

Zinc Toxicosis: Separating Fact from Fiction

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Introduction

Continually evolving changes and advances in avian veterinary medicine have led to changing and evolving diagnostic approaches towards laboratory testing the clinically normal as well as the obviously ill avian patient. These advances have come from controlled scientific studies, primarily from universities and from careful data collection and assessment by astute clinicians. Zinc toxicosis testing and diagnosis is included in this continually changing arena in avian medicine. As a general rule, avian practitioners must be particularly careful when using diagnostic tests, which are based primarily on testimonials rather than reproducible or validated data. Care is also needed when clinically interpreting laboratory test results. This discussion is focused on blood testing for zinc content and the interpretation and clinical application of those results.

Toxicity and Toxicosis

“Toxicity” is a characteristic of the toxin – i.e.: it’s relative ability to produce disease. “Toxicosis” is the clinical state or condition produced in a living entity by a toxin. Zinc toxicity is a characteristic of the metal, not a clinical condition. Zinc toxicosis refers to the clinical condition(s) caused by a toxin in a living entity. In some ways, the free interchange of these very different words predisposes us to clinically misinterpret our diagnostic test results, leading to a “high” blood zinc level being reported as “toxic”, and equal to a diagnosis of toxicosis. Even some laboratories will provide interpretive comments about reported blood zinc levels indicating “toxic” based on a set break-off level for clinical interpretation.

Zinc and Zinc Toxicosis

Zinc is an essential nutrient for avian species and is present in very low levels in the typical seed diet. Unpublished assays (Sigurdson-Scott, C) of Orange-winged Amazon parrots from a university colony yielded plasma zinc levels similar to those of poultry. These Amazons consumed 100% formulated diet (Roudybush). Conversely, cockatiels that were fed primarily a seed diet showed unmeasurable serum zinc levels. It is likely that some histologically supported zinc deficient dermatopathies occur in pet birds, but controlled studies have not been reported.

In some animals, plasma zinc levels are drastically altered by either dietary zinc or physiological status, and often provide a reflection of transitions in zinc metabolism in those individuals (12). In human studies, serum zinc levels have been shown to be controlled by a very effective homeostatic mechanism. The term “homeostatic control” means that various organs and associated physiological events act together to maintain constant conditions in the internal environment. With regard to zinc, the concept of homeostasis implies that absorption and excretion of this nutrient are regulated by a series of linked metabolic events. On a daily basis, it is difficult for animals with a varied diet to maintain a constant zinc supply. To minimize the effects of this variability, homeostatic mechanisms evoke changes in absorption, internal redistribution, and excretion, which help to ensure that a constant amount of zinc is available for distribution to various tissues. In man, with a tenfold increased zinc dietary intake, there is a correspondingly balanced tenfold increase in zinc elimination via feces and urine. Plasma zinc concentration can be drastically altered by either dietary zinc or physiologic status, and often provides a reflection of transitions in zinc metabolism. Plasma zinc concentration responds homeostatically to a dietary zinc load, in that elevations are transient and return to within normal limits quickly. However, zinc deficiency does not elicit homeostatic mobilization

of zinc stores to elevate plasma to within normal limits. Zinc uptake is known to occur by means of a high affinity system in animals, and serum zinc accumulation is increased by as much as 100% when glucocorticosteroid hormones are added to the medium(12). In man, it has been noted that serum zinc concentrations increased at certain stages of infectious disease processes. Furthermore, it was suggested that single collections of blood serum samples might be misleading and recommended that multiple samples be collected during the course of an infectious disease (12). The role of adrenal cortical steroids, concurrent disease, stress and their influence in serum zinc levels, at this point in time, are not known in nondomestic bird species. In clinically ill patients, many of the toxic effects attributed to zinc toxicosis may actually be due to other contaminating elements, such as Pb, Cd, or As (12). At this time, the interpretation of serum zinc test results in non-domestic bird species is still quite unclear. Although "normal" serum levels are becoming better understood for some adult pet bird species, the correlation of abnormal results with a clinical diagnosis of zinc toxicosis is far from clear. Some species, such as cockatoos, seem to have higher serum zinc levels than other pet bird species (9).

Excessive consumption of zinc can result in zinc toxicosis. Clinical signs can include anorexia, regurgitation, gastrointestinal stasis, polyuria, and somnolence. (1,2,3) Acute and chronic syndromes of zinc toxicosis have been described in cockatiels in one experimental study (4). In acute toxicosis, signs included lethargy, dullness (birds left perch and spent most of their time sitting on floor with feathers partly erect and eyes closed), shallow respiration, anorexia, dark green moist droppings, rapid weight loss, reluctance to move, recumbency, ataxia, and/or death. With acute toxicosis, mortality increased with increased levels of Zn consumed. In chronic toxicosis, birds showed variable, intermittent signs including lethargy/dullness, periodic dysphagia, and rapid weight loss. Many recovered spontaneously but a few in one study developed recumbency and ataxia and were euthanized (4).

