Nutritional Secondary Hyperparathyroidism in Non-production Birds

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1. INTRODUCTION.

1.1. The Importance of Available Plasma Calcium.

Calcium plays an essential role in many body processes, including neuromuscular excitability and muscle contraction, membrane permeability, enzyme activation and blood coagulation (Fowler 1986; Capen & Martin 1977). It is a vital constituent of the skeletal system, providing structural support and the capacity for movement and locomotion.

It is essential that the concentration of bioavailable calcium in the blood is maintained within appropriate physiological limits, despite variations in calcium intake and excretion. Control is provided principally by parathyroid hormone (PTH) and cholecalciferol (Vitamin D3), which increase blood calcium n, and calcitonin, which decreases blood calcium.

1.2. The Response to Physiological Hypocalcaemia.

1.2.1. Parathyroid Hormone.

PTH is secreted by the chief cells of the parathyroid glands, in the anterior cervical region (Capen & Martin 1977). The secretory cells respond rapidly to hypocalcaemia by releasing stored hormone, and more slowly by increasing its rate of synthesis (Capen & Martin 1977). PTH increases blood calcium and decreases blood phosphorus, by direct effects on bone, kidneys and intestinal mucosa (Capen & Martin 1977). Its main mechanisms of action are to:

- increase phosphorus excretion by the proximal tubules;
- increase calcium resorption by the distal tubules;
- increase the rate of bone remodelling and the net rate of bone resorption;
- increase the rate of osteocytic osteolysis and the number of osteoclasts on bone surfaces; and to
- increase the rate of formation of active metabolites of vitamin D3 in the kidneys (Capen & Martin 1977).

There may be an initial hypocalcaemia, due to the release of calcium phosphate from: bone and soft tissues (Capen & Martin 1977). Phosphate ions released depress available calcium levels by involving free calcium ions in the formation of insoluble salts. After this period however, PTH results in a sustained increase in bioavailable calcium. Feedback control of PTH secretion is provided by the serum concentration of available calcium, and to a lesser extent, magnesium ions (Capen & Martin 1977), allowing the parathyroid glands to provide minute-to-minute control over any calcium fluctuations that occur (Fowler 1986; Capen & Martin 1977).

1.2.2. Cholecalciferol (vitamin D3).

Cholecalciferol enhances calcium ion uptake from the intestine, via the activation of enzymes necessary for transcellular calcium transport by mucosal cells (Capen & Martin 1977).

It also increases the synthesis of calcium binding protein (CaBP) by the intestinal goblet cells, which incorporates into the glycocalyx of the microvilli surface. CaBP enhances calcium absorption by sequestering ingested calcium to areas near the absorptive surface of the gut (Capen & Martin 1977).

Cholecalciferol has a permissive effect on the action of PTH on bone (Fowler 1986; Capen & Martin 1977), and a small amount of this vitamin is necessary if osteoclastic resorption, osteocytic osteolysis and calcium mobilisation is to occur in adults.

1.3. The Pathogenesis of Nutritional Secondary Hvperparathyroidism.

There are several ways in which a bird's diet may lead to calcium deficiency. Under conditions of persistent hypocalcaemia, the parathyroid glands are constantly stimulated to secrete PTH and undergo hypertrophy and hyperplasia (Tangredi & Krook 1999; Filip-pich 1997; Macwhirter 1994; Fowler 1986; Harrison & Harrison 1986; Tollefson 1982; Conlogue et al 1979; Capen & Martin 1977; Arnold et al 1974).

As PTH secretion increases, more and more calcium is mobilised from bone reserves, and osteocytic osteolysis occurs at a far greater rate than in nutritionally healthy birds (Arnold et al 1974). The body's attempts to maintain normocalcaemia thus compromise the health of the bird (Fowler 1986), and clinical disease develops, characterised by systemic illness and skeletal system pathology as well as reproductive and integumentary system abnormalities.

2. THE CAUSES OF DIETARY CALCIUM DEFICIENCY IN BIRDS.

2.1. The Causes of Dietary Calcium Deficiency in Companion Birds.

A calcium deficiency will be induced by diets that lead to a low level of calcium absorption in the gut, contain low levels of vitamin D3, or a large amount of phosphorus. Although birds have a specific calcium appetite and should eat of this mineral readily, individuals may not have developed the ability to select an appropriate diet if they are not exposed to parents and a definite social structure under which these skills are learnt when young (Fowler 1986).

