Clinical and Radiographic Findings of Heavy Metal Poisoning in Psittacine Birds

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Abstract

Objective- To evaluate the radiology application in diagnosis of heavy metal poisoning in birds.
Design- Descriptive study.
Animals- In this article 4 suspected cases of heavy metal poisoning, including 3 African Grey Parrots and a Sulfur Crested Cockatoo are described.
Procedures- Presumptive diagnosis of heavy metal poisoning was made based on history and clinical signs including severe progressive depression, anorexia, regurgitation, vomiting, etc. Radiographic signs like presence of metallic densities in gastrointestinal (GI) tract, proventricular dilation and delayed GI passage time besides responses to treatment, confirm the diagnosis. Treatment including supportive care, rehydration and chelation therapy by administration of CaEDTA and D-penicillamine was initiated immediately after primary diagnosis in three cases out of four which resulted in dramatic response.
Results- Based on our study, radiology can be a valuable tool for heavy metal poisoning diagnosis in exotic birds and it can be confirmed by dramatic response to the initiation of mentioned therapy.
Conclusion and Clinical Relevance- Heavy metal poisoning is one of the most important toxicoses among pet and aviary birds and is commonly encountered in psittacine birds because of their curiosity and special behavior in biting and chewing inanimate objects. Results of this study can be useful for avian clinicians.
Key Words- Heavy Metal Poisoning, Psittacine, Chelation Therapy.

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Introduction

Heavy metal poisoning, commonly lead and zinc are one of the most important toxicoses among pet and aviary birds particularly in psittacine birds due to their curiosity and special behavior in biting and chewing inanimate objects. There are many sources of lead and other heavy metals like zinc, copper, and mercury around birds in captivity (different kinds of toys, metallic pieces of cages and aviaries, weights, bells with lead clappers, batteries, lead pellets, lead-based paints, lead-free paints with lead drying agents, hardware cloth, galvanized wire, drink bottle foils, linoleum, costume jewelry, stained glasses etc.). Considering this availability, metal poisoning is one of the main toxicoses in birds.  

Materials and Methods

An adult African Grey parrot (*Psittacus erithacus*) (case 1) with unspecified sex was presented with anorexia, depression, ataxia, dehydration, feather fluffing, and one week history of being off-fed. The client remarked that the bird chewed wires. No evidence of regurgitation and vomiting was observed by the owner. In physical examination the bird was extremely apathic and urate discoloration was obvious. Ventrodorsal (VD) and lateral (LL) whole body survey radiographs were taken.  
A circa 6-year-old Sulfur Crested Cockatoo (*Cacatua galerita*) (case 2), exhibiting anorexia, lethargy, depression and chronic intense regurgitation immediately or shortly after feeding was presented. The bird had a history of chronic vomiting once or twice weekly, in contrast to the severe green biliary vomits lately. Oral fluid therapy for the last 2 days had no therapeutic effect. The bird was freely roaming in the home and its regular diet consisted of sunflower seed, fresh fruit and vegetables. In physical examination, lack of aggressive and defensive reaction, apathy, 10% dehydration and lucid mucus vomitus were noticed. The vomitus was examined directly and after centrifugation with Giemsa and gram stains. Slight polyuria and urate discoloration to yellow/tan color were salient. A blood sample was submitted for Complete Blood Count (CBC). To over rule obstruction, plain VD and LL radiograph was followed by 20 ml/kg body weight Barium Sulfate (30%w/v) administration for contrast radiography. Normal transit time for barium sulfate administered by crop gavage through stomach of medium to large size psittacine is 10-30 minutes. Intravenous rehydration in tibiotarsal bone with 5 ml isotonic sterile saline/glucose solution was performed. As heavy metal poisoning was suspected 0.5 ml intramuscular injection of Calcium Disodium Ethylenediamine tetraacetate (CaEDTA) (50mg/kg, IM, SID) for 7 days with the purpose of chelating heavy metals in blood stream and bones. Besides D-penicillamine capsule content (50 mg/kg, SID) was fed by crop gavage for 10 days to inhibit GI absorption along with 5 ml liquid paraffin to ease excretion of metal particles by defecation. 
An adult African Grey parrot (case 3) was presented with adipsia, one week anorexia, depression, dehydration, polyuria, diarrhea, severe lucid mucus regurgitation and sometimes bile stained vomitus. The client remarked that the bird had chewed and eaten a metal cage toy bell 2 days prior to the clinical signs (Fig 1). In physical examination despite being depressed, the bird was found to react sharply to stimulus and vocalize weakly. VD and LL bird standard view radiographs were taken. Based on the history and clinical findings heavy metal poisoning was the presumptive diagnosis and treatment was performed by intramuscular injection of 50 mg/kg CaEDTA once daily and D-penicillamine with liquid paraffin.
An adult Grey Parrot (case 4) with signs of severe regurgitation, emesis, progressive anorexia and depression was presented. It had a history of eating and chewing metal rings and chain which were used as toys in the cage. Ventrodorsal and Laterolateral bird standard view radiographs were taken. Treatment was initiated with CaEDTA, D-penicillamine, SC administration of Saline/dextrose and B complex injection.

