Hypocalcemia Syndrome in African Greys:
a clinical update

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Two years ago we reported on the African Grey hypocalcemia syndrome as we have observed it over the years in clinical practice.[1] The purpose of this report is to update the members on recent findings and current recommendations for treatment.

THE SYNDROME

The syndrome has been most often noted in young African greys between the ages of two to five years. However, one case has been described in a ten year old bird. The birds are presented with a history of fainting spells or convulsions which are often stimulated by excitement. The clinical pathology workup may show a leukocytosis and other variable abnormalities. The one consistent finding is a blood calcium level of below 6.0 mg/dl (normal levels are 8.0 - 13.0 according to our data,[3] and around 10.0 according to Dr. Joel Murphy.[4]) Levels as low as 2.4 mg/dl have been described.[5]

Birds that do not survive consistently show enlarged parathyroid glands with severe parathyroid degeneration.[6,7] Pathologists at the University of Georgia describe the parathyroid degeneration as incredibly destructive. The only other consistent lesion seen in these birds is adrenal vacuolation, indicating active secretion and probable stress. Cortical bone sections examined by Dr. Joel Murphy while at the University of Georgia showed no calcium mobilization or thinning as would be expected in most hypocalcemic states.[8,9] This phenomenon is similar to what has been described in adult cattle.

CURRENT THEORIES ON ETIOLOGY

Dr. Joel Murphy, while at the University of Georgia, had an opportunity to study thirty necropsied African greys that had succumbed to the hypocalcemia syndrome. He feels that African greys *(Psittacus erithacus erithacus)* and Timneh greys *(Psittacus erithacus timneh)* may be unique in that they may not be able to mobilize skeletal calcium in response to low blood calcium, as is the case with adult cattle. He mentions that he has seen this in some Amazon parrots as well. We may have observed this phenomenon in some conures, also. Dr. Murphy is perplexed, as we all are, as to why not all African greys manifest this phenomenon. He postulates that there may be a virus involved, perhaps one that interferes with parathyroid function. Nothing has been found on extensive electron microscopic examinations, however. Another area he intends to investigate is African grey kidney function. It may be that whatever triggers this phenomenon may involve the kidney's release of calcium.

CURRENT RECOMMENDATIONS FOR EVALUATION AND TREATMENT

If African grey parrots are unique in their inability to mobilize skeletal calcium, then careful attention must be paid to dietary calcium supply. Other species will sacrifice all available calcium sources (i.e. skeleton, egg shells, etc.) to maintain normal blood calcium levels. In these species, pathologic fractures or soft eggs would be expected before signs of hypocalcemic tetany would occur. In African grey species, apparently no such "reserve" is available. Therefore, the first sign of severe dietary calcium deficiency would be expected to be seizure activity in African greys with calcium levels below 6.0 mg/dl. The diet must, therefore, be adequately supplied with calcium in all African grey species.

We recommend a periodic blood calcium test be run on all African greys and Timneh greys as part of their routine workup. Foods such as cheese or yogurt should be encouraged, along with the usual mineral blocks. A reduction in high fat seeds (i.e. sunflower seeds) will help prevent calcium binding and removal through the G.I. tract. Adequate vitamin D3 should be provided by supplementation as well (Avitron, Superpreen, etc.). Vita-Lites® may also aid in the absorption of dietary calcium. The use of water soluble forms of calcium such as Neocalglucon® or Avimin® are to be encouraged.

Treatment of an acute case may require 0.50cc/400g intravenous Calphosan® with a dosage of 0.50-1.0cc/400g b.i.d., Vits ADE (Injacom) with a dosage of .30cc/400g every three days, antibiotics, and fluids. Intramuscular Valium may be necessary in extreme cases (dosage - .05cc/400g) to prevent hyperthermia.

Initial maintenance (i.e. the first five to seven days) includes daily intramuscular calcium injections, antibiotics, and vitamins plus a careful evaluation of the diet. Long-term maintenance involves placing these birds on Neocalglucon 5-10cc/120cc drinking water plus a vitamin D3 source such as Avitron (20 drops/120cc drinking water) as a lifetime regimen.

PROGNOSIS

Prognosis depends on the degree of parathyroid degeneration that has occurred. If parathyroid failure occurs, then death obviously ensues. Periodic blood calcium evaluations must be performed and maintenance calcium therapy adjusted accordingly.

Dr. Murphy has not lost an acute case that he has treated and is following these cases to observe their long term outcome. We have several patients alive and asymptomatic after three years of the onset of their symptoms. Since the condition may be viral and parathyroid damage may have occurred by the time veterinary help has been sought, a guarded prognosis must be given, at this time, for long term longevity of these cases. If the condition is entirely dietary with the idiosyncracy of an inability to mobilize skeletal calcium, then the prognosis should be favorable in acute cases. Obviously, we need more knowledge about this perplexing problem!

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