2.1.1. Low Calcium Levels.

Many companion birds are fed seed mixtures, which if unsupplemented, are highly deficient in calcium (Schoemaker et al 1999; Filippich 1997; Macwhirter 1994; Lowenstine 1986; Tollefson 1982; Himmelstein & Bernstein 1978; Arnold et al 1974). Also, the high fat content of some seeds, particularly sunflower and safflower, reduce the absorption of ingested calcium, by forming insoluble, indigestible soaps in the gut (Filippich 1997; Macwhirter 1994; Fowler 1986; Lowenstine 1986).

Foods with adequate calcium may still cause a deficiency if they contain high levels of oxalates (eg rhubarb or spinach), which bind to the calcium present, preventing its absorption (Filippich 1997).

Unsupplemented diets composed only of animal muscle are also low in calcium (Filippich 1997; Lowenstine 1986; Tollefson 1982) and will rapidly result in a clinical calcium deficiency (Fowler 1986).

2.1.2. Low Vitamin D3 Levels.

Vitamin D3 can be obtained in the diet or synthesised in the skin in the presence of ultraviolet light. A vitamin D3 deficiency develops if there is an absolute deficiency of vitamin D3 in the ingesta, if the absorption of vitamin D3 from the ingesta is suboptimal (e.g. when the absorption of dietary fat is faulty (Fowler 1986)), when the activation of cholecalciferol in the liver is impaired (Tangredi & Krook 1999), or when the bird is denied exposure to natural light in the absence of an adequate diet.

2.1.3. High Phosphorus Levels.

Dietary phosphorus depresses calcium absorption by forming insoluble calcium salts in the gut, which are indigestible (Fowler 1986; Antillon 1977).

The important factor regarding dietary phosphorus levels is not its absolute amount, but the ratio of calcium to phosphorus, which is approximately 2:1 within the body (Fowler 1986) and should be approximately 1.5:1 to 2.5:1 in the diet (Filippich 1997; Fowler 1986; Harrison & Harrison1986).

The calcium:phosphorus ratio in unsupplemented seed diets is somewhere between 1:7 and 1:37 (Filippich 1997), and that of an unsupplemented meat diet is 1:25 (Filippich 1997), making both of these diets capable of inducing hypocalcaemia (Filippich 1997; Macwhirter 1994; Fowler 1986; Lowenstine 1986; Tollefson 1982; Himmelstein & Bern-stein 1978; Arnold et al 1974), and subsequent nutritional secondary hyperparathyroidism.

2.2. The Causes of Dietary Calcium Deficiency in Wild Birds.

As wild birds have free access to a variety of foods, and have presumably evolved the ability to choose their diet correctly, one would not expect to see specific nutrient deficiencies. However, dietary calcium deficiency and nutritional secondary hyperparathy-roidism have been observed in free-ranging wild birds and other wild animals (Tangredi & Krook 1999; Beinema et al 1997; Conlogue et al 1979) albeit less frequently than among captive animals.

It is also possible for wild birds to develop disease whilst in human care for rehabilitation, if the carer does not adequately balance the diet.

2.2.1. Improper Diet Choice.

Eighteen cases of nutritional secondary hyperparathyroidism were observed in fledgling American crows *(Corvus Brachyrhynchus Brachyrhynchua)* in a New York suburb, over two years. These birds were in good body condition (i.e. were not generally malnourished), and their disease was attributed to high human garbage and "road kill" consumption, which is low in bioavailable calcium and vitamin D3, due to its high connective tissue and fat content (Tangredi & Krook 1999).

It is also important to remember that the optimal foraging theory of diet choice does not hold true for all individuals, but rather for those of high fitness. It is possible therefore that individuals exposed to a range of foods will not choose their diet adequately, and may develop specific deficiencies.

2.2.2. Consumption of Exogenous Xenobiotics.

Calcium deficiency may also occur due to the consumption of exogenous xenobiotics in insecticides, including aluminium, cholinesterase inhibitor pesticides and DDT, either directly or through consumption of affected invertebrates (Tangredi & Krook 1999). These chemicals compete with the activation of cholecalciferol to 25-dihydroxy-cholecalciferol. By this mechanism, chronic exposure to such chemicals may induce a deficiency of vitamin D_3, which may suppress calcium absorption sufficiently to produce hypocalcaemia.

