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Low Calcium Rickets in Young Cats

by
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Johnny Hoskins†

INTRODUCTION

Due to the low calcium content of meat and meat by-products, nutritional secondary hyperparathyroidism and/or low calcium rickets can be a severe problem in domestic carnivores maintained on diets which are not balanced in calcium: phosphorus ratio. This type of diet results in poor mineralization of bones in young animals or excess bone resorption in older animals maintained on an improperly balanced diet.

Calcium, phosphorus, and vitamin D are all essential to proper bone growth and maintenance. All three are closely linked in their metabolism in the animal body. To prevent metabolic bone diseases it is essential that all three of these nutrients are maintained in a proper balance in the diet.

CASE REPORT

Clinical Findings—A 3½ month old male domestic cat was presented to the clinic in July, 1975. The cat had a history of lameness, constipation, swollen abdomen, and generalized body pain of approximately three weeks duration. The cat was on a diet consisting of Gerber high protein cereal, very small amounts of commercial cat food, and raw ground beef. Consultation with the owner revealed, however, that the animal refused the cereal and commercial food and ate almost entirely a ground beef diet.

On physical exam the animal showed generalized body pain on palpation, especially in the limbs and joints. The abdomen was distended and the animal had difficulty standing upright on its hind limbs. When the animal was placed in a standing position both hind limbs splayed out laterally.

Radiographic Findings—Lateral and ventral-dorsal scout films were taken and these showed very thin cortices in all the long bones and poor mineralization of bones. There was a sharp deviation of the spinal column at T10-T11 indicating a possible compression fracture at this location. There was a folding fracture on the distal end of one femur, the sternum was deformed dorsally, there was ventral lordosis of the lumbar spine, and the ulnar and fibular tarsal bones were curved abnormally. The pelvic area was very hard to delineate but appeared compressed and small for this age animal. In addition the long bones lacked the normal trabecular pattern which should be seen with proper mineralization of bone.

Blood Parameters—Blood profile was unremarkable except for an elevated serum phosphorus level of 7.7 mg%. Blood calcium was within the normal limits at 9.4 mg%.5

Based on the case history and the above clinical findings a tentative diagnosis of nutritional secondary hyperparathyroidism was made. Due to the many fractures seen radiographically and the possibility of a severe fracture of the spinal column, the

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owners decided to allow euthanization of the animal. The animal was kept in a ward for approximately two weeks and placed on a commercial canned diet during this period. During this time the animal experienced remission of some clinical signs including its pain and inability to walk. At the end of two weeks the animal was euthanized and a post mortem examination performed.

**Pathological Findings—Gross:** Major organs contained no gross lesions. The thyroids and parathyroids were normal in size and contained no gross lesions. Other systems of the body contained no gross lesions with the exception of the skeletal system. Examination of the ribs revealed seventeen pathological fractures at various locations throughout the rib cage. These fractures appeared to have well formed calluses around them. Pathological fractures were also seen in the right distal femur and the left proximal tibia, and both of these had well formed calluses. A compression fracture was found at thoracic vertebrae T10 and T11. No other gross lesions were observed.

**Histopathology:** Sections through the parathyroids revealed only mild hyper trophy of the chief cells. This was indicated by a small increase in the cytoplasmic content of these cells. No marked hyper trophy of the parathyroids was noted.

Sections through the fracture sights showed well formed cartilaginous calluses and good osteoid formation. However, not much evidence was seen to indicate new bone formation and bone remodeling at the fracture sights. In one sight it was noted that blood vessels had begun to invade the osteoid matrix in a possible attempt at mineralization. It was hypothesized that this beginning of new bone growth probably occurred after the animal was placed on a balanced diet in the clinic.

Based on the pathological findings, the final diagnosis was low calcium rickets.

**DISCUSSION**

It is relatively easy for the clinician to see the radiographic changes indicating poor mineralization of bones. The major problem facing the clinician is one of proper diagnosis and subsequent treatment of one of the many conditions which can cause these changes in bone. To do this one needs a good basic knowledge of calcium and phosphorus metabolism in relation to bone growth.

Ionized calcium is absorbed primarily in the ileum. This absorption process is markedly enhanced by the presence of vitamin D. Calcium absorption is markedly reduced when high fat levels are present in the intestine because insoluble calcium soaps are formed and the calcium is then lost in the feces. Severe gastroenteritis can also cause decreased absorption of calcium ions.

The normal excretory pathway of calcium is via the gastrointestinal tract in the feces. The kidneys excrete very little calcium but it is a threshold substance and in chronic hypercalcemia the transport maximum can be exceeded and calcium spills over into the urine in sufficient amounts to cause renal calculi. Normally all calcium is reabsorbed by the kidney tubules but in disease conditions in which bone is being resorbed decreased amounts of calcium are returned to the blood.

Serum calcium and phosphorus concentrations are normally inversely related but in disease conditions both may be elevated or lowered.

The two hormones, parathormone and calcitonin, along with vitamin D, are primarily responsible for controlling blood levels of calcium and phosphorus.

Parathormone acts in the kidneys, the skeleton, and the gastrointestinal tract. Its secretion is controlled primarily by calcium ion concentration in the blood.

In the kidneys parathormone binds with cells and causes increased tubular reabsorption of calcium and enhances excretion of potassium, phosphorus, and bicarbonate. Parathormone also increases the capacity of the kidney to produce active vitamin D, which aids calcium absorption from the gut and increases calcium mobilization from bone.

Parathormone acts on the gastrointestinal tract indirectly through vitamin D to increase calcium absorption through the ileal mucosa.

Parathormone acts on bone at the cellular level to decrease bone binding capacity for calcium and increase osteoclastic activity
which results in erosion of well calcified bone.

