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CASE REPORT

Nutritional Secondary Hyperparathyroidism in an African Lion Cub (*Panthera leo*)

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A B S T R A C T

The present case report describes nutritional secondary hyperparathyroidism (NSH) in an African lion cub of 4 month age presented at Veterinary Teaching Hospital (VTH), University of Agriculture, Faisalabad, Pakistan with the complaint of lethargy, lameness and pelvic limb paresis from 2 weeks. Diagnosis of NSH was arrived on the basis of history of feeding lean meat, characteristic radiographical findings including epiphyseal fracture, femoral cupping, marginal radiopacity and pelvic collapse along with low serum calcium and high alkaline phosphatase (ALP) level. The patient was recovered within 6-7 weeks after administration of calcium and vitamin D source along with application of plaster of paris (POP) bandage in order to enhance recovery.

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INTRODUCTION

Nutritional secondary hyperparathyroidism (NSH) is a metabolic disorder of wild but captive handfed individuals of certain species of animals, characterized pathologically by mild to severe bone resorption followed by osteopenia. This metabolic condition is caused by a high dietary intake of phosphorus and chronic decrease in calcium. Rapidly growing young dogs, cats, lions, tigers requiring high dietary calcium are by far the most commonly affected animals. NSH is directly related to poor nutritional management of these animals as captive animals are usually fed on balanced commercial diets, meat or organ tissues. Normal skeletal growth needs Ca:P of 1:1 to 1:2 (Hazelwinkel, 1989) but the red meat based diet contains calcium and phosphorus in the ratio of 1:10 to 1:50 (Pedersen, 1983; Badiani *et al*., 1997). As a corollary, these diets induce chronic hypocalcemia and increase the secretion of parathyroid hormone (PTH). This PTH goes in the circulation and tries to neutralize the excess inorganic phosphorus and normal blood calcium by eliminating phosphorus and calcium re-absorption through kidneys. It also increases the production of renal calcitrol (active form of vitamin D). Calcitrol increases the blood calcium by its absorption through GI tract andbone re-sorption. Continued feeding of animals on red meat based diets induces a state of sustained hyperparathyroid activity leading to appearance of signs attendant with hyperparathyroidism. Vitamin D deficiency can also play a minor role in etiopathogenesis of NSH (Reiland, 1978). Clinical signs in young animals are more severe than in adult ones and include lameness of various intensity, disinclination to move and muscular or skeletal pain. Swelling of costo-chondral junctions and pyrexia may be present. Persistent osteopenia increases liability to fractures at mild trauma and sometimes seizures may accompany (Tomsa *et al*., 1999). Osteopenia associated changes include generalized decrease in bone density, double cortical lining, thin cortices and occasionally subperiosteal re-sorption (Lamb, 1990; Johnson *et al*., 1991). As far as could be ascertained, description on NSH in wild captive felids is limited to reports of Herz and Kirberger (2004) and Won *et al*. (2004).

Case history: A three and a half months old female African lion (*Panthera leo*) cub weighing about 14kg was presented to Veterinary Teaching Hospital (VTH), Department of Clinical Medicine and Surgery, University of Agriculture, Faisalabad, Pakistan with presenting complaint of lethargy, lameness and pelvic limbsparesis. The signs had progressively increasedin the wake of 2 ft jump of the cub from bed about 1 month ago. Anamnesis revealed that cub was weaned at the age of 25 days when it was relocated to residence of its new guardian. She had been fed on goat meat and beef @ 7-10% of her body weight after weaning. Cardinal parameters of health were in normal range (temperature = 38.7ºC, respiration rate = 21 breaths/minute, heart rate = 40 beats/minute). Microscopic examination of thick and thin fecal smears was negative for endoparasites.

