

Renal Mineralization in a Red-Tailed Black Cockatoo (*Calyptrorhynchus banksii*)

Bob Doneley BVSc MACVSc (Avian Health)
West Toowoomba Vet Surgery
194 West Street, Toowoomba 4350

Summary.

Mineralization of the renal parenchyma is often visualised radiographically in the diagnostic investigation of psittacine birds with suspected renal disease. This report discusses the successful treatment of a cockatoo with renal mineralization and discusses the possible aetiologies and pathogenesis of this condition.

Introduction.

Pathological calcification is the abnormal deposition of calcium salts (usually calcium phosphate and calcium carbonate) and other ions, such as iron and magnesium, in tissues other than bone and teeth.^{2,3} Because these deposits are chemically impure some authors prefer the term mineralization.³

When the deposition occurs in non-viable or dying tissue it is known as *dystrophic calcification*. It occurs despite normal serum calcium levels, probably as a result of changes to the injured cell membranes. On the other hand, when it occurs in otherwise healthy tissue, usually in the presence of hypercalcaemia, the process is known as *metastatic calcification*.^{1,3,4}

Dystrophic calcification is seen at sites of scarring, haemorrhage and old infections.¹ It occurs in tissue injured by vascular, toxic, metabolic or inflammatory conditions. It is most prominent when a good blood supply is present in the injured tissue.⁴ This calcification may be one way in which the body disposes of dead tissue, since the calcified material is functionally inert.³

Although these calcium deposits are relatively permanent, they are usually harmless unless, by virtue of their location, they cause weakness or organ dysfunction (eg arteriosclerosis).^{2,3}

Metastatic calcification is commonly associated with hypercalcaemia or disturbances of calcium metabolism.^{2,3,4} Conditions that have been associated with it include:

- a) primary hyperparathyroidism;
- b) renal failure, with decreased phosphate excretion leading to secondary hyperparathyroidism;
- c) hypervitaminosis D₃;
- d) increased bone metabolism (eg neoplasia);
- e) calcinogenic plant toxins (*Solanum*, *Cestrum*, *Trisetum*);
- f) calcinogenic bacteria; and
- g) Excessive dietary calcium.^{1,2,3,4,5}

Metastatic calcification may occur widely throughout the body but for unknown reasons it principally affects the interstitial tissues of the blood vessels, kidneys, lungs, and gastric mucosa.^{2,4}

The distinction between dystrophic and metastatic calcification, although useful for classification, may be somewhat artificial. Metastatic calcification, through the toxic effect of calcium ions on the cell membrane, can cause tissue injury and death, making the distinction between dystrophic and metastatic calcification difficult.⁴

Case Report.

An immature female Red-Tail Black Cockatoo (*Calyptorhynchus banksii*) was presented for chronic weight loss and polyuria of several months' duration. The bird was being reared on a commercial hand rearing diet (Lake's Hand-Rearing Formula) with no vitamin/mineral supplementation. Although the bird was being fed adequate amounts of food three times daily the owner, in retrospect, felt he might have erred in not offering drinking water earlier.

The bird was depressed, anorexic, polydipsic and polyuric. On physical examination it was underweight (500g) and had a flaccid, distended crop. No other abnormalities were noted. Abnormal laboratory findings included glucosuria, granular urinary casts, elevated uric acid (759 IU/L), and elevated creatinine kinase (493 IU/L)

A tentative diagnosis of chronic renal failure was made, and then confirmed by radiological evidence of nephromegaly and renal mineralization.

An intravenous catheter was inserted and the bird started on Lactated Ringer's Solution t.i.d. and lincomycin-spectinomycin b.i.d. The diet was changed to another commercial hand-rearing diet (Roudybush Formula 3). The crop was supported by an elastic bandage and feeds were reduced in quantity but increased in frequency.

Within 2 days the bird was noticeably improving, with an improved appetite. Polydipsia, polyuria and glucosuria were still present. Antibiotics and fluids were given for one week, and over the next few weeks the bird was gradually weaned. During this time the polyuria decreased until at weaning it was only slightly above normal.

One year later the bird was presented for surgical sexing. At this time the kidneys were observed to be macroscopically normal, and radiographs showed the mineralization had disappeared. She now weighed 720g and was, to all intents, clinically normal.

Discussion.

Hypervitaminosis D₃ is regarded as a common cause of renal mineralization in psittacine chicks. It is particularly common in macaws. Clinical signs include weight loss, crop stasis, regurgitation, polyuria and polydipsia, and general depression.^{6,7} Feed analysis usually shows the feed to have Vitamin D₃ in excess of 1,000 – 4,000 IU/kg. Additionally, Speer has shown that adult breeders oversupplemented with Vitamin D₃ can cause hypervitaminosis D₃ in their offspring.⁶

However, soft tissue mineralization can occur when the diet has Vitamin D₃ levels as low as 400 IU/Kg. Most commercial hand-rearing diets have been implicated in this problem. (Speer, personal communication). Obviously factors other than an absolute excess of Vitamin D₃ are involved.

Dystrophic calcification can occur with any cellular injury. Speer considers it to be common in birds, occurring within days of the initial insult to the injury. When the initiating problem (bacterial infection, glomerular nephropathy, etc.) is resolved, the mineralization also often resolves, disappearing within 30 days in some cases. (Speer, personal communication).

Schmidt (personal communication) observes that the initial calcium deposition occurs within the renal tubular epithelium. At this stage correction of the original problem can prevent further deposition and the mineralization resolves as the tubular epithelium is shed. With more advanced disease, mineralization occurs within the renal interstitial tissue where it remains permanently. In these cases, renal calcification may be observed radiographically either associated with renal disease or as an incidental finding some time after the renal disease has resolved.⁵

In retrospect, determination of dietary Vitamin D₃, serum calcium and a renal biopsy would have been invaluable in elucidating the aetiology of this bird's renal disease. Although this case turned out well, it could have just as easily gone the other way. Improved diagnostic techniques will improve the success rate of renal disease treatment.

Conclusion.

Renal mineralization occurs commonly in birds. Although hypervitaminosis D₃ is usually implicated as a cause, any inflammatory of the kidney can result in mineralization.

Although a general diagnosis of renal disease can be made by history taking, physical examination, radiography and clinical laboratory tests, a definitive diagnosis can only be achieved by renal biopsy. Although general treatment for renal disease can often be successful, in some cases a successful outcome will be dependent on a definitive diagnosis.

Acknowledgements.

The author would like to thank Brian Speer and Bob Schmidt for their tireless correspondence on this matter via the internet.

References.

1. Cheville NF (1983) Cell Pathology, 2nd Ed. Iowa State University Press. Pp 160-163
2. Coltran RS, Kumar V, Robbins SL (1989) Robbins Pathological Basis of Disease, 4th Ed. WB Saunders. Pp 35-36
3. Jones TC, Hunt RD. (1983) Veterinary Pathology, 5th Ed. Lea & Febiger. Pp 65-69
4. Slauson DO, Cooper BJ (1990) Mechanisms of Disease 2nd Ed. Williams & Wilkins pp 81-82
5. Smith BJ, Smith SA (1997) Radiology. In Altman RB, Clubb SL, Dorrestein G, Queensberry K. Avian Medicine and Surgery. WB Saunders. Pg 164
6. Speer BL (1995) Non-infectious diseases. in Abramson J, Speer BL, Thomsen JB. The Large Macaws: their care, breeding and conservation. Raintree Publications. Pp 321-323
7. Takeshita K, Graham DL, Silverman S. (1986) Hypervitaminosis D in baby macaws. Proc. Annual Conf Assoc. Av. Vet, Miami. Pp 341-345.