Figure 1. Left Lateral (LL) plain radiograph of a gray parrot with history of chewing metallic wires reveals several small metal dense particles in gizzard and probably intestines.

Results

The VD and LL whole body radiographs of the case 1 revealed several 1-2 mm diameter radiopaque objects in gizzard (Fig 2). Unfortunately the case died before any therapeutic measures could be taken. The client did not permit us to perform necropsy. Metal dense particles were visible in the gizzard of the case 2. Gaseous impaction caused light proventricular dilation (Fig 3). Radiographs evinced movement of these dense particles cranially in the gastrointestinal (GI) tract, immediately after regurgitation.

Figure 2. Lateral radiograph of the cockatoo. In proventriculus (p) gaseous impaction (white arrow), and spleen (s) are clearly visible. Many radioopaque particles are seen in the gizzard.

Figure 3. Ventrodorsal (VD) radiograph of the cockatoo: Radioopaque objects (white arrows) with metal density are visible in gizzard and mild dilated proventriculus.
Figure 4. A metallic bell which was partly chewed by the gray parrot. The bird had 3 other similar bells in its cage which one of them was completely destroyed by the bird.

Figure 5. A piece of metallic ring which was used as toy in the patient cage.

Figure 6. Ventrodorsal radiograph of the grey parrot reveals many metallic particles in gizzard and severe proventricular dilation.

Figure 7. Lateral radiograph of the grey parrot reveals many metallic particles in gizzard and severe proventricular dilation.

Despite the sluggish passage and the barium sulfate surplus in proventriculus, contrast radiography ruled out any esophageal or GI obstruction after one hour. Microscopic examination of the vomitus material showed gram positive cocci which could not be the cause of clinical condition of the bird. Blood profile showed anemia (PCV=22.3%) and mild leukocytosis (20000/ml). No basophilic stippling was reported. However, there were a few toxic heterophils recorded. The bird improved obviously following the first injection of the CaEDTA and by the second injection, the patient's appetite returned to normal. Despite these positive responses to the treatment, the therapy was continued for other 5 days. In the day 3–4 after the treatment initiation,
urate color and defecation had normal appearance. One week later no radiodense objects were visible in plain radiographs (Fig 4). In contrast radiography with barium sulfate, the passage rate was regular. To suppress recurrences, D-penicillamine was fed for further 10 days. As administering this drug without intubations could induce regurgitation, alternatively CaEDTA was administered intramuscularly in alternate days. The bird recovered completely and found to be healthy in all respects in following months.

Radiopaque particle was visible in radiographs of case 3 and the response to the treatment was satisfying in the first and second days after initial therapy and the patient completely recovered after a week.

Radiographs of the case 4 revealed: metal dense objects in the gizzard and proventriculus. Severe proventricular dilation was also noticed (Figs 5 and 6). Presumptive diagnosis was made based on clinical signs and history. Two days after the initiation of treatment the bird health status improved so that the client asked for stopping the treatment. Eight days after initial treatment, another radiograph showed that metallic objects were still present, but proventricular dilation had been recovered completely (Fig 7). Oral liquid paraffin administration was continued for another 5 days. The bird was healthy two months later.

Discussion

As seen with these cases, history is suggestive in the diagnosis of heavy metal poisoning. Birds roaming freely in home, having no toys which may lead to chew different unsafe objects, availability of inappropriate toys e.g. metallic bell, and imbalanced nutrition as a cause of pica are guiding points in suspecting heavy metal toxicosis. It is highly recommended to bird owners to present some kind of safe toys like pine cone or pieces of wood for their birds to avoid any dangerous conditions. The dramatic improvement in the condition of the bird after initiation of CaEDTA therapy, suggested the radiodense particles could be lead. Poisoning by other heavy metals, poorly respond to CaEDTA. Once ingested, lead, zinc, and other heavy metals will remain in gizzard for some time. Lead and its relevant salts are partially insoluble and very little amount of these are absorbed by the GI tract and deposited in bones and soft tissues of the affected birds. Lead toxicosis causes neurological, hematological and gastrointestinal signs. Blindness, seizure, tremor, paresis, and depression are the neurological symptoms of lead intoxication. Vascular damage and endothelial cell necrosis in brain are the main cause of these signs. Wing drooping, leg paralysis, ataxia, blindness, head tilt, circling, head tremors, and seizures are central nervous signs of heavy metal poisoning. None of the cases were blind and neurological signs were not obvious in them except a ring-necked parakeet (Psittacula krameri), which is not mentioned here, with involuntary movement of the head (tic). The only neurological symptom was apathy because of the severe dehydration.

