



Metabolic bone disease and central retinal degeneration in a kitten due to nutritional inadequacy of an all-meat raw diet

Journal of Feline Medicine and Surgery
Open Reports
 1–5

© The Author(s) 2015
 Reprints and permissions:
sagepub.co.uk/journalsPermissions.nav
 DOI: 10.1177/2055116915579682
jfmsopenreports.com



Catherine Lenox¹, Iveta Becvarova² and Wendy Archipow³

Abstract

A 5-month-old castrated male Sphynx kitten presented with left hindlimb lameness shortly after adoption. Prior to adoption, the breeder had fed the kitten an exclusively raw chicken diet. Radiographs revealed generalized osteopenia and a left tibia–fibula fracture. Ophthalmic examination revealed corneal vascularization and opacity in the right eye, and lesions suggestive of feline central retinal degeneration in the left eye. The patient's diagnoses included metabolic bone disease and feline central retinal degeneration, which can result from taurine deficiency. The kitten's nutritional diseases were managed with a complete and balanced canned diet designed for kitten growth and with taurine supplementation.

Accepted: 28 July 2014

A 5-month-old castrated male Sphynx kitten presented to the Virginia–Maryland Regional College of Veterinary Medicine with a chief complaint of left hindlimb lameness. The kitten had been adopted 11 days prior to presentation from a breeder. At the time of adoption, the kitten had a small right eye and left hindlimb lameness. The owners reported that, prior to adoption, the breeder had fed the kitten a diet that consisted exclusively of raw chicken without any additional food or supplements. Since adoption, the kitten had been fed two different types of commercially available kitten food (Iams Premium Pate with Gourmet Chicken – Kitten; Hill's Science Diet Kitten canned formula), and seemed to be eating and consuming water normally. The referring veterinarian had referred the case to the orthopedic surgery service for evaluation and management of the left hindlimb lameness.

The patient weighed 1.5 kg and its body condition score (BCS) was below ideal at 4/9. A mild decrease in muscle condition was noted. It had a small right eye, brown ocular discharge and that eye also had corneal vascularization. The skin was dry and scaly. A weight-bearing left hindlimb lameness was observed when the patient's splint was removed. There were no other significant findings on physical examination.

The initial diagnostic plan for the patient included radiographs of both hindlimbs and an ophthalmology consultation. Radiographic findings included generalized osteopenia, a folding fracture of the left tibia with partial callus formation and a transverse fracture of the left fibula with malunion (Figure 1). The right hindlimb showed no evidence of fracture. There was widening of the distal right femoral physis (not shown). Radiographs were also taken of the pelvis and left forelimb, which also indicated generalized osteopenia (not shown).

¹Royal Canin, 500 Fountain Lakes Blvd Ste 100, St Charles, MO, USA

²Hill's Pet Nutrition Manufacturing, Prague, Czech Republic

³Brisbane Veterinary Specialist Centre, Albany Creek, Australia

Presented in abstract form at the 2011 American Academy of Veterinary Nutrition Clinical Nutrition and Research Symposium, Denver, CO, USA.

Corresponding author:

Catherine Lenox DVM, DACVN, Royal Canin USA, 500 Fountain Lakes Blvd Ste 100, St Charles, MO 63301
 Email: catherine.lenox@royalcanin.com





Figure 1 Radiograph of the left hindlimb at presentation, showing a pathologic tibia–fibula fracture

Given the dietary history prior to adoption, the radiographic findings of generalized osteopenia and the pathologic fracture, a tentative diagnosis of metabolic bone disease caused by consumption of an unbalanced diet was made. Differential diagnoses included nutritional secondary hyperparathyroidism (NSHP) and/or rickets. Blood was collected and submitted for a complete blood count (CBC) and serum biochemistry profile, and for intact parathyroid hormone (PTH), ionized calcium and 25-hydroxyvitamin D levels, which were all within normal limits (Table 1). The CBC indicated a leukocytosis at 20,010 cells/ μ l (reference interval 4250–14,610 cells/ μ l), a mature neutrophilia at 11,086 cells/ μ l (reference interval 2272–9639 cells/ μ l), and a monocytosis at 1181 cells/ μ l (reference interval 0–952 cells/ μ l). The serum biochemistry profile showed an elevated blood urea nitrogen (42 mg/dl; reference interval 18–32 mg/dl), hyperphosphatemia (7.1 mg/dl; reference interval 2.4–5.9 mg/dl), and an elevated alkaline phosphatase (196 U/l; reference interval 9–73 U/l). The reference intervals for serum phosphorus and alkaline phosphatase were not adjusted for a growing animal.