2.2.3. Acidification.

Hypocalcaemia-induced rickets was observed in free-living Black terns *(Chlidonas niger)* nesting on acid bogs in the Netherlands (Beinema et al 1997). Acidification resulted in fish mortality (so that the birds were forced to subsist on insects) and a low calcium content of these insects. Almost complete juvenile mortality was observed and this species became locally extinct. It is likely that a similar acidification process in forest soils and other habitats could have the same effect upon insectivorous bird species in those areas.

3. CLINICAL SIGNS.

The clinical signs of the disease vary with age, sex and reproductive status (Filippich 1997) as well as the degree and duration of deficiency and the presence of concurrent disease (Fowler 1986).

Clinical signs may include a combination of the following (Filippich 1997; Macwhirter 1994; Fowler 1986; Harrison & Harrison 1986; Tollefson 1982; Himmelstein and Bern-stein 1978):

Skeletal System Signs:

- Skeletal fractures, resulting in progressive or intermittent lameness.
- Cage paralysis, where there is reluctance or complete refusal to move.
- Compression fractures of the vertebral column resulting in paraplegia.
- Pain upon manipulation or palpation of bones or joints.
- Inability to perch, attributed to clenching of the feet.
- Palpable deformities in the keel or long bones in young animals.
- Bowing of the weight-supporting long limb bones.
- Stunted posture in birds affected since youth, as a result of impaired ossification causing growth restriction.

Muscular Signs:

- Drooping of the wings due to muscle weakness.
- Muscular trembling (fasciculation).
- Hypocalcaemic tetany, where the bird appears weak and drowsy, and sways back and forth slightly on its perch.

Systemic Signs:

- Anorexia, attributed to pain in the bones and joints responsible for mastication.
- Polyuria, in an attempt to increase phosphorus excretion, with secondary polydipsia and sometimes dehydration.
- Depression and apparent drowsiness.
- Pathological regurgitation.

- Hypocalcaemic seizures, especially when startled. They last between 15 and 45 seconds, and will cause death if they persist long enough to prevent respiration.

Integumentary System Signs:

- Poor quality feathering, moulting abnormalities and feather picking.
- Reddened claws and legs.

Reproductive System Signs:

- Soft-shelled eggs.
- Egg-binding.
- Cessation of egg-laying.

In mammals, progression to fibrous osteodystrophy entails facial deformity due to thickening of the mandibles, maxilla and premaxilla (Fowler 1986). Facial swelling may impinge upon the nares causing respiratory problems (Fowler 1986). There were no reports of a similar problem occurring in birds suffering nutritional secondary hyperparathyroid-ism in the available literature.

4. HISTOPATHOLOGICAL FINDINGS.

4.1. Histopathology of the Parathyroid Glands.

The parathyroid glands become hypertrophic and hyperplastic with persistent hypercal-caemia. This may be observable grossly during necropsy (Arnold et al 1974), and histologically after collection and appropriate fixation. The light (active) chief cells are hypertrophic and have enlarged nuclei with several nucleoli (Tangredi & Krook 1999). Water clear cells, representing exhausted cells at the end stage of a prolonged period of hyperactivity, are abundant throughout histological sections (Tangredi & Krook 1999).

4.1.2. Histopathology of the Bone.

The main histopathological features seen in bone will differ with each case, depending on the degree and duration of calcium deficiency.

The general features expected include:

- *Intense osteocytic osteolysis*, evidenced by large round osteocytes and matrix basophilia with a scalloped appearance to bone margins when resorption is subperiosteal (Tangredi & Krook 1999). There may be pronounced osteopenia (reduced bone mass) and a virtual absence of the growth plate in epiphyseal regions (Tangredi & Krook 1999). Osteoclastic bone resorption is not of great importance in the pathogenesis of this disease (Antillon et al 1977).

- *Impaired fracture healing.*. Longitudinal sections of tibiotarsal fractures in the wild crows showed poor alignment and a chronic callus extending throughout the diaphysis, consisting of bone trabeculae and fibrous tissue surrounded by bone lined by wide osteoid seams (Tangredi & Krook 1999).

- Osteodystrophiafibrosa, where there is replacement of resorbed bone by fibro-osseus tissue. Transverse sections of affected bone will show thin trabeculae,

separated by large amounts of fibrous tissue, and a lack of primary osteons (Arnold et al 1974).

