SC fluids (lactated Ringer solution [LRS]); otherwise, it was diluted 1:10 in a syringe with LRS prior to administration. A combination of oral DMSA and injectable CaEDTA was utilized when blood lead levels were > 65 µg/dL. All patients were treated with one of the above chelation therapy protocols from 4 to 17 days, then a 48-hour washout period was observed and blood lead levels were rechecked. Chelation therapy was continued until blood lead levels were reduced to < 10 µg/dL or until the patient died or was euthanized. Chelation therapy was discontinued when a patient's blood lead level was < 10 µg/dL. After cessation of chelation therapy, blood lead levels were rechecked 7 days later, and, if needed, chelation therapy was resumed as above until blood lead levels were again < 10 µg/dL. If metallic fragments were noted in the GI tract, chelation therapy was continued until the fragments were removed or passed through naturally.

Hematology interpretation

All peripheral blood smears were reviewed by the same 3 licensed veterinary technicians throughout the study. Whole blood for PCV was obtained on the same day of presentation for all patients. A blood sample was immediately placed in a heparinized microhematocrit tube for centrifugation (1,534 X *g*; E8 centrifuge; LW Scientific) and analysis. Blood was also collected for CBC, differential, and cytology within 1 to 3 days of presentation and evaluated from a peripheral blood smear created immediately after venipuncture. Species-specific hematologic reference intervals were retrieved from published reference ranges or accessed through the Zoological Information Management System for all avian species evaluated.[15–22] There were 18 species and 91 individuals with bloodwork available for analysis; 4 individuals treated for elevated blood lead levels did not have bloodwork available for review. There were 15 of 18 species with available published hematologic reference intervals to assist in evaluation for potential hematologic effects of elevated blood lead levels. Packed cell volume data were available for 89 of 91 individuals; peripheral blood smear evaluation for RBC morphology and WBC differentials were evaluated in 80 of 91 and 66 of 91 individuals, respectively.

Statistical analysis

Descriptive statistics were used to evaluate intake blood lead levels and several variables including case outcome and presenting clinical signs such as neurologic disease, GI disease, respiratory abnormalities, and evidence of trauma. Prevalence was calculated by dividing the number of cases with a determined variable by the overall cases with that variable.

Results

Record review

A total of 6,301 avian patients presented to the wildlife center during the study period. Of these, there were 1,174 birds (18.6 %) from 56 species that were tested for blood lead concentrations on the basis of clinical signs at presentation. Ultimately, 95 birds (1.5%) from 19 species were subsequently treated for lead toxicosis. The most common presenting sign of avian patients treated for elevated blood lead levels was the inability to fly (66.3% [63 of 95]). The most common species treated for elevated blood lead levels were bald eagles (34.7% [33 of 95]), mallard ducks (*Anas platyrhynchos*; 11.6% [11 of 95]), and turkey vultures (8.4% [8 of 95]). Species prevalence based on total admissions during the study period demonstrated that lead toxicosis treatment was most common in tundra swans (*Cygnus columbianus*; 35.5 % [6 of 17]), bald eagles (28.9% [33 of 114]), and turkey vultures (13.1% [8 of 61]). Species that were frequently treated for lead toxicosis but were infrequently presented include the black vulture (*Coragyps atratus*; 100% [1 of 1]), domestic goose (100% [1 of 1]), trumpeter swan (*Cygnus buccinator*; 40% [2 of 5]), common loon (*Gavia immer*; 20% [1 of 5]), and hybrid duck (20% [1 of 5]; **Table 1**). A low blood lead level was the most commonly diagnosed category across all patients evaluated (43.2% [41 of 95]), followed by high (40% [38 of 95]), then moderate (16.8% [16 of 95]). Avian patients that were eventually released were most commonly diagnosed with low lead levels on intake examination

Table 1—Avian species presenting to a wildlife rehabilitation clinic from 2014 to 2023 that were tested and subsequently treated for lead toxicosis (n = 95).