Musculoskeletal examination using palpation elicitedpain in hind limbs and hip joints.Hematological examination did not reveal any significant alteration (Table 1). Serum biochemistry revealed significantly raised value of alkaline phosphatase (ALP), slightly below normal level of calcium, however levels of inorganic phosphate and creatinine were normal (Table 2). Serum biochemical determination at the time of follow up visit 3 weeks post completion of treatment showed nearly 3 folds decrease in ALP, 0.5 fold increase in creatinine level and a slight increase in total calcium. Radiographic examination indicated a marked narrowing of pelvic area, increased marginal radiopacity of femur bones, femoral cupping and left proximal epiphyseal fracture of femur in ventral view (Fig. 1), osteopenia and decreased radiopacity of lumber and sacral vertebrae (Fig. 2).

Table 1: Summary of Hematological values of an African lion cub affected with nutritional secondary hyperparathyroidism as compared with reference values

Parameters	Values at presentation to VTH	Reference values*
Red blood cells (10^{12}/L)	7.60	5-10
Packed cell volume (L/L)	0.42	0.251-0.520
Hemoglobin(g/L)	122	44-230
Mean corpuscular volume (fL)	55.3	29.9-76.0
Mean corpuscular hemoglobin (pg)	16.1	11.2-27.2
Mean corpuscular hemoglobin concentration (g/L)	290	231-428
White blood cells (10^9/L)	9.7	5.5-19.5
Neutrophils (10^9/L)	7.00	0.000-8.690
Lymphocytes (10^9/L)	2.60	0.007-8.340
Monocytes (10^9/L)	0.30	0.000-2.912
Eosinophils (10^9/L)	0.10	0.000-1.575

*International Species Information System (ISIS) Physiological Reference Values for *Panthera leo.*

Table 2: Pre and post treatment serum biochemical parameters of an African lion cub affected with nutritional secondary hyperparathyroidism as compared with references values

Parameters	Values at presentation to VTH	Values 3 weeks post treatment**	Reference values*
Blood Urea Nitrogen (BUN) (mmol/L)	18.207	18.950	4.641-25.35
Serum creatinine (umol/L)	106	159	0-389
Serum alkaline phosphstase (U/l)	279	73	0-166
Inorganic phosphate (mmol/l)	2.85	2.52	0.84-2.91
Total calcium (mmol/l)	2.01	2.26	2.03-3.03

*International Species Information System (ISIS) Physiological Reference Values for *Panthera leo*; **Post treatment values taken after 3 weeks.

On the basis of nutritional plan, clinical findings and radiographic examination, a diagnosis of NSH was deduced. Treatment consisted of restriction of cub to a well lighted and ventilated cage, application of plaster of paris (gypsona bandage) on left hip joint to support the epiphyseal fracture which could be maintained only for 3 weeks. In view of nearly normal levels of serum calcium, the patient was treated with Calcium Carbonate @ 100 mg/kg PO (Tab. Calsan, Novartis Company) and Vitamin D @ 0.05 µg/kg PO (Inj. Indrop D, Neutro pharma Company) for 4 weeks. Improvement in clinical signs was observed at the follow up visit 3 weeks post-treatment.

