

THE TOXIC EFFECT OF INGESTED LEAD SHOT PELLETS BY CAPTIVE FALCONS

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Summary

Paper summarising clinical experience with treating lead poisoning in hunting falcons used in Middle East falconry. In years 1999 and 2001 eighty-five falcons suspected to be exposed to shotgun lead pellets were tested for lead poisoning. Tested falcons were saker falcons (*Falco cherrug*), peregrine falcons (*Falco peregrinus*), gyr falcons (*Falco rusticolus*) and their captive bred hybrids. Positive cases were confirmed by

- 1) Presence of radio dense heavy metal particles in the gastro-intestinal tract,
- 2) Elevated whole blood lead (Pb) level,
- 3) Inhibited delta-aminolevulinic acid dehydratase (ALAD) activity.

32% of all the tested falcons were negative on lead poisoning. 52% were sub clinical cases with elevated whole blood Pb level or decreased ALAD activity. 16% of the falcons had severe symptoms of lead poisoning. Radiogram only in seven falcons (8.5%) showed heavy metal particles in ventriculus. Very good treatment success was achieved with repeated (2-3 times) regime of 5% CaEDTA (Calciumedetate), ANIMALCARE (35 mg/kg TID or 50 mg/kg BID) for 5 days. In very severe cases when ALAD activity was 100% inhibited and blood Pb level exceeded 100 µl/l, 80-100 mg/kg EDTA i.v. with 20 ml Lactated Ringers solution for 5 days markedly reduced the blood Pb level and the clinical symptoms. The falcons fully recovered and performed very well as falconry birds.

Introduction

Lead poisoning is a well-known problem in different species of wild waterfowls, swans or scavenging - sanitary raptors like eagles and condors (KRAMER and REDIG, 1997). The most common sources of lead are ingested lead shots from shotgun cartridges used in game bird hunting. From the raptors the most affected are the sanitary species which ingest lead shot from the bodies of wounded or killed carcasses (SAITO, 1997). Lead poisoning in wild falcons is very rare and is exclusively related to a captive management. In the Middle East falconry is an ancient cultural heritage and a large number of falcons are kept in captivity. The usual food sources are farm bred pigeons, quails, day old chicks or rats. Some of the falconers believe that they can improve the performance ability by providing "better" quality food. This is a reason to feed falcons with shotgun shot birds. Falcons often regurgitate the lead shots with the daily cast together with the indigestible parts of the food (feathers, bones), but small sized pellets (1-2 mm) can stay a longer time (few days or even weeks) in the ventriculus, especially when casting material is absent from the diet. Sub clinical lead poisoning due to repeated exposure and ingestion of lead shots is a common veterinary problem; it reduces performance ability and affects the health of falcons used for falconry.

Toxic effect of lead

Lead is not a biogenic element. It is considered to be hemotoxic, neurotoxic, nephrotoxic and immunotoxic. In organisms lead inhibits different enzymes by its binding affinity to sulfhydryl (SH-) groups. From a clinical point of view main the diagnostic values are its hematotoxic abilities (SCHEUHAMMER, 1987). Lead causes microcytic hypo chromic anaemia's in chronic exposures. The following enzymes incorporated to the cascade of haemoglobin synthesis are inhibited (PAGLIUCA et al., 1990):

- 1) δ -ALAD (delta-aminolevulinic acid dehydratase),
- 2) Ferrochelatase,
- 3) Uroporphyrin and protoporphyrin dekarboxylase,
- 4) Pyrimidin-5-nukleotidase.

As the effect of lead increases, the activity of δ -ALAD is decreased and its substrate, ALA (aminolevulinic acid), concentration is increased in the blood. The enzyme ferrochelatase is also inhibited. Ferrochelatase is responsible for incorporation of iron molecule (Fe^{3+}) into porphyrin cycle. As result of this inhibition in the acute cases the concentration of Fe^{3+} and also free porphyrins increase in the blood.