Species	Total No. (%) treated for lead toxicosis	Species prevalence (%)	Intake blood lead level range (μg/dL)
Bald eagle (*Haliaeetus leucocephalus*)	33 (34.7)	33/114 (28.9)	5 to > 2,400
Mallard duck (*Anas platyrhynchos*)	11 (11.6)	11/2,061 (0.53)	9.1 to 512
Turkey vulture (*Cathartes aura*)	8 (8.4)	8/61 (13.1)	9.8 to 1,692
Canada goose (*Branta canadensis*)	7 (7.4)	7/371 (1.9)	< 2 to > 65
Red-tailed hawk (*Buteo jamaicensis*)	7 (7.4)	7/540 (1.3)	10 to > 65
Tundra swan (*Cygnus columbianus*)	6 (6.3)	6/17 (35.5)	> 65 to 1,111
American crow (*Corvus brachyrhynchos*)	4 (4.2)	4/376 (1.1)	5.6 to 14.7
Ring-billed gull (*Larus delawarensis*)	3 (3.2)	3/94 (3.2)	12.5, 16.7, 19.3
Sandhill crane (*Grus canadensis*)	3 (3.2)	3/131 (2.3)	3.8, 13.9, > 65
Eastern screech owl (*Megascops asio*)	2 (2.1)	2/124 (1.6)	17.1, 21.5
Great horned owl (*Bubo virginianus*)	2 (2.1)	2/418 (0.48)	9.6, 11.3
Trumpeter swan (*Cygnus buccinator*)	2 (2.1)	2/4 (50)	17.4, > 65
Black vulture (*Coragyps atratus*)	1 (1.1)	1/1 (100)	217
Blue jay (*Cyanocitta cristata*)	1 (1.1)	1/163 (0.61)	18.2
Common loon (*Gavia immer*)	1 (1.1)	1/5 (20)	16.5
Common merganser (*Mergus merganser*)	1 (1.1)	1/6 (16.7)	380
Domestic goose	1 (1.1)	1/1 (100)	10.8
Hybrid duck	1 (1.1)	1/5 (20)	10.5
Mourning dove (*Zenaida macroura*)	1 (1.1)	1/911 (0.12)	4.6

The total number of each species treated is listed, with the percentage of the total in parentheses, as well as prevalence during the study period for each species. The intake blood lead level range includes single values or values separated by commas for species having 1, 2, or 3 representatives; species with > 3 representatives have a range of values. The highest known value is listed.

(60.7% [17 of 28]), whereas the majority of patients that died or were euthanized in care had high blood lead levels on intake (50.7% [33 of 65]).

Blood lead levels and clinical signs

Presenting clinical signs were found to vary on the basis of intake blood lead levels. The prevalence of neurologic abnormalities seen on intake examination in patients treated for lead toxicoses (56% [53 of 95]) was higher than the prevalence of GI (23% [22 of 95]) or respiratory (22% [21 of 95]) clinical signs. Patients presenting with neurologic signs were more likely to have high blood lead levels (45.3% [24 of 53]) compared to moderate (20.8% [11 of 53]) or low lead levels (34% [18 of 53]). Among patients presenting with traumatic injuries and poor body condition, it was most common for animals to have low blood lead levels (trauma, 49% [29 of 59]; poor body condition, 47% [22 of 47]). While respiratory clinical signs were overall uncommon in patients with elevated blood lead levels, the majority of avian patients with respiratory clinical signs had high blood lead levels (61.9% [13 of 21]) compared to moderate or low levels (both, 19% [4 of 21]). Overall, bright green feces were an uncommon clinical sign, seen in only 8 patients (8.4% [8 of 91]) representing 3 species: the bald eagle (n = 5), Canada goose (*Branta canadensis*; 2), and tundra swan (1). The majority of patients with green feces presented with high blood lead levels (75% [6 of 8]).

A metallic foreign body was identified on radiographs in 29% (27 of 93) of birds treated for lead toxicosis, with the majority (62.9% [17 of 27]) of these patients having a foreign body located in the GI tract compared to soft tissue structures such as the pectoral muscles or wings. One patient had metallic fragments in both the GI tract and soft tissue structures, and another patient did not have the location specified in the record. Two patients did not have radiographs taken. Patients with a high blood lead level were more likely to have metal in the GI tract (77.8% [14 of 18]), while patients with low blood lead levels were equally likely to have metal in soft tissues and the GI tract (both, 50% [4 of 8]). Waterfowl were the most common patients with metal identified in the GI tract (64.7% [11 of 17]), followed by birds of prey (17.6% [3 of 17]).