5. BIOCHEMISTRY AND URINALYSIS.

Biochemistry and urinalysis are useful diagnostic tools, and changes expected during nutritional secondary hyperparathyroidism include:

- *Increased Alkaline Phosphatase,* due to increased osteoblastic activity (Tangredi & Krook 1999; Fowler 1986; Capen & Martin 1977).
- *Normal or decreased blood calcium levels,* depending on the efficacy of compensation. A decrease in the plasma calcium:phosphorus ratio may be attributed to a hyper-phosphatasaemia, assumed to be a biochemical expression of bone resorption (Tangredi & Krook 1999).
- *Decreased blood vitamin D3 levels*
- Increased urinary phosphorus levels due to excessive phosphorus excretion by the proximal tubules (Capen and Martin 1977).
- *Decreased urinary calcium levels,* due to enhance calcium resorption by the distal tubules (Capen & Martin 1977).
- *Increased blood PTH Levels.* Increased levels of PTH in the circulation as demonstrated by radioimmunoassay in man has been beneficial in the early detection of hy-perparathyroidism (Capen & Martin 1977).

6. RADIOGRAPHIC LESIONS.

The most obvious and common lesions are those involving the skeletal system, ra-diographically visible when approximately 40% of the original bone minerals have been resorbed (Fowler 1986). Lesions characteristic of hyperparathyroidism include:

- *Subperiosteal bone resorption,* due to the activation of osteoprogenitor cells in the periosteal bone envelope and the formation of metabolic units on the periosteal surface (Capen & Martin 1977). Medullary bone is generally affected to a greater degree than cortical bone (Antillon et al 1977; Capen & Martin 1977), reflecting its naturally higher turnover rate.

- *Osteomahtcia,* due to deficient bone mineralisation with excess accumulation of os-teoid (Tollefson 1982).

- *Rickets,* in growing birds due to deficient mineralisation of the growth plate cartilage (Filippich 1997). Chicks often show bowing deformities of the weight-bearing bones (Capen & Martin 1977).

- *Increased incidence of skeletal fractures,* due to inadequate mineralisation, causing bone weakness. The disease is characterised by folding fractures of the weight-supporting long bones, the vertebral column and occasionally the ribs (Tangredi & Krook 1999; Fowler 1986). Fractures are slower to heal than in normal birds (Filippich 1997; Harrison & Harrison 1986), and complete fractures may heal at peculiar angles (Fowler 1986), or not at all.

- *Osteodystrophiafibrosa,* where cortices appear thicker than normal, due to the replacement of mineralised bone with fibrous connective tissue (Harrison & Harrison 1986).

- *Pelvic compression*, with medial displacement of the acetabula and twisting of the ilia (Fowler 1986), causing dystocia and obstipation.

- *Spinal Lesions*, including scoliosis (lateral deviation), lordosis (a downward curvature of the lumbar spine), kyphosis (increased convexity in the thoracic spine when viewed laterally) and compression fractures (Fowler 1986).

7. DIAGNOSIS.

7.1. Differential Diagnoses.

7.1.1. Renal secondary Hyperparathyroidism.

Renal secondary hyperparathyroidism is also caused by excessive secretion of PTH (Capen & Martin 1977), in which there is no primary pathology in the parathyroid glands themselves. As with nutritional secondary hyperparathyroidism, the secretion of PTH is not autonomous and remains responsive to changes in blood calcium concentration (Capen & Martin 1977). The disease shares many clinical features with nutritional secondary hyperparathyroidism but is caused by chronic progressive renal disease, in which a reduced glomerular filtration rate causes phosphate retention and subsequent hyper-phosphataemia (Capen & Martin 1977). Free phosphorus stimulates PTH release indirectly via the reduction of blood calcium, which is exacerbated by impaired gut absorption due to the inadequate production of active metabolites of cholecalciferol in the diseased kidney (Capen & Martin 1977).

7.1.2. Primary Hyperparathyroidism.

Primary hyperparathyroidism occurs when there is excessive autonomous release of PTH due to a functional lesion within the parathyroid gland (Capen & Martin 1977), indepen-dant of calcium levels. The clinical consequences of primary hyperparathyroidism are similar to that of renal and nutritional secondary hyperparathyroidism. Nephrocalcinosis, resulting in renal insufficiency, and urolithiasis may result from the increased urinary excretion of phosphorus and calcium (Capen & Martin 1977).

7.1.3 Pseudohvperparathyroidism.

Pseudohyperparathyroidism results in persistent hypercalcaemia and hypophosphataemia, due to the ectopic secretion of PTH or PTH-like polypeptides, or other bone-resorbing substances from neoplasms. Clinical signs are similar to those of other differentials, however all abnormalities reverse if the tumour is removed.

