Journal of Avian Medicine and Surgery 29(4):340–344, 2015

Lead Toxicosis in a Southern Ground Hornbill *Bucorvus leadbeateri* in South Africa

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Abstract: The southern ground hornbill (*Bucorvus leadbeateri*) has been classified as globally vulnerable and, in South Africa, regionally endangered, with a negative population trend. Factors contributing to the population decline in South Africa are poisoning, electrocution, and illegal capture for trade, coupled with slow reproductive rates and extensive habitat requirements. Lead toxicosis is a previously undescribed threat for the population. An adult southern ground hornbill presented with acute lead toxicosis due to lead particles in the gizzard, which required intensive treatment. Two other hornbills were likely exposed. The source of the lead in these cases was likely a carcass of a porcupine that was killed with lead shot. This report highlights the importance of the use of lead-free ammunition within the habitat of the southern ground hornbill in South Africa.

Key words: toxicosis, lead, endangered species, South Africa, avian, southern ground hornbill, *Bucorvus leadbeateri*

Clinical Report

A female southern ground hornbill (SGH) (*Bucorvus leadbeateri)* was presented to the Johannesburg Zoo Veterinary Hospital (JHBVH), Gauteng, South Africa, in September 2013. The bird had been captured from the Kruger National Park as a day-old chick and hand-reared as part of the reintroduction program of the Mabula Ground Hornbill Project. It had been soft released from an aviary into a 45 000–ha game farm near Thabazimbi, Limpopo Province, South Africa. The bird frequently visited the release aviary, where it received supplementary feed and where 2 additional male ground hornbills awaited release. The female had been free-ranging for 103 days.

The caretaker responsible for monitoring and caring for the birds reported that the female did not fly in to the feeding site as usual for supplementary feed but walked in. Three hours after the initial report, a relief team found the hornbill lying on its back with seizure-like activity and moved it to the JHBVH for treatment. On presentation, the bird was weak and severely dehydrated and had a mucopurulent upper respiratory discharge. Both lateral and ventrodorsal (Figs 1 and 2) whole body radiographs were taken. Three elongated radio-dense objects were seen in the gizzard. One measured 3.6 × 1.55 mm on the lateral view and 3.6 × 2.4 mm on the ventrodorsal view, and another 2 were only visible on the ventrodorsal view and measured 5.56 × 2.4 mm and 5.06 × 2.7 mm, respectively.

Examination of a blood smear showed an increased white blood cell (WBC) count of 40,900 cells/μL (40.9 × 10^9/L) (International Species Inventory System reference mean, 15 270 cells/μL [15.27 ± 7.52 × 10^9 cells /L])[1] with severe heterophilia and toxic changes. Marked immature red blood cells (RBCs) were visible, but no tear-shaped RBCs were observed. Results of plasma biochemical analysis showed increased concentrations of total protein, albumin, globulin, lactate dehydrogenase, and alanine aminotransferase (Table 1). Aspartate aminotransferase, gamma glutamyltransferase, bile acid, and uric acid concentrations were within the reference intervals. Blood zinc concentration was within the reference interval (100 μg/dL [15.3 μmol/L]) when compared with serum zinc levels of California condors (*Gymnogyps californianus*) (37–288 μg/dL [5.7–