Hematology interpretation

Overall, 39.3% (35 of 89) of individuals with elevated blood lead levels were considered anemic on the basis of published species-specific hematologic reference intervals. Most anemic patients had a low blood lead level (54.3% [19 of 35]). Polychromasia was present in 27.1% (19 of 70) of patients with elevated blood lead levels. Interestingly, most patients with polychromasia were bald eagles (63.2% [12 of 19]), with only 2 of the eagles concurrently anemic **(Figure 1)**. Leukocytosis was seen in only 17.5% (10 of 57) of patients with elevated blood lead levels. Basophilic stippling of erythrocytes was noted in the peripheral blood smear of only 1 patient, a bald eagle with a blood lead level severely elevated at 1,884 μg/dL.

Lead toxicosis treatment and case outcome

Lead chelation therapy for birds in this study consisted of 1 of 3 treatment types: (1) CaEDTA only (32.6% [31 of 95]) administered with an IM or SC loading dose and subsequent primarily IM or SC maintenance doses; (2) a combination of CaEDTA IM or SC and DMSA PO (40.0% [38 of 95]); or (3) DMSA PO only (27.4% [26 of 95]). A single patient received a CaEDTA loading dose IV **(Supplementary Table S1)**.

Birds that were released following lead chelation therapy were most commonly diagnosed with low

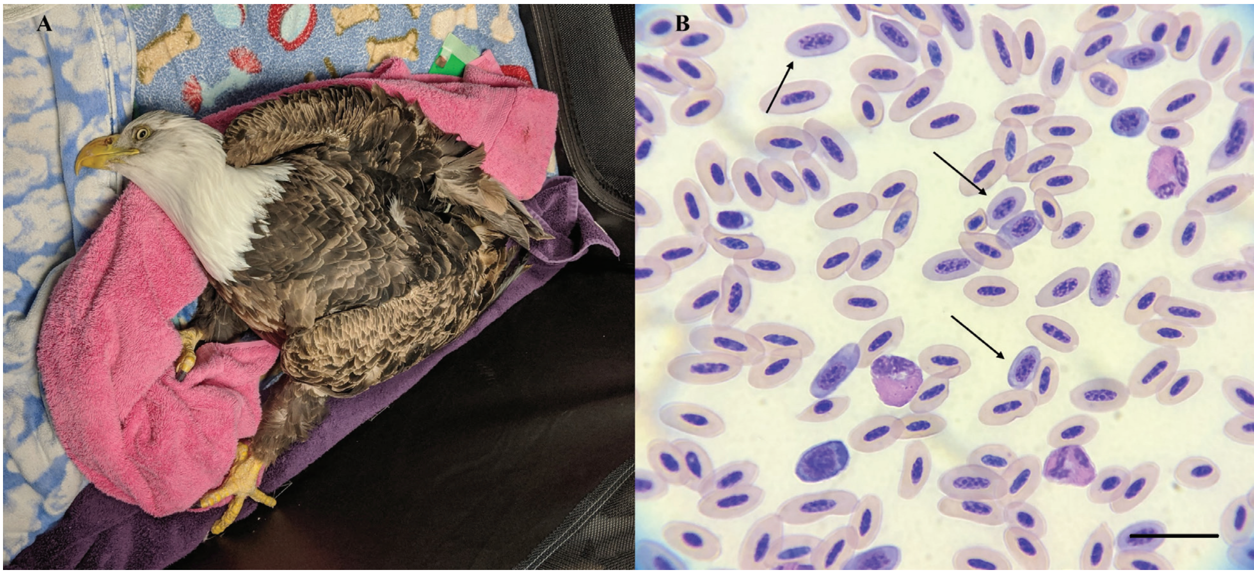

Figure 1—A—A bald eagle displaying neurologic signs due to lead toxicosis, including stargazing, opisthotonus, depressed mentation, and recumbency. B—A peripheral blood smear from a bald eagle with lead toxicosis displaying polychromasia (arrows). Diff-quik stain; bar = 20 µm. This eagle presented with a blood lead level of 328 µg/dL, which would be categorized as severe lead toxicosis.

lead levels on intake (37.5% [18 of 40]), while high blood lead levels had the highest percentage of natural death or euthanasia (62.2% [23 of 37] and 27.0% [10 of 37], respectively). Overall, 89.2% (33 of 37) of patients with a high blood lead level died or were euthanized in care **(Figure 2)**. Two patients that were initially transferred to another institution were not included in the final calculations; one patient is alive but still in a managed setting, and the other patient's final case outcome is unknown.