In the Howard study (4), their normal group of clinically healthy cockatiels had blood zinc levels of 1.63 ppm +/- 1.44 SD. This means that those normal birds within one standard deviation were as high as 3.07 ppm. This is in direct contradiction of other authors' suggestion that levels of 2.0 ppm = "toxic", suggesting diagnostic merit for true toxicosis, sometimes regardless of clinical signs or species (3,13). Cockatiels that were gavage fed high doses of zinc had blood levels as high as 48.72ppm +/- 66.87. Although the blood zinc values varied greatly between birds, they were logarithmically related to dosage rate. Birds dosed with < 8 mg had minimal changes in blood zinc levels, and then equilibrated rapidly towards the pre-dosage levels that the bird had. The rapid equilibration towards pre-dosage blood levels is consistent with the zinc homeostatic mechanism that most vertebrate species have (12). Death was seen in some birds dosed with as low as 2 mg/week of particulate zinc. Blood zinc levels correlated poorly with the severity of clinical signs.

The Challenge of Diagnosis

The described clinical signs of zinc toxicosis are not specific. Hematologic changes associated with zinc toxicosis in avian species are mostly non-specific; anemia is present in some cases with no specific or predictable red blood cell morphologic changes. Anemia, however, is not specific to zinc toxicosis. Marked hyperamylasemia (Values greater than 1,000 IU/L) often occurs in clinical zinc toxicosis, presumably because the pancreas is frequently pathologically effected by toxicosis.(6,4,7,8) Hyperamylasemia, however, is not specific to zinc toxicosis. The ingestion of metal (household materials, hardware wire, pennies minted after 1983) will in many cases correlate with positive radiographic findings, (metallic densities visible in the gastrointestinal tract) however many metallic alimentary tract densities turn out to be ferrous and/or non-toxic. As such, radiographically identified metal in the gastrointestinal tract is not specific for zinc or heavy metal toxicosis, just as the absence of metallic densities is not specific for the absence of heavy metal toxicosis. Zinc toxicosis in a bird that is clinically ill often, but not always, results in significant elevations of plasma zinc levels (4,9). These levels, when reported, are often multiples of normal reference ranges (10,11). When this occurs, appropriate chelation therapy may be indicated plus endoscopic, aspiration or surgical removal of metal may also be necessary. Not all patients with zinc toxicosis, however, require specific chelation treatment. (4). Zinc is not stored within the body, and the proportion absorbed is thought to be inversely related to the amount ingested, suggesting that chronic exposure and toxicosis may not necessarily require chelation treatment (12).

Rubber stoppers on glass blood collection vials (Vacutainer- BD- Cockeysville, MD) contain zinc, and the use of these types of containers can result in false or misleading elevations in test results. Some zinc assays are invalidated by sample hemolysis, resulting in elevations in test results. Complete and rapid separation of avian plasma from whole blood is important to avoid this problem, which in turn, can result in incorrect interpretation of "elevated" levels that may be reported.

The Rise in Popularity of Blood Testing for Zinc

Over the past few years, there has been an increased awareness of the potential for zinc toxicosis among bird owners and practicing veterinarians. Requests for action (testing) from the public and veterinary recommendations to test and screen avian sera for zinc have increased with this rise in awareness. Some of these requests and recommendations for serum zinc testing have included virtually every patient as part of a well bird checkup, and virtually every sick bird in the absence of exposure history or supportive clinical signs of zinc toxicosis. Serum zinc testing has more frequently been recommended and included as a standard part of a diagnostic workup for various feather-damaging (picking) disorders (13). In reality, blood zinc test results that are greater than a specific "cut off value" most likely is being presumed to be diagnostic for toxicosis, and whatever clinical signs the patient has is then tied into the diagnosis of zinc poisoning. In reality, there remains to be a single peer-reviewed case report of feather damaging behavior in a bird associated with zinc toxicosis. This author would forward that in some cases, feather picking may be associated with "elevated" serum zinc levels, but may not at all clearly linked with clinical zinc toxicosis. Often, the inclusion of serum zinc testing in many medical workups occurs seemingly regardless of physical examination findings, history, and a prioritized differential diagnosis. It may be in part, that the relatively easy ability to obtain a "positive" test result and establish a diagnosis in some problem cases proved attractive enough to some clinicians to the point where other key clinical variables were downplayed if not ignored.

Concerns and Conclusions

The very real potential result of increased testing activity in clinical settings is that many avian patients may be being over-diagnosed with zinc toxicosis based on serum zinc levels, sometimes resulting in unnecessary, expensive, and even toxic therapies in some circumstances. Furthermore, incorrect diagnoses and therapies may result in delayed timeframes for proper diagnosis and treatment to be accomplished.

Careful selection of our diagnostic tools, and even more careful interpretation of serum zinc test results will be important in the proper recognition and treatment of true zinc toxicosis in nondomestic bird species.

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