Table 1 Parathyroid hormone, ionized calcium and 25-hydroxyvitamin D levels

Parameter	Reference interval	Value at initial visit
Intact parathyroid hormone (pmol/l)	0.00–4.00	2.50
Ionized calcium (mmol/l)	1.00–1.40	1.30
25-hydroxyvitamin D (nmol/l)	65–170	93

On ophthalmic examination, the patient seemed to have some visual capacity in both eyes. The right eye was small and it was not possible from the diagnostics performed to determine for certain whether it was small owing to microphthalmia or phthisis bulbi due to an acquired infection. The kitten had some brown ocular discharge for which it was receiving amoxicillin–clavulanic acid from the referring veterinarian, so infectious keratitis was a differential. The right cornea was vascularized and the intraocular structures were poorly visualized. However, the left cornea was clear, and fundus examination could be performed. Examination revealed a focal area of retinal hyper-reflection in the area centralis. This type of lesion is suggestive, but not pathognomonic, of taurine deficiency-associated retinal degeneration or feline central retinal degeneration (FCRD). However, owing to the kitten's size, it was not possible to collect enough blood to submit whole blood taurine levels to confirm taurine deficiency. In addition, owing to the fact that the kitten had consumed a complete and balanced diet for about 10 days, taurine levels at the time of presentation may not have been helpful.

Given the history, physical examination findings and radiographic findings, the patient's problem list included metabolic bone disease (NSHP and/or rickets), left tibia–fibula fracture, and potential past or current taurine deficiency. The past raw chicken diet was analyzed by comparing nutritional profiles obtained from the US Department of Agriculture's (USDA) Food Composition Database with recommended allowances for kitten growth set by the National Research Council (NRC) and minimum recommendations for kitten growth of the Association of American Feed Control Officials (AAFCO) (Table 2).

Nutritional intervention was recommended to address these problems. Nutritional goals for this patient included feeding adequate nutrients for growth in the form of a complete and balanced diet and providing nutrients to improve bone health. In addition, special attention was paid to the patient's life stage and specific nutritional needs of a growing kitten, and to the patient's current low BCS. Key nutritional factors for this kitten included energy and energy density, protein, taurine, fat, docosahexaenoic acid, calcium, phosphorus, vitamin D, potassium, sodium, zinc, copper, magnesium and vitamin C.

Table 2 Analysis of the kitten's past diet

Nutrient content	NRC recommended allowance for kitten growth ¹	Raw chicken*
Protein	5.6 g/100 kcal ME	18.62 g/100 kcal ME
Fat	2.25 g/100 kcal ME	2.27 g/100 kcal ME
Carbohydrate (NFE)	N/A	0 g/100 kcal ME
Crude fiber	N/A	0 g/100 kcal ME
Calcium	200 mg/100 kcal ME	4.39 mg/100 kcal ME
Phosphorus	180 mg/100 kcal ME	184.21 mg/100 kcal ME
Sodium	35 mg/100 kcal ME	101.75 mg/100 kcal ME
Potassium	100 mg/100 kcal ME	324.56 mg/100 kcal ME
Magnesium	10 mg/100 kcal ME	22.81 mg/100 kcal ME
Taurine	10 mg/100 kcal ME	13.96 mg/100 kcal ME
Vitamin D	5.6 IU cholecalciferol/100 kcal ME	4.38 IU/100 kcal ME
Vitamin C	N/A	1.05 mg/100 kcal ME
Copper	0.21 mg/100 kcal ME	NR
Zinc	1.85 mg/100 kcal ME	0.51 mg/100 kcal ME
EPA + DHA	2.5 mg/100 kcal ME	NR

*Raw chicken: from Spitze et al (taurine content, using average content, converted to caloric content basis)² and the US Department of Agriculture's Food Composition Database (<http://www.nal.usda.gov/fnic/foodcomp/search/>) searched for 'chicken, broilers or fryers, breast, meat only, raw', accessed 28 July 2013, and converted to caloric content basis (all other nutrients)

NRC = National Research Council; NFE = nitrogen-free extract; EPA = eicosapentaenoic acid; DHA = docosahexaenoic acid; ME = metabolizable energy; N/A = not applicable; NR = not reported

These nutrients were selected as the most crucial for kitten growth, tissue development, and bone health and development.