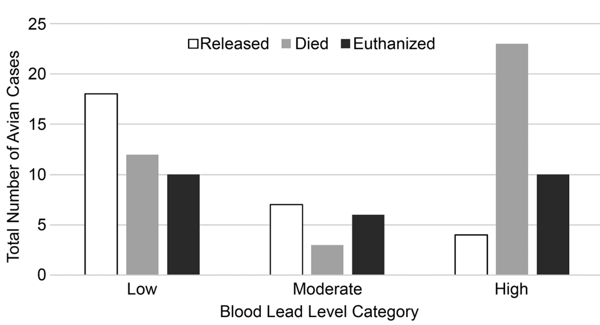

Figure 2—Case outcome for avian patients treated for lead toxicosis at a wildlife rehabilitation clinic in Wisconsin from 2014 to 2023. Blood lead levels on intake examination were categorized as follows: low (< 20 µg/dL), moderate (20 to 60 µg/dL), or high (> 60 µg/dL).

Discussion

The extent to which moderate and low blood lead levels affect overall patient health is difficult to quantify in a wildlife setting due to multiple patient comorbidities occurring at once.[9] In the cases evaluated here, traumatic injuries, poor body condition, and anemia found on intake examination were more likely to occur with low blood lead levels. This correlates with previous avian research, which showed that elevated, but subclinical, blood lead levels decreased fitness and led to a variety of illness and injuries.[9] Dehydration and other problems, including parasites (both GI and hemoparasites), hypoproteinemia, and hypoglycemia, were more common in the low blood lead level group, which may be indicative of chronic illness and immunosuppression in these patients.[7] When clinical signs such as abnormal mentation and bright green feces were present, they were more common in birds with a high blood lead level. In a small case series, green feces and respiratory changes were noted in 100% (3 of 3) of griffon vultures (*Gyps fulvus*) treated for lead intoxication.[23] Although bright green feces is a reported clinical sign of lead toxicity in birds, it was found uncommonly in this study.[11] Overall, the most commonly identified clinical signs for birds with elevated blood lead levels in this study were consistent with what has been previously reported, with the exception of GI signs, as these were overall uncommonly found.[7,11] In the cases presented, there was an overlap between clinical signs and blood lead level categories. Therefore, clinical signs noted in a patient may have been attributed to multiple comorbidities. Bald eagles were the most common species treated for lead toxicosis, which is further evidence that they are more susceptible to lead's toxic effects, which has been previously reported.[24] This increased susceptibility may be driven, at least in part, by their scavenging habits.[24]

In this evaluation, on intake examination, metallic foreign bodies were only present in 29% of avian patients with elevated blood lead levels. This is similar to previous reports,[13,25] which showed that 35.4% of raptors and 29% of bald eagles with elevated blood lead levels had metal in the GI tract identified on radiographs. When patients had elevated blood lead levels and a metallic foreign body on radiographs, the foreign body was more likely to be located in the GI tract, supporting previous studies[7,11] that found heavy metals in the GI tract contribute to higher blood lead levels. While lead fragments that are embedded in muscle do not generally appear to be broken down and absorbed in the same way, there have been rare reports of elevated blood lead levels associated with fragments in soft tissues, so if lead is present in soft tissues and no other source of exposure is identified, removal can be considered.[7,11,26] In the present study, lead fragments in the GI tract were most commonly found in waterfowl (64.7%), indicating that these species may be at higher risk for ingesting lead fragments. Overall, while it is important to take radiographs of each patient presenting for suspected lead toxicosis to help guide treatment and identify any other comorbidities, lack of metallic foreign body on radiographs should not rule out lead toxicosis as a differential, and blood lead levels should still be evaluated if there is a clinical suspicion for lead toxicosis.

In the present study, anemia was found most commonly in birds with low blood lead levels. This may be due to overall decreased fitness secondary to lead toxicosis, which has been previously reported; however, lead toxicosis as a primary cause of the anemia cannot be ruled out.[9] In 1 study[13] focusing on bald eagles, anemia was present in 66% of patients with a blood lead level > 100 µg/dL. In contrast, this study observed a lower percentage of anemic bald eagles (30%) with known blood levels > 100 µg/dL. In our study, bald eagles were more likely to have evidence of polychromasia in their peripheral blood smears than other species. This finding may have been due to lead toxicosis or patient comorbidities or could be a normal variation for bald eagles. Elevated WBCs, specifically heterophils, have been previously identified as a clinical pathology finding in avian patients with lead toxicosis. However, leukocytosis was present in only 1 patient with high blood lead levels in this study.[11,27,28] Basophilic stippling of RBCs is commonly described in mammals with lead toxicosis but is rare in avian species.[27] One study[29] suggests that it may only present with severely elevated blood lead levels above 300 µg/dL in trumpeter swans and Canada geese. Only 1 patient in the current study, a bald eagle with a blood lead level of 1,884 µg/dL, was found to have basophilic stippling, which is consistent with previous publications. This suggests that the presence of basophilic stippling should not be used as an initial parameter when assessing for lead toxicosis in avian patients.