Materials and methods

Tested falcons were presented for routine clinical examination with different complaints. Most often the owner noticed decreased performance, muscle weakness, reduced appetite or constant weight loss even during excess feeding. In the most severe cases greenish discoloration of the urine, crop stasis and seizures were presented. The CNS symptoms manifest usually after short-term exercise for only 0.5-2 minutes. The most typical symptom of acute lead poisoning with markedly increased blood Pb level was disvocalisation. The falcons during exhalation vocalised a “ka-ka-ka-ka” – like voice. Endoscopic examination of the trachea revealed a narrowed lumen of the syrinx due to decreased tension of the external tympanic membrane. In cases where radiologic examination showed metallic, radiodense particles in the ventriculus the falcon was anaesthetised with Isofurane and the particles were flushed out using \varnothing 5mm and 30 cm long gastric tube and 60 ml warm water. From all falcons with the aforementioned symptoms or anamnesis data confirming a possible lead shot ingestion 1.5-2.5 ml whole blood was collected from metatarsal vein. The blood tubes contained CaEDTA as an anticoagulant, because 90% of circulating lead is in the red blood cells. 0.5 ml unclothed blood was sufficient for blood Pb^{2+} detection using GFAAS (graphite furnace atomic absorption spectrometry) techniques. As additional tests CBC, RBC, Fe, Hb and δ -ALAD activity were detected. For the determination of ALAD activity, using the method of MODER (1983) 0.3 ml of whole blood was essential. This test is based on the reaction of haemolysed blood and aminolevulinic acid (ALA) as substrate. The product of the reaction is porfobilinogen, which quantity is detected by spectrophotometer. ALAD is a very unstable enzyme and fast detection of its activity is necessary. Pb^{2+} and ALA are very stable substances in the blood and samples can be stored for a long time. In cases positive for lead poisoning, treatment was initialised with intramuscularly injections of 10% CaEDTA, 35 mg/kg TID or 50 mg/kg BID for 5 days. If seizures or vocalisation was presented CaEDTA 80-100 mg/kg SID was used intravenously with 20 ml of Lactated Ringers solution for 5 days. At the same time, in all treated cases, daily 60 ml oral fluids and 0.2 ml/kg i.m. hemoplastic vitamin – amino acids injection Hemo 15, STERIVET Canada were administered as part of supportive therapy. 10-14 days after the 5 days treatment regime the patient was reevaluated. If blood Pb level was still high and ALAD activity did not increased markedly, the CaEDTA treatment regime was repeated.

Results and Discussion

Blood Pb level and activity of ALAD were used as indicators of lead poisoning as well as markers of efficiency of detoxifying treatment. In falcons with known history that were never exposed to lead, blood lead level was very low 0.5-2 µg/dl (0.005-0.02 ppm) and ALAD activities were in range 30-50U/dl. If lead exposure had occurred, ALAD activity was inhibited within 2-3 days. In the most severe cases activity were 100% inhibited (0 U/dl detected level) and correlated with 60 µg/dl Pb level and above. The highest detected blood levels were 308 µg/dl, 240µg/dl and 172 µg/dl. In our experience blood Pb²⁺ level did not correlate with the clinical symptoms of lead poisoning in all treated cases and did not reflect the whole body Pb²⁺ content. A single injection of CaEDTA can significantly reduce the blood Pb²⁺ levels. This decrease is temporary and Pb level will rise up again within few days. The inhibition of ALAD activity correlates with clinical symptoms. In our clinical experience the blood lead level did not correlate with the total amount of absorbed lead. 10-14 days after the termination of CaEDTA treatment the blood Pb level began rising up again. The reason is that CaEDTA chelates the lead only from the blood and lead is released from organs and interstitial tissue. As a good indicator of recovery is when ALAD activity rises above 20-25 U/l with it simultaneous blood lead level decrease. Other haematological indicators, such as Hb, RBC indices, show changes only after chronic, high dose exposure and are not suitable to monitor sub clinical patients. The absorbed lead circulates in the blood for about 25-30 days. Then it starts a slow deposition into the bones.

Clinical symptoms	0	+	++	+++
Pb (µg/dl)	0-5	5-15	15-40	40<
ALAD (U/dl)	50-30	30-10	10-2	2-0

Tab.: Relation of blood Pb²⁺ level and ALAD activity from the intensity of clinical symptoms.

In three cases after a long period of sub clinical lead exposure falcons started to show symptoms similar to lead poisoning (vocalisation, seizures, reduced ALAD activity). All three falcons had a history of being fed with mutton and goat meat, which are low in calcium. We expect that a long-term low calcium diet can induce calcium release from the bones and at the same time the release of Pb²⁺ deposits can cause recurrent lead poisoning. Repeated intravenous administration of CaEDTA even in higher dose (80-100 mg) did not have any nephrotoxic side effect, especially if sufficient rehydration was provided. If this treatment is repeated 2-3 times it is a very effective way to treat lead poisoning in falcons. The birds can fully recover and perform well. The parallel blood sampling for detecting blood Pb level and ALAD activity provides more exact prognostic information to evaluate the patient and monitor efficiency of the treatment. The exposure time and the total amount of absorbed lead are the factors responsible for sudden onset of clinical symptoms related to lead poisoning in falcons.

References

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