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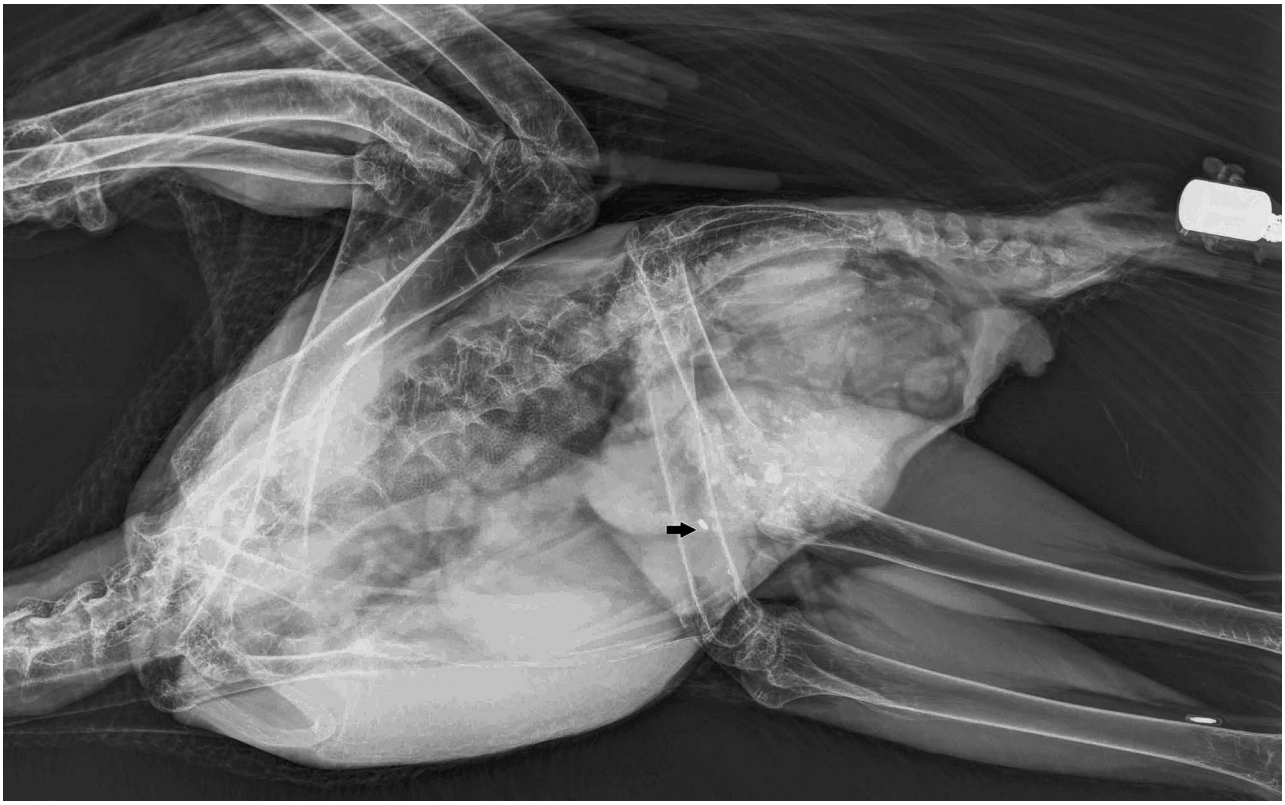

Figure 1. Lateral radiographic view of a southern ground hornbill, showing a radio-dense object in the gizzard (black arrow).

38.3 μmol/L]).[2] The blood lead concentration was very high (232 μg/dL) (Table 2).

The bird was treated with amoxicillin and clavulanic acid (120 mg/kg IM q24h for 5 days), amikacin (7 mg/kg IV q12h for 3 days), ketoconazole (10 mg/kg q24h PO for 15 days), lactated Ringer's solution (150 mL IV bolus q24h for 5 days), and vitamin E/selenium (0.06 mg/kg IM). Because of the radio-dense material in the gizzard, the hornbill was also treated with calcium EDTA (35 mg/kg IM q12h for 5 days).

After 5 days of chelation therapy with calcium EDTA, blood lead values decreased to 80 μg/dL and therapy was repeated after a 5-day interval of no treatment. The final blood lead level was 42 μg/dL, 17 days after initial presentation. The WBC count decreased to 23 740 cells/μL (23.74×10^9/L) after 5 days and 15 710 cells/μL (15.71×10^9/L) by day 17. Heterophilia also decreased by day 17. Positive response to chelating therapy further supported the diagnosis of lead toxicosis.

The remaining 2 hornbills in the aviary were subsequently tested for lead exposure. One (SGH 2) had high lead levels without clinical signs and was moved to the JHBVH for chelation therapy for 5 days administered at the same dosages as used in the first case. In the second bird (SGH 3), lead levels were not increased (Table 2).

A porcupine (*Hystrix africaeaustralis*) carcass that had been shot with a 12-gauge shotgun using one AAA cartridge (lead shot load of 32 pellets) was fed to all 3 birds 2 weeks before the first clinical signs had been observed. The porcupine had been shot in the head, which was removed before feeding. Hornbills both inside and outside the aviary were offered meat from this carcass. Because lead from lead shot can be present in deer and sheep carcasses as far as 45 cm from the wound site,[3] the porcupine carcass may have had lead fragments some distance along its length, given that the average adult porcupine has a total body length of 72 cm.[4] We concluded that as both free-ranging and aviary birds had high lead levels, the porcupine carcass was responsible for the lead poisoning in these cases.