The patient's ideal weight was calculated based on the current BCS of 4/9 and current weight of 1.5 kg. Using a BCS of 5/9 as the ideal body condition, the patient's current weight was considered to be 90% of its ideal. This assumes that each BCS point, on a nine-point scale, accounts for approximately 10–15% of body weight.³ The ideal weight was estimated to be 1.7 kg ($[1.5 \text{ kg}/90\%] = [1.5 \text{ kg}/0.9] = 1.7 \text{ kg}$). This weight would change quickly owing to the age of the kitten, but the goal to achieve an ideal BCS was important along with the primary goals listed above. Resting energy requirements (RER) were calculated for the ideal weight ($\text{RER} = 70 \times [1.7 \text{ kg}]^{0.75} = 103 \text{ kcal metabolizable energy [ME] per day}$). Daily energy requirements (DER) were calculated using a DER factor of 2.5 ($\text{DER} = 2.5 \times \text{RER} = 2.5 \times 103 = 257 \text{ kcal ME per day}$). This is the factor that is recommended for kittens under 6 months of age.⁴ An energy-dense and highly digestible diet (4.0–5.0 kcal/g dry matter) was recommended to allow the patient to consume enough energy without it having to consume a large volume of food.

The primary nutritional recommendation after evaluating the key nutritional factors for growth and bone healing and development was to choose a complete and balanced diet formulated for growth, and to feed this diet in energy levels sufficient to support growth. The specific recommendations were to feed a commercially available canned kitten diet (Hill's Science Diet Kitten canned formula) in two flavors at 1.25 cans per day (256–263 kcal ME/day) divided into three meals, with fresh water available at all

times. This diet was recommended to meet or exceed NRC recommended allowances for kittens postweaning,¹ and the AAFCO minimums for growth and reproduction.⁵ Based on assessment of the label of the selected diet, the diet had undergone an AAFCO animal feeding protocol for kitten growth. Crystalline taurine was prescribed at 250 mg once daily, in case taurine deficiency was present. This dose was chosen in order to exceed NRC minimum requirements for taurine for kittens postweaning, and has been shown to reverse dilated cardiomyopathy in cats associated with taurine deficiency.⁶

The tibia–fibula fracture was managed with cage rest and a lateral splint. Radiographs were taken 4 weeks after the patient's initial visit by the referring veterinarian and showed remarkable bone healing of the tibia and fibula fractures. Radiographs taken 4 weeks later showed complete healing. The client reported that the kitten had made a full recovery after 2 months, and that it was extremely active and fully ambulatory.

Nutritional secondary hyperparathyroidism represents a normal physiologic response to nutritional imbalances. Diets that are capable of inducing this condition are low in calcium, low in vitamin D or high in phosphorus, with low-to-normal levels of calcium.⁷ After a diet with one of these characteristics is fed for a period of time, calcium levels decrease and PTH levels increase to compensate. PTH is involved in calcium and phosphorus homeostasis. Parathyroid stimulation results in calcium removal from bone to maintain serum calcium levels, resulting in osteopenia. Compensation in patients with NSHP is typically adequate for the maintenance of blood calcium and phosphorus levels within reference

intervals, but increased levels of PTH are needed to maintain normal blood calcium and phosphorus concentrations.⁷ NSHP is most commonly seen in animals that ingest unbalanced all-meat diets.⁷

Rickets occurs in young animals and is most often caused by dietary vitamin D deficiency. Vitamin D deficiency frequently occurs in conjunction with calcium and phosphorus imbalances,⁸ and, owing to overlap of clinical and physiologic signs, can be difficult to separate from NSHP. Vitamin D aids in intestinal absorption of calcium. Vitamin D and PTH work together to maintain calcium and phosphorus within their respective physiologic ranges.⁹ Vitamin D also has a variety of functions unrelated to calcium metabolism including modifying gene expression.

All-meat diets are commonly associated with nutritional deficiencies and excesses, and resulting nutritional disorders, especially in growing animals. The patient's previous raw chicken diet was deficient in calcium and vitamin D, with a reversed calcium to phosphorus ratio (Ca:P ratio). According to the USDA's National Nutrient Database (<http://www.nal.usda.gov/fnic/foodcomp/search/> searched for 'chicken, broilers or fryers, breast, meat only, raw' on 28 July 2013), 100 g raw chicken breast contains 5 mg calcium and 210 mg phosphorus, making the Ca:P ratio 0.024:1. The recommended Ca:P ratio for growing kittens is 1:1–1.5:1.⁴ One hundred grams of raw chicken breast also contains 0.1 µg of vitamin D₂ + D₃. A growing feline patient weighing 1.5 kg would have to consume almost 400 g of raw chicken per day to meet the NRC's recommended allowances for vitamin D₃ (0.381 µg/day).¹ The owners of the kitten were uncertain of what cut of chicken and whether or not skinless chicken was fed, but this does not significantly affect the dietary calcium or vitamin D concentrations. The reversed Ca:P ratio (<1:1) in the raw chicken diet resulted in the suspected NSHP, while the probable vitamin D deficiency indicates that rickets was also likely a component of the bone disease. The nutritional history, combined with radiographic evidence of both generalized osteopenia and widening of the right distal femoral physis, indicates that this kitten likely had both NSHP and rickets. Generalized osteopenia with thin cortices is a radiographic sign of NSHP,¹⁰ while widening of the physes has been described in kittens with rickets.¹¹ In addition, there were likely other nutritional deficiencies and excesses associated with the diet that this kitten was fed prior to adoption.