It is worthy to cite that lead intoxication is major cause of regurgitation and vomiting in adult cockatoos. Three out of these 4 cases revealed regurgitation of lucid mucus material. The progressive regurgitation worsened to bile stained vomitus. The bird wills to drink but after a while it will regurgitate. Diarrhea was encountered frequently and was not a determinant sign as the feces consistency changed from watery to solid during the day. Urate discoloration and billivedinuria was suggestive especially in the case 2 and 3.
Radiodense metallic objects distinct from gizzard contents and grit, leads to primary diagnosis.\(^\text{11}\) The opaque objects should be distinguished from grit in the gizzard as the grit reveals opacity equal to the bones and the metallic particles are usually denser than the bones. Visibility of metal dense particles with different shapes and size are the most suggestive signs. In encountered cases, the particles in the GI tract were small and no need for surgery aroused since oral administration of liquid paraffin and D-penicillamine led to fast defecation of the particles even sooner than what was expected in the two recovered cases.

Proventricular dilatation may be visible in the radiograph which is due to vagus paresis. It proves that gastrointestinal toxicity is adversely affected by the heavy metal toxicosis as it disappeared shortly after CaEDTA administration. It is advisable that in cases suspected to proventricular dilatation syndrome (PDS) without visible metallic objects, heavy metal poisoning should not be ruled out.

Anemia is due to effect of lead in reducing both hemoglobin production and Red Blood Cell (RBC) lifetime.\(^\text{9}\) Heamoglobinuria has been reported as a clinical sign of lead poisoning in Amazon and African Grey Parrot\(^\text{5}\), but it did not occur in our cases.

Confirming heavy metal poisoning is determinant after evaluating lead blood level; whole blood sample (0.5 ml) in a heparinized tube should be sent to toxico logical laboratory.\(^\text{4}\) Lead intoxication will be suggestive if lead level exceeds 0.2 ppm. In contrast, levels greater than 0.5 ppm are diagnostic.\(^\text{10}\) These thresholds vary minimally in relation to species.

One to three times, 0.25 - 0.75 ml of 50 mg/ml injectable solution of CaEDTA according to the bird's weight daily is recommended, which can be continued for up to 5 days in relation to the patient's response to the treatment.\(^\text{10}\) Two days respite is essential for preventing occurrence of renal toxicosis, and then the therapy could be resumed periodically for up to 14 days. Orally administered chelating agents like D-penicillamine prohibits lead GI absorption and can be combined with EDTA.\(^\text{3},\text{4},\text{10}\) Continuous usage of this drug up to 36 weeks, until the bird is asymptomatic may prove to be the best therapeutic regime for lead poisoning.\(^\text{10}\) Even when there is no radiographic sign, treatment with penicillamine should be continued for halting recurrence.

Meso-2, 3-dimercaptosuccinic acid (DMSA) is an orally administrable agent which may be easier for the owner to administer at home but the narrow margin of safety of DMSA comparing to CaEDTA indicates that this agent should be used with caution.\(^\text{12}\) Ascribed to unavailability of laboratory facilities for determining lead concentration or postponed results, and considering little adverse effects of chelating agents, chelation therapy based on the clinical findings, radiographic symptoms, and rapid response that is obtained if the diagnosis is correct, is highly recommended.

References


چکیده

یافته های بالینی و رادیوگرافیک مسمومیت با فلزات سنگین در طوطی سانان

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هدف- ارزیابی رادیوئوگرافی به عنوان وسیله ای برای تشخیص مسمومیت با فلزات سنگین در طوطی سانان.

طرح مطالعه- بررسی توصیفی.

حیوانات- در یک دوره یک ساله، تعداد زیادی برندگان مشکوک به مسمومیت با فلزات سنگین ارژاع شدند. در این مطالعه ۴ مورد شامل سه طوطی خاکستری و یک کاکاوتی تاج گوکرینگ مورد بررسی قرار گرفتند.

روش کار- بر اساس تاریخچه، علائم بالینی شامل افسردگی پیشینه، ای اشتهایی، تهوع، استفراغ و نیز با مشاهده دانه‌های فلزی در ناحیه رادیوگرافیک، تشخیص اولیه مسمومیت با فلزات سنگین داده شد. درمان حمایتی، رهیتراسیون و تجویز داروهای شلائیه کننده نظیر CaEDTA و D-پنتاسیلامین نظیر گرافت توصیف شده است.

نتایج- با توجه به این مطالعه، رادیوئوگرافی به عنوان وسیله ای کارآمد جهت تشخیص اولیه مسمومیت با فلزات سنگین در برندگان می‌تواند به لطفی قابل اعتماد پس از آن درمان نیز مورد تأیید قرار گیرد.

نتیجه‌گیری- گروه‌بندی بالینی - مسمومیت با فلزات سنگین یکی از مهم‌ترین مسمومیت‌ها در برندگان خنکی و زینتی می‌باشد.

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