Discussion

This is the first report of lead toxicosis in a SGH due to secondary ingestion of lead fragments. The SGH is considered vulnerable globally with a decreasing population.[5] In South Africa, the

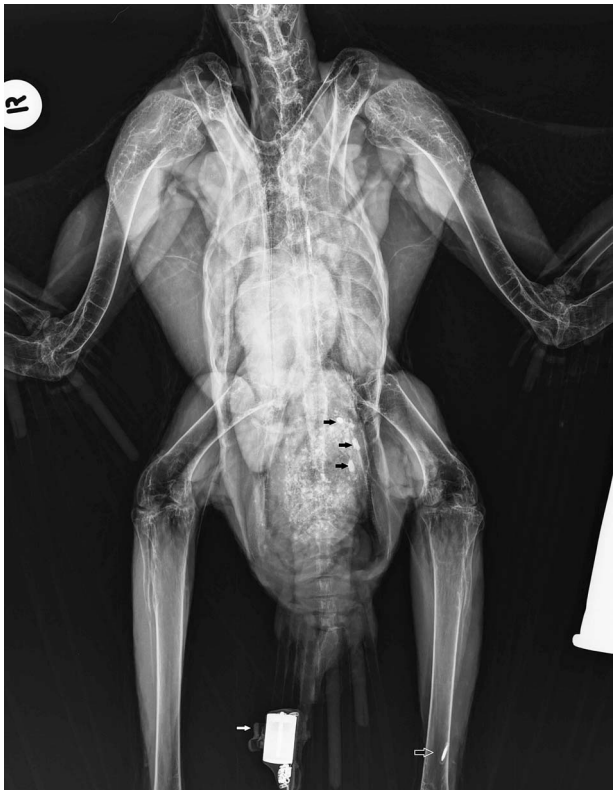

Figure 2. Ventrodorsal radiographic view of the southern ground hornbill described in Figure 1, showing 3 radio-dense objects in the gizzard (black arrows). Radio transmitter (white solid arrow) and microchip (white unfilled arrow) are also noted.

species has lost an estimated 66% of its former range, with an estimated 420 breeding groups remaining,[6] and therefore it has been reclassified as regionally endangered.[7] The SGH has a low breeding rate, with long-term data from the Kruger National Park showing that on average only 1 chick is fledged every 9 years.[8]

A vortex model from a population and habitat viability assessment predicted that a loss of just 4 individuals per year to unnatural mortality could further drive the population into decline.[9] Factors that contribute to unnatural mortality in SGHs in South Africa are poisoning, both primary and secondary; electrocution on transformer boxes; shooting; and illegal capture for trade (both for aviculture and traditional medicine).[10]

An undescribed threat is lead contamination of hunting offal from the use of lead-based ammunition. South Africa has a large and growing hunting industry, with an estimated 53 885 animals shot annually for trophy hunting alone. This occurs mostly on private land, with the most commonly hunted species being impala (*Aepyceros melampus*), warthog (*Phacochoerus aethiopicus*), and kudu (*Tragelaphus strepsiceros*), which all share habitat with the SGH.[11] The biltong (dried cured meat) industry alone, using the same species, contributes ZAR (South African rands) 6 billion to the gross domestic product,[12] although no estimate of the numbers of animals and species shot was provided. The remaining wild ground hornbill population is thus potentially at risk of eating lead contaminated carcasses if they have access to discarded offal left in the veld after a hunt or the carcasses of shot, maimed, and unclaimed animals that die in the veld. Southern ground hornbills feed by moving through their habitat as a group, looking for small prey to hunt. They will, however, also scavenge on carcasses and hence are susceptible to poisoning when scavenging on poisoned baits.[8,13]

Table 1. Plasma biochemical results in a southern ground hornbill with lead toxicosis, compared with International Species Inventory System values.[1]