DISCUSSION

Wild captive felids are prone to a variety of metabolic, nutritional and confinement associated disease conditions. The cub described in this case was maintainedon full meal diet of goat meat and beef since 2 months having predictable calcium content of approximately 6.6 mg/ 100 g and phosphorus content 290mg/100g (Nutritional composition of red meat, University of Wollongong), that clearly yields the Ca:P of 1:43. Early weaning probably also deprived the cub of its potential calcium source in diet. The cub exhibited the drastic signs of lameness, and reluctance to move after jump of 2 feet height. These signs may be provoked by osteopenia and bone liability to fracture as a result of consistent hypocalcemia. NSH is caused by chronic hypocalcaemia due to persistent deficiency of calcium in diet which leads to de-mineralization and excessive calcium re-sorption from bones making the bone more fragile to fractures and skeletal defects (Williams, 2007). In normal animals, only narrow limits of blood free calcium level is maintained by parathyroid hormone, as this free calcium in extra-cellular fluid is essential for enzyme, secretary and neuromuscular functions (Hazelwinkel, 1989). So by compensatory mechanism, increased level of parathyroid hormone will try to maintain normal calcium level despite severe downfall (Pedersen, 1983), as in the subject of this report calcium level was slightly below normal range initially, but recovered to normal following 3 weeks post-treatment. NSH can be seen with intermittent lameness and greenstick fracture (Herz *et al*., 2004), muscle twitching has beenobserved in a study (Tomsa *et al*., 1999), seizures, paralysis and constipation could be observed but not seen in this cub.Serum alkaline phosphatase (ALP) and phosphate values should interpreted carefully with the reference values as these are usually high in growing animals. Hypophosphataemia can also be seen with NSH as meat diets are actually low in phosphorus; however phosphorus relative to calcium is very high (Tomsa *et al*., 1999). Hyperphosphatemia in this case was not typical to NSH, but hypocalcemia and characteristics radiographic findings potentiated the diagnosis towards NSH as indicated by anamnesis. Serum creatinine, BUN, total protein was in normal ranges as indicated in Table 2. In electrolytes, serum sodium, potassium and chloride were all normal. Radiographically, loss of 30-50% bone mass is enough to express osteopaenia as decreased bone opacity, thin cortices, coarse trabeculation and fractures of long bones (Lamb *et al*., 1990). Marginal radiopacity, pathological epiphyseal fracture of left femur was also observed in this case study, however femoral cupping was a unique feature to this case which has not been previously reported in literature so far. Scapular cupping was observed as a rare finding in NSH (Herz *et al*., 2004). Pelvic collapse and osteodystrophic changes of lumbosacral vertebrae resulting in decreased radiopacity of these bones (Fig. 2). This cub was weaned at age of 1 month and was reared on meat diets, and showed symptoms at 3.5 months of age, whereas the cubs are totally dependent on mother until 3 months of age and should be weaned at 6-8 months (Mills and Bester, 2005). Moreover, the cubs are different to take care from domestic kittens and puppies because it is necessary to follow their proper weaning time and to keep this unsound creature in its natural environment. Increased parathyroid hormone was observed with NSH in cats (Crager and Nachreiner, 1993) but this hormone cannot be determined due to unavailability of same age control sample.

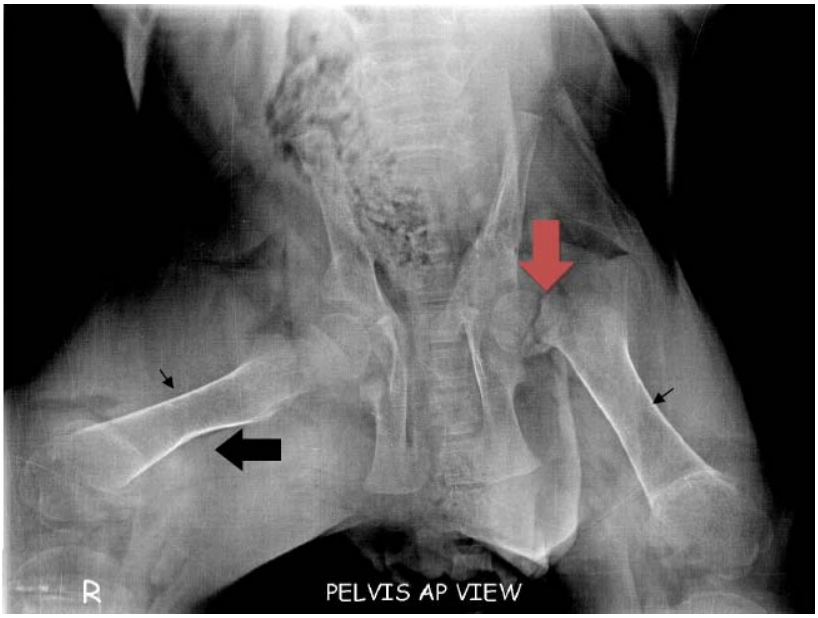

Fig. 1: Ventro-dorsal (VD) view of the pelvis of a cub affected with nutritional secondary hyperparathyroidism showing epiphyseal fracture (brown arrow), femoral cupping (black arrow) marginal radiopacity of right and left femurs (small arrows), with marked pelvic collapse.