Calcitonin acts primarily on the bone and secondarily in the kidney. Calcitonin inhibits bone resorption and decreases loss of calcium from bone with a resulting hypocalcemia and hypophosphatemia. It also accelerates calcium and phosphorus removal from blood.

As previously mentioned, vitamin D enhances calcium absorption from the intestine, but it should also be noted that excess vitamin D intake can cause bone resorption of calcium and phosphorus with a resulting overflow of calcium into the kidney causing failure due to formation of renal calculi.

There are several diseases associated with calcium and phosphorus metabolism which the clinician must include in his differential diagnosis when poor bone mineralization is diagnosed radiographically.

Primary hyperparathyroidism due to a parathyroid neoplasm is extremely rare. It has been reported only a few times in the dog and never in the cat. This disease can be diagnosed by a progressive increase in blood calcium and decrease in blood phosphorus levels. The only treatment is to surgically remove the affected parathyroid gland.2,8

Rickets can be caused by several things including a low calcium diet, low phosphorus diet, low vitamin D intake, a chronic gastroenteritis preventing adequate absorption of calcium, or by excess fat in the diet resulting in the formation of insoluble calcium salts.3,6 Diagnosis of rickets requires a careful analysis of the diet of the affected animal. In addition, blood profile can be helpful. In low phosphorus rickets serum phosphorus will be low and serum calcium is usually normal.2,8 Low calcium rickets is usually always accompanied by nutritional secondary hyperparathyroidism because the low calcium levels in the blood will stimulate parathormone secretion. Thus, the hypocalcemia and hyperphosphatemia one would expect with this disease can readily be compensated by increased resorption of calcium from bone and by increased excretion of phosphorus through the kidneys. In this disease blood values for these two nutrients can be very misleading. Treatment for the condition consists of dietary correction of the imbalance and/or correcting the gastroenteritis or fat intake in the diet.

Renal secondary hyperparathyroidism is caused by a chronic renal insufficiency which prevents normal calcium reabsorption in the tubules and stops excretion of phosphorus.3,4 The phosphorus builds up in the blood and consequently blood calcium is lowered and the parathyroids are stimulated. In diagnosis blood values are helpful and the clinician will see a high phosphorus which is not compensated because of the kidneys decreased ability to excrete phosphorus.4 However, parathormone activity may compensate the low calcium levels and blood calcium may be normal. Treatment is primarily aimed at the renal problem first.

Nutritional secondary hyperparathyroidism can be caused by a low calcium intake in the diet, a calcium-phosphorus imbalance with excessive phosphorus, or by a low vitamin D3 intake.3 Diagnosis of this disease also requires a careful analysis of the diet and monitoring of blood calcium and phosphorus. The clinician must be careful in relying too much on blood levels in this disease because hypocalcemia and hyperphosphatemia are readily compensated by increased bone resorption and increased excretion of phosphates. Treatment consists of putting the animal on a diet with a calcium: phosphorus ratio of 1.5 to 1. Use of a good commercial diet is recommended for young animals and a mineral supplemented beef diet for older animals who will not accept a commercial diet.

Several precautions should be followed when treating metabolic bone diseases.5

1. Steroids are contraindicated in all cases due to their detrimental effects on osteoblasts. They also cause necrosis of osteocytes in bone.

2. Generalized therapy with minerals and vitamins in large doses should be avoided until the specific cause of the problem is identified.

3. Only one analysis of blood for mineral levels is not sufficient for diagnosis of metabolic bone diseases. Several
analyses should be done on successive days to establish a trend in serum levels of calcium and phosphorus.

REFERENCES


Eperythrozoonosis In Swine

by
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Eperythrozoonosis is an "acute febrile, icteroanemic disease of shoats" caused by the rickettsial organism *Eperythrozoon spp.* There are two species which are commonly associated with swine, *E. suis*, which produces a febrile disease, and *E. parvum*, which is innocuous. Other common names for eperythrozoonosis include epy, swine icteroanemia, yellow belly, and anaplasmosis-like disease.

*E. suis* organisms are relatively large, coccoid bodies (average size of 0.8μ in diameter) found attached to RBC's and also free in the plasma. Transmission of the organism occurs via several methods: 1) bloodsucking ectoparasites especially mange (*Sarcoptes scabiei var suis*) and lice (*Hematopinus suis*), 2) mechanical transmission (needles, ear notching, tail docking, castration, etc.), and 3) in utero transmission.

In utero transmission was shown to exist by an experiment conducted by Dr. A. R. Smith at the University of Illinois. A bred gilt which had been serologically positive (titer 40) for at least 5 months previously was purchased and her pigs were delivered via cesarian section (germ free). The pigs, therefore, had no contact with the sow or other infected pigs. Out of a litter of 13 pigs, parasites were observed in the blood of all the pigs during the first week. Wright-Giemsa stained blood smears were made to confirm the infection. Three of the 13 died and all dead and moribund pigs had hemoglobins of 1.9-3.0 g/100 ml. and PCV's of 10.5-12.5 g/100 ml.

Most parasites in swine don't change the blood picture much, *E. suis* being an exception. The total red count may drop as low as 1-2 million cells/cubic mm. (normal is 7 million cells/cubic mm.), thus creating a severe anemia and icterus. Hemoglobin levels decrease to 2-4 gm. (normal is 13 gm.) and the bone marrow becomes hyperplastic while total white count increases slightly or remains the same.

Incubation time for the disease is approximately 6-10 days. Increased temperature of 104° to 107°, anorexia, depression, and severe and rapid blood destruction with a subsequent decrease in the numbers of parasites are characteristic clinical signs of epy. Later in the course of the disease icterus, polyneia, weakness, and bile stained feces are observed.

On posting a diseased animal, icterus, yellow liver and a soft, enlarged spleen are