



Nutritional Secondary Hyperparathyroidism in A Cat

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Abstract: This case report was aimed to provide information about nutritional secondary hyperparathyroidism diagnosed in a cat. The case material consisted of a 1-year-old, tabby breed female cat who was a weakness, inability to stand up and not to walk. According to the anamnesis information, it was learned that the appetite of the animal was good, but its nutrition consisted only of foods with high phosphorus content such as salami, sausage, and meat. Radiological examination revealed lordosis in the cervical spine, kyphosis in the lumbar spine, and a small amount of free fluid in the abdomen. The hematologic examination was normal. It was also suggested that meat and meat products such as salami and sausages should not be included in the diet. Kyphosis and lordosis were found to be permanent. As a result, it is concluded that the possibility of nutritional secondary hyperparathyroidism should be evaluated in cats who were fed diets high in phosphorus and with fatigue, inability to stand up, and unable to walk.

Keywords: Calcium, Cat, Nutritional hyperparathyroidism, Phosphorus.

INTRODUCTION

Nutritional secondary hyperparathyroidism (NSH) is a metabolic disease characterized by bone resorption and development of osteopenia as a result of feeding cats and dogs with diets with incorrect and unbalanced phosphorus/calcium (Ca/P) ratio (1, 2). Insufficient vitamin D3, excessive P, and/or insufficient Ca in the diets consumed are responsible for the formation of the disease. The widespread use of commercial formulas in recent years has reduced the incidence of the disease (3, 4).

For bone health, the ideal Ca/P ratio in the diet should be 1/1 or 1/2. If the P ratio of this balance is disturbed, there is an increase in the secretion of parathormone (PTH), and as a result, hyperparathyroidism is formed. Ca is released from the bones and P is excreted from the kidneys due to hyperparathyroidism. Subsequently, Ca retention occurs in the kidneys. As this situation continues, osteopenias and bone fractures occur in animals due to excessive bone resorption (4). Symptoms of the disease include constipation, weakness, limping,

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muscle and bone pain on palpation, inability to walk, and sometimes even bone fractures (5-7).

In the diagnosis of the disease, it is reported that calcium and phosphorus levels are normal or decreased, and blood PTH, and vitamin D levels are increased (4,8,9). In the radiographic diagnosis of the disease, it is stated that there is an increase in opacity in the bones forming the skeleton, postural disorder of the spinal cord, pelvic deformation, thinning of the bone cortices, trabeculation in the bones, and bone fractures (4, 10).

Treatment of the disease requires a complete, and balanced diet. For hypocalcemia, parenteral, and oral calcium supplements can be administered. Cage rest can be applied to reduce the risk of bone deformity, and prevent the development of fractures. For animals with fractures or bone pains, analgesia may be required. Ionized light therapy can be applied for bone growth (1).

This case report, it is aimed to present the clinical, radiographic, and laboratory findings of a rare cat with NSH.

CASE PRESENTATION

The case material consisted of a 1-year-old, 2,5 kg, tabby, the female cat was brought to the animal hospital of the Veterinary Faculty of Atatürk University. In the anamnesis, it was learned that the patient had complaints of constipation, weakness, inability to stand up, and walk and that her appetite was good. In addition, in the anamnesis, it was learned that the diet of the cat only consisted of foods with high phosphorus content such as meat,

and meat products such as salami, sausages. Pulse on clinical examination (200/dk), respiratory (64/dk), and body temperature (38.2°C) were detected. Blood samples from the cat were taken from vena cephalica antebrachial and collected into EDTA vacutainers (Vacutainer, K2E 3,6 mg, BD, UK) and plain tubes (Vacutainer, BD, UK) for hematological and biochemical analyses. After leaving for ten minutes at room temperature for clotting, sera were obtained by centrifugation (Beckman Coulter, Allegra® X-30R, USA) at 3000 rpm for 10 minutes and serum sample, the concentration parameters by biochemistry autoanalyzer (Beckman Coulter, AU5800, USA). The hematological analysis was determined by a hematology analyzer (Abacus Junior Vet5, Hungary).

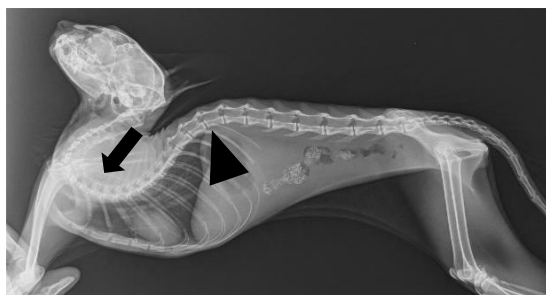
The data of hematological, and biochemical examinations are presented in Table 1. While no abnormality was detected in the hemogram results, an increase in Ca, and P levels were detected in biochemical analyzes.

Table 1. Biochemical findings of the case.