Chelation methods used here included injectable CaEDTA, which is effective at chelating lead from bone and increasing urinary excretion, and oral DMSA, which is effective at chelating lead from the nervous system and other soft tissues.[7,11] In humans, CaEDTA is diluted in 5% dextrose or 0.9% NaCl to administer IV or undiluted to administer IM, with renal damage as a reported side effect.[30,31] In veterinary medicine, specifically cats and dogs, CaEDTA can be administered SC and is regularly diluted into 5% dextrose prior to administration.[32] Renal adverse effects of CaEDTA are not described in avian patients regardless of administration route.[7,11,25] Dimercaptosuccinic acid is administered enterally in avian patients due to adequate absorption in the GI tract.[7,11,16] The main adverse effect of DMSA reported in birds is regurgitation.[11] In this study, all methods and combinations of chelation were shown to successfully reduce blood lead levels. Therefore, specific chelation therapy, or a combination of chelation therapies, should be individualized to the patient. The manufacturer's drug administration instructions for CaEDTA state that it is incompatible with LRS when intended for IV use.[31] The clinical efficacy of CaEDTA was not affected in our patients when diluted in LRS for SC administration on the basis of all values decreasing on repeat blood lead levels. However, a controlled study would need to be conducted to substantiate this clinical observation.

The prognosis for successful lead toxicosis treatment was poor when patients had a blood lead level > 60 µg/dL, with a mortality rate of almost 90%. These findings are similar to those of a previous study,[26] which demonstrated an 11% release rate for clinically affected bald eagles as well as a correlation between elevated blood lead levels and negative rehabilitative outcomes (death or euthanasia). Patients that had a low or moderate blood lead level had an increased survival rate compared to those with a high blood lead level. This may be a helpful guideline for veterinarians and rehabilitators when choosing where to allocate resources for wildlife patients.

Limitations for this study reflected the large scope of data evaluated and the retrospective nature of case review. It is difficult to separate the characteristics and effects of lead toxicosis from other possible comorbidities in wild patients, which may confound mortality data. Additionally, measuring blood lead levels offers insight into acute exposure to lead, rather than long-term exposure, which may be another confounding aspect to these findings.[24] Given the nature of wild animals, it is likely that more animals are affected by lead toxicosis than are treated.[10,33] Finally, these data represent a single rehabilitation center in southern Wisconsin and may not be comparable to what is experienced by other rehabilitation facilities.

In conclusion, this manuscript outlines a robust retrospective investigation into the characteristics, treatment, and outcome of lead toxicity in wild birds. When patients had clinical neurologic changes such as lethargy, recumbency, or head tilt, they were more likely to have a high blood lead level > 60 µg/dL. This study demonstrated that various chelation methods can be used to effectively lower blood lead levels and that CaEDTA can be diluted and administered both IM and SC with equal efficacy. Overall, having a high blood lead level is a poor prognostic indicator for patients, with almost 90% of these patients eventually dying or being euthanized. Future directions for investigation may include how clinical signs and clinical pathology findings in various avian species correlate with specific blood lead levels and mortality, which will help clarify species-specific susceptibility to lead toxicosis. Data can also be evaluated for seasonal and geographic trends. Duration of chelation therapy in relation to various chelation protocols could also be evaluated. Clinical signs consistent with lead toxicosis, such as neurologic disease, were identified in all blood lead level groups in this study, and anemia was seen most frequently in the low blood lead level group. Given the overlap of these clinical signs between blood lead level groups, and to reflect that no amount of blood lead should be considered normal, recategorization of established avian blood lead groups to the categories outlined in this manuscript is encouraged. Overall, this information will further assist veterinarians and licensed wildlife rehabilitators in triaging and providing treatments and resources to avian patients that are presented to wildlife rehabilitation centers and have lead toxicosis.

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Supplementary Materials

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