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Nutritional secondary hyperparathyroidism and osteodystrophia fibrosa in a Hodgson's hawk-eagle (*Spizaetus nipalensis*)

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1 Introduction

Nutritional secondary hyperparathyroidism caused by long term deficiency of vitamin D₃, calcium or phosphorus, or by imbalances in the last two nutrients causes excessive production of parathyroid hormone (PTH), resulting in osteoclastic bone absorption (Woodard, [1997](#)). Rickets, which has the same pathogenesis, occurs in various kinds of young birds and mammals and is well described (Whitehead & Wilson, [1992](#); Thorp, [1994](#)). However, description of osteodystrophia fibrosa (fibrous osteodystrophy) in avian species is limited (Long et al., [1983](#)). Here, we describe the disease secondary to nutritional hyperparathyroidism in a Hodgson's hawk-eagle (*Spizaetus nipalensis*) bred for falconry.

2 Materials and Methods

Necropsy was performed and the liver, kidneys, heart, lungs, intestine, pancreas, adrenal glands, thyroid glands, parathyroid glands, sternum, femur, tibiotarsus and skeletal muscles were routinely fixed in 10% neutral buffered formalin, embedded in paraffin wax and stained with haematoxylin and eosin. Selected samples of the sternum, femur and tibiotarsus were decalcified by formic acid before embedding.

3 Results



decalcified and cortical bones in these regions had become thinner. The bird was surgically treated for these fractures by intramedullary pinning and given a vitamin B and calcium compound by oral administration. Biochemical examination eight days later revealed lower serum calcium (72.0 µg/ml) and phosphorus (29.0 µg/ml) and higher alkaline phosphatase (ALP) (3100 IU/l) than normal. Normal ranges of these inorganic substances or enzyme in raptors are 89.3–101.9 µg/ml (calcium), 30.3–43.4 µg/ml (phosphorus) and 31–257 IU/l (ALP), respectively (Samour, [2000](#)). The bird died ten days after the initial examination.

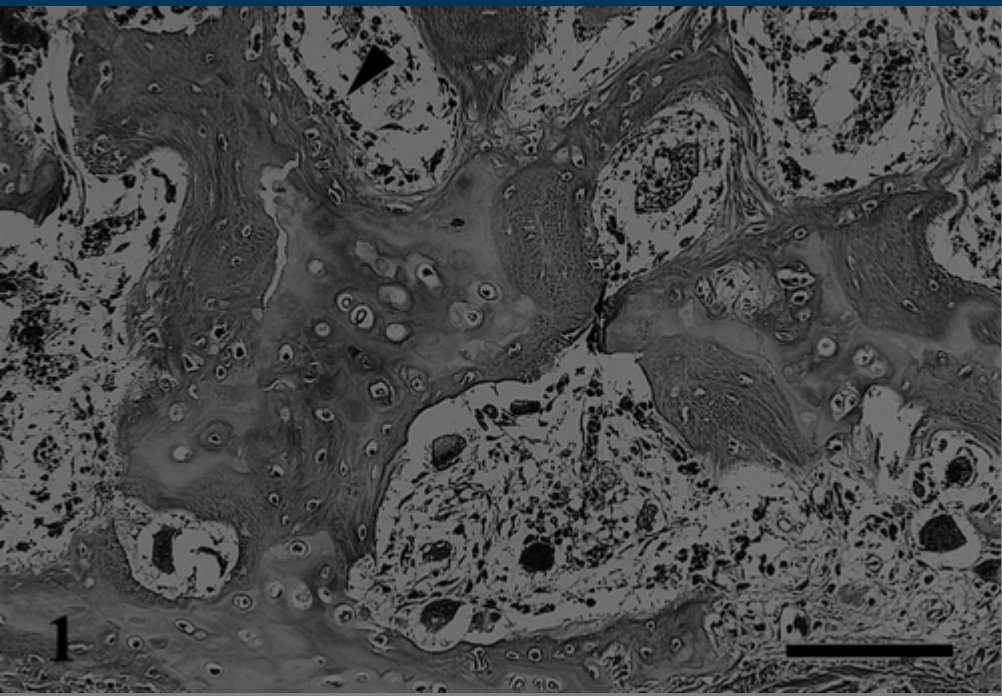
Gross examination confirmed that bone was very fragile with multiple complete and/or incomplete fractures. The parathyroid glands were bilaterally enlarged, up to 6 mm in diameter, and they were even larger than thyroid glands.

Histological changes of the affected bone mainly consisted of osteomalacic lesions and fractures. The former was characterized by a significant decrease in the amounts of mineralized trabecular bone associated with an increase in the width of unmineralized osteoid ([Fig. 1](#)). The osteoid borders were occasionally lined by osteoblasts and distinct osteoid seams were recognized. These changes occurred in the cortical and cancellous bone of the sternum, femur and tibiotarsus. The cortical bones of the diaphyses were also markedly thinned with dilation of the Haversian canals. The femur and tibiotarsus partly showed subperiosteal proliferation of trabeculae and loose fibrous tissue ([Fig. 2](#)).