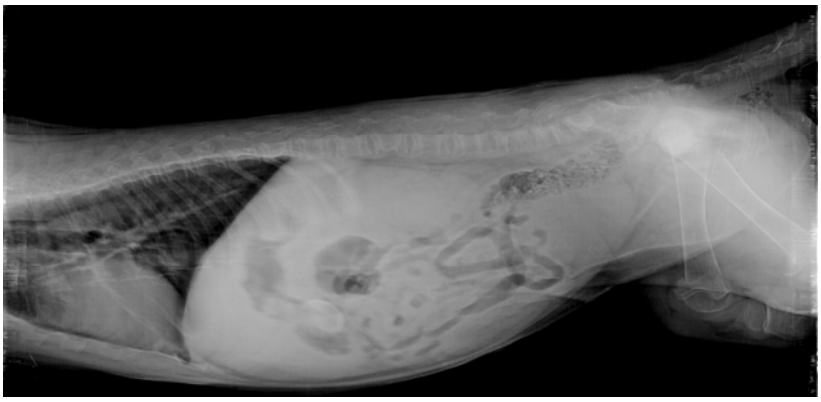

Fig. 2: Left lateral view of the body showing decreased radiopacity of vertebrae as of the soft tissues.

Conclusion: It can be concluded that NSH can be diagnosed by history and some characteristic radiographical findings alone, in addition, serum biochemical values may endorse the diagnosis. The authors believe that NSH is a common condition in captive wild species of lions due to several nutrition related problems. This mute creature can be saved from extinction by early diagnosis of this condition and efficient nutritional tools to overcome its occurrence.

REFERENCES

Badiani A, N Nanni, PP Gatta, B Tolomelli and M Manfredini, 1997. Nutrient profile of horsemeat. J Food Comp Anal, 10: 254-269.

Crager CS and RF Nachreiner, 1993. Increased parathyroid hormone concentration in a Siamese kitten with nutritional secondary hyperparathyroidism. J Am Anim Hosp Assoc, 29: 331-336.

Hazelwinkel HAW, 1989. Nutrition in relation to skeletal growth deformities. J Small Anim Pract, 30: 625-630.

Herz V and RM Kirberger, 2004. Nutritional secondary hyperparathyroidism in a white lion cub (*Panthera leo*), with concomitant radiographic double cortical line. J S Afr Vet Assoc, 75: 49-53.

Johnson KA and AD Watson, 2005. Skeletal Diseases. In: Textbook of Veterinary Internal Medicine (Ettinger SJ and EC Feldman, eds). 6th Ed: WB Saunders Co. Philadelphia, USA, pp: 1965-1991.

Lamb CR, 1990. The double cortical line: a sign of osteopenia. J Small Anim Pract, 31: 189-192.

Mills MG and MN Bester, 2005. Order carnivora. In: The Mammals of the Southern African Sub Region (Skinner JD and HN Smithers, eds). 2nd Ed, Struik Publishers, University of Pretoria, Pretoria, New Zealand, pp: 364-365.

Pedersen NC, 1983. Nutritional secondary hyperparathyroidism in a cattery associated with feeding of a fad diet containing horse meat. Feline Pract, 13: 19-26.

Reiland S, 1978. Effect of vitamin D and A, calcium, phosphorus and protein on frequency and severity of osteochondrosis in pigs. Acta Radiol Suppl, 358: 91-105.

Tomsa K, T Glaus, B Hauser, M Flückiger, P Arnold, G Wess and C Reusch, 1999. Nutritional secondary hyperparathyroidism in six cats. J Small Anim Pract, 40: 533-539.

Williams PG, 2007. Nutritional composition of red meat. Nutr Dietetics, 64(Suppl 4): S113-S119.

Won DS, C Park, YJ In and HM Park, 2004. A case of nutritional secondary hyperparathyroidism in a Siberian Tiger Cub. J Vet Med Sci, 66: 551-553.