7.2. Reaching a Definitive Diagnosis.

Secondary hyperparathyroidism should be easily differentiated from primary hyperpara-thyroidism or pseudohyperparathyroidism by the measurement of serum calcium concentration. Due to autonomous release of PTH (which does not occur in secondary hyper-parathyroidism), marked hypercalcaemia will only be a consistent feature of primary hy-perparathyroidism (Capen & Martin

1977) and pseudohyperparathyroidism, as will low phosphorus levels due to the inhibition of tubular resorption of phosphorus.

In renal secondary hyperparathyroidism, the blood urea and creatinine levels are consistently elevated, and the glomerular filtration rate and renal blood flow are decreased (Capen & Martin 1977). A moderate hypercalcaemia may develop because of impaired PTH degradation by the kidneys, decreased urinary calcium excretion or hypercitricemia (Capen & Martin 1977).

A diagnosis of nutritional secondary hyperparathyroidism can thus be made when there is the clinical signs of hyperparathyroidism in the presence of hypocalcaemia and the absence of renal insufficiency. This diagnosis will be supported by the demonstration of dietary inadequacy.

8. TREATMENT.

When examining a patient suspected of this disease it is important to be aware of vulnerability of the skeleton to fractures, during the most routine of handling procedures (Harrison & Harrison 1986).

Intensive calcium therapy is required in order to reverse the deficiency, until the body reserves are restored (Macwhirter 1994). Calcium should be given orally or parenterally for 4-6 weeks, and if the deficiency is severe, a vitamin D3 supplement should also be given for the first 2-4 weeks of treatment (Filippich 1997). Parenteral supplements are preferable in acute situations (Fowler 1986). The response to such treatment is usually dramatic and most birds will attain clinical normality within one month (Himmelstein & Bernstein 1978) of a diet containing a 2:1 calcium:phosphorus ratio.

During recovery, the bird should be confined to a padded box or suspended in a sling in a dark, quiet and warm environment to prevent skeletal injury (Filippich 1997), and the skeleton will remain vulnerable for some weeks (Capen & Martin 1977). The improvement in the skeleton can be monitored radiographically if finances permit.

If the feet are twisted, clipping of the nails to prevent injury to the skin of the feet, will have to be weighed against the possibility of causing bone injury during the procedure (Himmelstein & Bernstein 1978).

Generally the prognosis with treatment is good, unless there has been spinal fractures or compression leading to nerve damage.

9. PREVENTION AND CONTROL.

A crucial aspect of prevention and control is to correct the dietary faults that caused the hypocalcaemia. The owner or wildlife carer must be made aware of the importance of management of the calcium:phosphorus ratio of the diet. In general terms, the calcium requirement expressed as a percentage of diet in growing, laying and non-laying birds is 0.55%, 0.35% and 0.05-1% respectively, with phosphorus in the correct ratio.
Calcium supplements are diverse and should be accepted by the bird due to their specific calcium appetite (Filippich 1997). The various forms of calcium supplements available for use in birds include (Filippich 1997; Macwhirter 1994):

- Calcium syrup, added to the water, the seed or given directly. There is evidence that the addition of supplements to the water may make the water unpalatable and so encourage dehydration. As seed-eaters dehusk the seed before eating it, a coating of calcium syrup on

the outside of the seed will only be ingested when the bird mouths the seed during the dehusking process.

- High calcium foods such as bones, cheese, almonds, dandelion, parsley and yoghurt.
- Calcium carbonate powder, sprinkled onto food or mashed in to soft foods.
- Free-choice access to cuttle-fish, calcium blocks and calcium-containing grit mixtures It is important to ensure the bird is actually ingesting the supplement, as most birds do not accept new food items readily. Supplements can be force-fed easily using a crop needle or stomach tube if necessary. Calcium absorption may be enhanced by the addition of psyllium (Macwhirter 1994) or high levels of protein (Filippich 1997) to the diet.

Vitamin D3 may require supplementation (Macwhirter 1994), and in-water supplements are cheaply available commercially. The easiest and cheapest way to prevent a vitamin D3 deficiency is to expose the bird to natural sunlight at frequent intervals.

Intensive supplementation should not exceed the period needed for the body reserves to return to normal, and the permanent diet should not contain more than 1.2% bioavailable calcium (Filippich 1997). Oversupplementation of calcium may interfere with magnesium and zinc absorption, and lead to renal calcium deposition, and ultimately renal failure (Macwhirter 1994).

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