In addition, the bone marrow was severely atrophic. The trabeculae of the bone marrow were very thin and the space between these trabeculae was very wide. Although there were some areas of necrotic bone of the bone marrow, the parathyroid glands were slightly enlarged and atrophic.

Fig. 1 Decalcified and stained section of the bone marrow showing unmineralized osteoid. Haematoxylin and eosin (H&E) stain, 100x magnification.





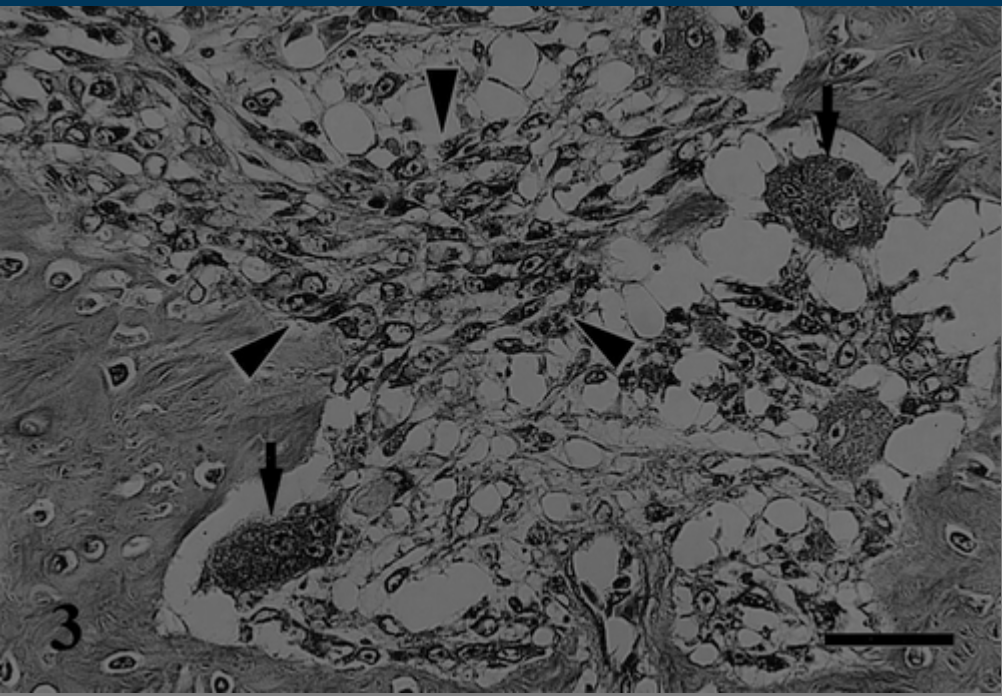
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Fig. 2 Decalcified section of the tibiotarsus. Marked periosteal bone reaction with proliferation of osteoid trabeculae and loose fibrous tissue at the exterior of thinning cortex bone (arrowheads). HE. Bar=500 μ m.



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Fig. 3 Decalcified section of the tibiotarsus. Marked periosteal bone reaction with proliferation of osteoid trabeculae and loose fibrous tissue at the exterior of thinning cortex bone (arrowheads). HE. Bar=500 μ m.



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Fig. 4 Parathyroid gland showing proliferation of chief cells with cytoplasmic swelling and vacuolation. HE. Bar=50 μ m.



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Other histological features include moderate interstitial fibrosis.

renal cortex, myocardial

Osteodystrophia fibrosa is the result of continuous and excessive secretion of PTH and is characterized by marked osteoclastic resorption and fibrous replacement (Palmer, [1991](#)). Locomotory disorders with fractures in birds of prey including those for falconry, have been reported as "cramps" or "fits"; protracted dietary deficiency of calcium or imbalance of calcium/phosphorus ratio has been considered important causative factors (Wallach & Flieg, [1970](#); Cooper, [1975](#)). However, pathological descriptions of osteodystrophia fibrosa due to nutritional secondary hyperparathyroidism have rarely been reported in avian species (Long et al., [1983](#)).

The present case was considered nutritional secondary hyperparathyroidism that resulted in severe osteodystrophia fibrosa. The active osteoclastic bone resorption and fibrous tissue proliferation between trabeculae in the examined bones are characteristic findings of osteodystrophia fibrosa. On the other hand, accumulation of osteoid and formation of osteoid seams in the medullary cavity are specific features of osteomalacia, which frequently progresses to osteodystrophia fibrosa when severe hyperparathyroidism develops in the course of the disease (Woodard, [1997](#)). Compensating subperiosteal bone proliferation occurs in some cases of osteopenia (Riddell, [1996](#)). However, subperiosteal proliferation of osteoid and fibrous tissues in the present case was considered as unmineralized reactive callus resulting from microscopic fractures, because it was observed only in part of the femur and tibiotarsus.

The calcium range from 1.5 to 2.0 mmol/L in these diets may be due to an abnormal parathyroid hormone secretion. Hyperparathyroidism is characterized by osteodystrophia fibrosa. Although the type of birds, including the type of prey, is not stated as 1.5:1 (Wallach & Flieg, 1970) in these diets, in addition to the calcium of the diets, the type of prey is important for the calcium balance. In addition, the type of prey is important for the calcium balance. In addition, the type of prey is important for the calcium balance.



Acknowledgments

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