Analyte	SGH 1	Mean ± SD
AST, U/L	523	324 ± 258
Albumin, g/L (mg/dL)	17.0 (1.7)	12 (1.2) ± 4 (0.4)
ALT, U/L	79	46 ± 22
Bile acid (serum), μmol/L (μg/mL)	21.8 (8.9)	17.5[a] (7.1)
Calcium, mmol/L (mg/dL)	2.27 (9.1)	2.13 (8.5) ± 0.43 (1.7)
CK, U/L	953	591 ± 457
Globulin, g/L (mg/dL)	28.7 (2.9)	17 (1.7) ± 4 (0.4)
GGT, U/L	23	15 ± 18
LDH, U/L	4707	1147 ± 764
Total protein, g/L (mg/dL)	45.7 (4.6)	25 (2.5) ± 5 (0.5)
Uric acid, μmol/L (mg/dL)	343 (5.8)	364 (6.1) ± 190 (3.2)

Abbreviations: SGH 1 indicates southern ground hornbill 1; AST, aspartate aminotransferase; ALT, alanine aminotransferase; CK, creatine kinase; GGT, γ-glutamyltransferase; LDH, lactate dehydrogenase.

[a] Mean value for California condors, age 6 months to 5 years.

Table 2. Blood lead levels in 3 southern ground hornbills with exposure to lead from a porcupine carcass before and after chelation therapy with calcium EDTA.

	Lead level, μg/dL		
Bird	Initial	After first treatment	After second treatment
SGH 1	232	80	42
SGH 2	80	10	N/A
SGH 3	4	N/A	N/A

Abbreviation: SGH indicates southern ground hornbill.

The remaining wild SGH population outside of formally protected areas is thus potentially at risk of eating lead-contaminated carcasses if access is not properly prevented, and SGHs are able to scavenge on lead-contaminated carcasses. Microscopic lead fragments may remain throughout the carcass even if the muscle tissue surrounding the wound is entirely removed.

Fatal lead toxicosis, resulting in death, has been reported extensively in California condors[14,15] and is the primary reason for the wild population being unsustainable without extensive management. Annual capture is required to monitor blood lead levels and administer chelating therapy if required.[16] Lead is the most common toxicosis found in free-ranging waterfowl, with approximately 2 million ducks dying annually due to lead toxicosis before the ban on use of lead shot was instated in North America and parts of Europe.[17] Onset of clinical signs subsequent to lead exposure depends on the number of pellets ingested as well as the fiber content of the diet. Clinical signs were observed 10 to 72 days after exposure in waterfowl.[17] Little work on the effects of lead in South African wildlife has been conducted but is reported for vultures, hence the provision of "safe" carcasses at vulture feeding sites.[18,19]

There are no published ranges for blood lead levels in the SGH. Lead levels between 15 and 59 μg/dL are associated with lead exposure in California condors, with levels above 60–70 μg/dL associated with clinical signs.[14,15] Values above 100 μg/dL are associated with acute toxicosis in California condors.[14] In Cape vultures (*Gyps coprotheres*), blood lead levels above 100 μg/dL are associated with clinical signs of toxicosis, while lower levels have been associated with poor hatchability of eggs and bone abnormalities in chicks.[20] Comparing the lead levels found in the second SGH (80 μg/dL) with the levels associated with clinical signs in condors, SGHs might be able to tolerate a higher blood lead level before showing clinical signs. More tests are needed to confirm this hypothesis. The chronic effects of subclinical lead exposure are currently not known, but reproduction may be affected, as seen in the Cape vulture. The blood smear showed an indication of lead toxicosis, with presence of increased immature RBCs,[21] but the classic tear-shaped RBCs were absent.

Lead toxicosis is of great concern for future releases of the SGH into parts of its historic range and for remaining wild populations in areas where hunting occurs. Upon release, the reintroduced birds are closely followed and monitored using tail-mounted telemetry devices. If the bird described here had not been conditioned to supplementary feeding and had not returned to the aviary at the time of the onset of seizures, it would have been vulnerable to predation if the lead did not kill it outright, as it was unable to fly. Recommendations for use of lead-free ammunition at release sites of the SGH have been made through custodianship agreements to reduce the risk and incidence of lead ingestion, and the Mabula Ground Hornbill Project is working on a national awareness campaign to reduce risk for this and other vulnerable species.

Acknowledgments: We thank Albert Mathukwi, Delecia Gunn, Rubin Els, Marthinus Prinsloo, Natasha Nienaber, Hein Nel, and the Johannesburg Zoo Veterinary Team for their role in bringing these particular birds back to the wild.

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