An additional potential nutritional deficiency was detected in this patient during the ophthalmology consultation. The retinal lesion noted in the left eye indicated that the patient may have had taurine deficiency-associated retinal degeneration or FCRD. Owing to low levels of activity of two enzymes – cysteine dioxygenase and cysteine sulfinic acid decarboxylase – taurine is an essential amino acid for cats.¹² These enzymes are present in the

liver of other species and are involved in the production of taurine from cysteine. Taurine is involved in fetal development, growth, reproduction, sight, cardiac function, bile acid conjugation and other biological processes.¹

The mean taurine level of raw, boneless, skinless chicken breast is reported to be 159 mg/kg wet weight (range 102–216 mg/kg wet weight).² This suggests that this kitten, with a taurine requirement of approximately 28 mg/day,¹ using the reported mean taurine concentration of raw, boneless, skinless chicken breast, would have had to consume 176 g raw chicken breast (201 kcal/day) to meet taurine requirements. It is unlikely that an underweight kitten weighing 1.5 kg could consume this amount of chicken on a daily basis.

Taurine deficiency in cats can result in FCRD, which can progress to blindness; dilated cardiomyopathy (DCM), which can lead to heart failure; poor neonatal growth; and other problems in growing and adult animals.¹ A series of papers published in the 1970s indicated that dietary taurine is necessary for maintaining normal retinal function in the cat and that retinal taurine concentration could be significantly reduced within 10 weeks of feeding a taurine-free diet (casein) to cats.^{13–15} Another study indicated that retinal degeneration was noted after 3–7 months of feeding a semi-purified, casein-based diet.¹⁶ If this patient did have FCRD, it would have likely been on a taurine-free or severely taurine-restricted diet since weaning. FCRD is irreversible but its progression can be halted by taurine supplementation.

Another consequence of taurine deficiency is DCM. A low plasma taurine concentration has been associated with myocardial failure in cats, and many cats can improve after taurine supplementation.⁶ Ideally, an echocardiogram would have been performed on this patient to evaluate for evidence of DCM, despite the absence of a heart murmur, but owing to the financial concerns of the owners it was not. However, not all cats with FCRD have DCM, and vice versa.¹

The most remarkable parameter on the serum biochemistry panel was the elevated blood urea nitrogen. A urinalysis would have ideally been collected to rule out renal azotemia, but was not. It could have been due to renal disease, dehydration or a high protein diet, among other causes. Although the kitten's hyperphosphatemia could have been related to its metabolic bone disease, it is more likely that it was related to not having a laboratory reference interval for growing animals. Alkaline phosphatase is also commonly elevated in growing animals. Intact PTH, ionized calcium and 25-hydroxyvitamin D were all within normal limits. This is likely owing to the fact that the diet was changed 11 days prior to sampling. While the patient had radiographic evidence of metabolic bone disease, serum values likely corrected more quickly than the skeletal system. The patient's

white blood cell count was elevated with a neutrophilia. This could have been owing to an issue with the previously fed raw meat-based diet (RMBD) causing infection, or it could have been related to the ocular discharge and potential infection causing it.

In addition to the nutritional adequacy issues resulting in metabolic bone disease and FCRD, the all-meat raw chicken diet was also unlikely to contain adequate levels of other essential nutrients. It is possible to have fatty acid deficiencies when feeding cats RMBDs containing muscle meat.¹⁷ However, the specific cut of meat and the amount of meat fed to this kitten was unknown, making it difficult to determine which nutrients were deficient in the previous diet.

RMBDs can also have issues with food safety. Feeding a pet raw meat can expose the animal to potentially lethal infections such as salmonellosis or *Escherichia coli*, *Campylobacter* species or protozoal infections. In one case series, two cats fed a beef-based homemade RMBD contracted lethal *Salmonella* infections. The bacteria isolated from necropsy of one cat matched the *Salmonella* species cultured from the diet.¹⁸ Another study performed in dogs consuming a *Salmonella*-infected, commercially available RMBD indicated that dogs shedding *Salmonella* did not have clinical signs of salmonellosis.¹⁹ These studies indicate that while clinical signs may or may not be present after dogs and cats consume a RMBD, the risk is real. The kitten described did not have a fecal examination or culture performed to rule out exposure to *Salmonella* or other potentially hazardous bacteria or protozoa. In addition to the food safety issues associated with feeding a raw diet, the nutritional adequacy of all-meat and RMBDs is also a major consideration.