Parameter	Result	Reference Values (11)
WBC	16.95x10 ³ /μL	5.5-19.5x10 ³ /μL
Ca	11.87 mg/dl	9.4-11.4 mg/dl
P	5.84 mg/dl	2.5-5 mg/dl
Mg	2.48 mg/dl	1.1-2.4 mg/dl
Vitamin D (25 OH)	22.04 ng/ml	14.9-61 ng/ml

WBC: White blood cell, Ca: Calcium, P: Phosphorus, Mg: magnesium

Figures 1 and 2 of the radiographic examination are given. Lordosis of the vertebrae as a result of radiographic examination, and kyphosis image; humerus, radius, ulna, costa, and medial parts of vertebral bones decrease in opacity was detected.

Figures 1 and 2. Radiographic findings of the case.**Figure 1.** Lordosis (black arrow), and kyphosis (black arrowhead) (Black arrowhead in the vertebral vertebrae).**Figure 2.** Reduction in radiopacity of humerus, radius, ulna, costa, and vertebral vertebrae (black arrow).

The patient was diagnosed with NSH by the anamnesis information obtained from the patient, the high Ca, and P levels detected, and the radiographic examination images. Informed consent form was obtained. At a dose of 50-60 ml/kg/day for 7 days in treatment calcium gluconate at a dose of 95-140 mg/kg was added to 0.9% NaCl and administered slowly intravenously. In addition, furosemide was administered at a dose of 2.5-5 ml/10 kg to accelerate the excretion of P from highs. Afterward clinical evaluations, it was recommended to switch to commercial food with balanced Ca/P content. Serum Ca level was measured again 3 days after the start of the treatment, and it was seen that it regressed to the reference values. However, it was concluded that the formed lordosis and kyphosis may be irreversible due to the severe calcium release.

DISCUSSION and CONCLUSION

Nutritional secondary hyperparathyroidism is a disease that begins with calcium deficiency in the diet and continues with associated hypocalcemia. The response of the organism to the formed hypocalcemia is excessive Ca mobilization from the bones to the blood. This situation leads to demineralization of bones, skeletal defects, and fractures (1, 12). It is stated that there may be a decrease in vitamin D levels in the diagnosis of the disease (12, 13). The Ca/P ratio changes depending on the excess phosphorus intake in the diet. Increased parathyroid activity tends to normalize

calcium and inorganic phosphate in the blood. It causes mineral mobilization from bones. Diets with a Ca/P ratio of 1/2 are recommended for treatment. In line with clinical findings, symptomatic treatment, and bone fractures, if any, should be repaired. Vitamin D and calcium supplements should be administered (10).

The increase in osteopenia decreased bone opacity in the humerus, radius, and ulna, and osteodystrophy, and thin cortex findings in the vertebral vertebrae seen in the radiographs of our case overlap with the radiographic images of other cases (14). Therefore, the diagnosis of NSH was strengthened in our case. Anamnesis information obtained from the patient, constipation from clinical symptoms, and radiographic examination images were associated with the mobilization of Ca from the bone under the influence of PTH. Our case Graham et al. (15) coincides with the phenomenon described by (4, 10).

The hypercalcemia detected in our case is not typical for NSH, and the Ca level was higher than expected in NSH. Similarly, in another case with hyperparathyroidism, blood Ca level was found to be higher than normal values. This was associated with an increase in ionized calcium in total serum calcium. This increase was thought to be caused by hyperparathyroidism (10). Diets high in phosphorus cause the organism's P balance to deteriorate, increase PTH secretion, and increase Ca release. The increase in Ca, and P levels, in this case, can be

explained by the above mechanisms. In cases of NSH, PTH secretion increases due to the hemostasis of the organism due to insufficient Ca intake in the diet. PTH levels were found to be high in 6 cats diagnosed with NSH (1, 4, 9). PTH level could not be measured. Because no cat-specific kit was used for PTH analysis. This is a shortcoming of the study. However, it is understood that the case is NSH based on the anamnesis, clinical, and radiographic findings, and other biochemical parameters (4, 16). On the other hand, a severe decrease in serum Ca level in NSH has not been reported in every case (10). In another reported case of a cat with NSH, serum calcium, and elevated values are between reference values. It has been stated that this situation may also be caused by nutrition (15).

The interaction of Mg, and Ca in the organism is complex and, Mg and Ca are in balance in the organism. The hypermagnesemia detected in our case is explained by this balance (14). In a few cases of secondary hyperparathyroidism, the serum magnesium level did not differ from the serum Mg levels of healthy cats. Therefore, it was stated that magnesium results are not an important marker in the diagnosis of NSH (4, 17).

In the biochemical examination, it was determined that the phosphorus level was above the reference values. Rowland et al. found elevated serum phosphorus levels in a cat with NSH and reported that serum phosphorus levels tended to be elevated in animals whose diet consisted of mostly meat products (17). Similarly, in the anamnesis information obtained from the owner of the cat, which constitutes the case material, the fact that the diet consisted mostly of meat products was thought to be the reason for the increase in serum P levels. Despite the widespread use of commercially formulated pet diets, pet owners sometimes do not feed their cats these foods. Therefore, nutritional-related bone diseases are encountered. As a result, those fed with diets that do not have sufficient calcium/phosphorus ratio such as salami and sausage. It was concluded that NSH should also be

evaluated in cats with complaints of weakness, inability to stand up, and walk. It was concluded that this situation should be supported by radiographic images and serum biochemical values.

Conflict of Interest

The authors declare that they have no conflict of interest.

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