Conclusions

This case highlights the importance of feeding a complete and balanced diet formulated for the appropriate species and life stage.

Funding The authors received no specific grant from any funding agency in the public, commercial or not-for-profit sectors for the preparation of this case report.

Conflict of interest The authors declare no conflicts of interest.

References

- 1 NRC. Nutrient requirements of dogs and cats. Washington, DC: The National Academies Press, 2006.
- 2 Spitze AR, Wong DL, Rogers QR, et al. **Taurine concentrations in animal feed ingredients; cooking influences**

- 3 **taurine content.** *J Anim Physiol Anim Nutr (Berl)* 2003; 87: 251–262.
- 3 Laflamme DP. **Understanding and managing obesity in dogs and cats.** *Vet Clin North Am Small Anim Pract* 2006; 36: 1283–1295.
- 4 Gross KL, Becvarova I and Debraekeleer J. **Feeding growing kittens: postweaning to adulthood.** In: Hand MS, Thatcher CD, Remillard RL, et al (eds). *Small animal clinical nutrition*. Topeka, KS: Mark Morris Institute, 2010, pp 429–436.
- 5 AAFCO. 2013 official publication. Oxford, IN: Association of American Feed Control Officials, Inc, 2013.
- 6 Pion PD, Kittleson MD, Rogers QR, et al. **Myocardial failure in cats associated with low plasma taurine: a reversible cardiomyopathy.** *Science* 1987; 237: 764–768.
- 7 Feldman EC and Nelson RW. *Canine and feline endocrinology and reproduction*. St Louis, MO: Saunders, 2004.
- 8 Wedekind KJ, Yu S, Kats L, et al. **Micronutrients: minerals and vitamins.** In: Hand MS, Thatcher CD, Remillard RL, et al (eds). *Small animal clinical nutrition*. Topeka, KS: Mark Morris Institute, 2010, pp 107–148.
- 9 Holick MF. **Vitamin D.** In: Stipanuk MH (ed). *Biochemical, physiological, and molecular aspects of human nutrition*. St Louis, MO: Saunders, 2006, pp 863–883.
- 10 Wisner ER and Konde LJ. **Diseases of the immature skeleton.** In: Thrall DE (ed). *Textbook of veterinary diagnostic radiology*. 4th ed. Philadelphia, PA: Saunders, 2002, pp 146–160.
- 11 Geisen V, Weber K and Hartmann K. **Vitamin D-dependent hereditary rickets type I in a cat.** *J Vet Intern Med* 2009; 23: 196–199.
- 12 Rucker RB, Morris J and Fascetti AJ. **Vitamins.** In: Kaneko JJ, Harvey JW and Bruss ML (eds). *Clinical biochemistry of domestic animals*. Burlington, MA: Academic Press, 2008, pp 695–730.
- 13 Berson EL, Hayes KC, Rabin AR, et al. **Retinal degeneration in cats fed casein. II. Supplementation with methionine, cysteine, or taurine.** *Invest Ophthalmol* 1976; 15: 52–58.
- 14 Schmidt SY, Berson EL and Hayes KC. **Retinal degeneration in cats fed casein. I. Taurine deficiency.** *Invest Ophthalmol* 1976; 15: 47–52.
- 15 Schmidt SY, Berson EL, Watson G, et al. **Retinal degeneration in cats fed casein. III. Taurine deficiency and ERG amplitudes.** *Invest Ophthalmol Vis Sci* 1977; 16: 673–678.
- 16 Rabin AR, Hayes KC and Berson EL. **Cone and rod responses in nutritionally induced retinal degeneration in the cat.** *Invest Ophthalmol* 1973; 12: 694–704.
- 17 Kerr KR, Beloshapka AN, Morris CL, et al. **Evaluation of four raw meat diets using domestic cats, captive exotic felids, and cecectomized roosters.** *J Anim Sci* 2013; 91: 225–237.
- 18 Stiver SL, Frazier KS, Mauel MJ, et al. **Septicemic salmonellosis in two cats fed a raw-meat diet.** *J Am Anim Hosp Assoc* 2003; 39: 538–542.
- 19 Finley R, Ribble C, Aramini J, et al. **The risk of salmonellae shedding by dogs fed *Salmonella*-contaminated commercial raw food diets.** *Can Vet J* 2007; 48: 69–75.