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JUVENILE OSTEOMALACIA IN A COYOTE*

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Abstract: Juvenile osteomalacia was diagnosed in a 1-month-old coyote puppy *(Canis latrans incolatus)* reared on an artificial diet. Hematologic and radiographic findings during the course of the disease are presented. A successful treatment regime consisting of administration of parenteral and oral calcium and supportive treatment is outlined.

Osteomalacia, in most instances, is caused by inadequate amounts of calcium in the diet, improper absorption of calcium from the intestinal tract, insufficient vitamin D, deficiency of phosphorus, or a severely unbalanced calcium-phosphorus ratio. In adult animals, renal disease, lactation, and excesses of certain minerals can cause osteomalacia.[3] Young animals are particularly susceptible because of the demands for large amounts of calcium during the period of rapid bone growth. The pathologic changes that occurred in the young coyote *(Canis latrans incolatus)* presented in this report were similar to those that occur in foxes fed a high meat diet, with the exception that osteodystrophia fibrosa did not develop.[2]

CASE REPORT

A female coyote maintained in captivity for 4 years at the Institute of Arctic Biology, University of Alaska, gave birth to five young on April 28, 1971. Immediately after parturition she ate three of the five pups; the remaining two were removed from her pen. One of the surviving pups died during a fostering attempt with a lactating Beagle. The surviving male pup was given an artificial diet and is the subject of this report.

During the first week of life, the newborn coyote consumed approximately 75 ml daily of a formula that consisted of 120 ml of evaporated milk, 120 ml of water, one egg yolk, and 15 ml of a saturated sugar solution. This mixture was offered at 3 hour intervals. There was a weight gain from 220 gm to 323 gm during the first 10 days of life. Rapid weight gains continued until the pup was 21 days old (Fig. 1), at which time he weighed 676 gm and was consuming 90

1. Coyote puppy at 33 days of age with posterior paralysis.

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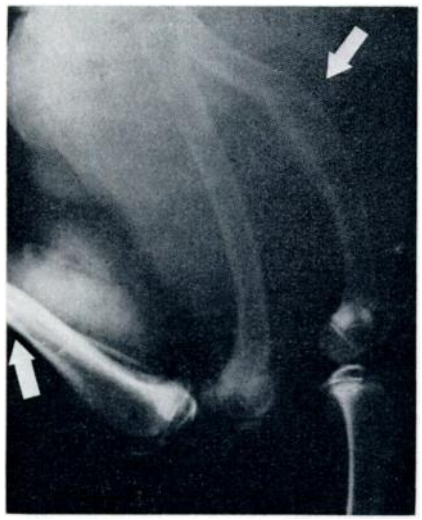
ml of formula daily, during four feedings. At this time a meat and creamed rice mixture was added to the diet and at 30 days of age the pup weighed 805 gm. Sporadically, during this period, the feces were of a watery consistency.

At 33 days of age signs of a slight ataxia were observed that progressed to complete posterior paralysis. At that time body temperature was normal, and the packed cell volume (PCV) was 23%, a value that is below normal for coyote puppies.[1] The white blood cell (WBC) count was 15,500 per mm^3; however, the relative numbers of the white cells were normal (segmented neutrophils 58%, nonsegmented neutrophils 2%, lymphocytes 38%, and eosinophils 2%). Radiographs of the entire body revealed very limited calcification of all bones, especially the cortices of the diaphyseal portion of the long bones, which were reduced in thickness. Treatment was initiated at this time by administering 2.5 ml of calcium glycerophosphate,[1] 0.5 ml of a hematinic,[2] and 0.5 ml of a preparation composed of procaine penicillin G, dihydrostreptomycin sulfate, dexamethasone, and chlorpheniramine maleate.[3] The calcium glycerophosphate was continued daily for 6 days at which time a second radiograph was taken (Fig. 2). These radiographs revealed two fracture sites, one located at the midshaft of the right femur, the other, midshaft fractures of the left tibia and fibula. After 6 days of treatment the pup was again walking normally despite the presence of fractures. He continued to improve and was administered bone meal in the feed as a calcium supplement. At 40 days of age the PCV was 31%, the WBC count was 8,650 per mm^3, and the relative numbers of the white cells remained within normal limits (segmented neutrophils 62%, nonsegmented neutrophils 3%, lymphocytes 29%, monocytes 1%, and eosinophils 4%). Serum electrolytes were as follows: sodium 343 meq/1, potassium 6.32 meq/1, calcium 5.90 meq/1, and magnesium 1.61 meq/1.

2. Radiograph of fracture sites in right femur and left tibia and fibula (arrows). Notice thinness of bone cortices.

[1] Calphosan, Carlton Corp., Tenafly, N.J.
[2] Liviron, Trico Pharmaceutical Co., San Francisco, Calif.
[3] Azimycin, Schering Corp., Bloomfield, N.J.

The pup gained weight rapidly after treatment and at 60 days of age weighed 2,152 gm. By 90 days of age he weighed 3,400 gm. At this time due to a change of personnel the bone meal was mistakingly omitted from his diet and within ten days tremors and a generalized myalgia developed. Calcium glycerophosphate was administered for 2 days and bone meal was again added to the diet. At the end of the second day all signs of tremors and myalgia had disappeared. At this time the PCV was 36%, WBC was 8,650 mm^3, with little change in the relative numbers of white cells (segmented neutrophils 60%, nonsegmented neutrophils 8%, lymphocytes 27%, and eosinophils 5%). Serum electrolytes were: sodium 150 meq/l, potassium 5.33 meq/l, calcium 5.70 meq/l, and magnesium 1.65 meq/l. The coyote is presently 130 days old and has had no further clinical signs of illness. Radiographs taken at 125 days of age revealed normal bone density and healed fracture sites; however, the right femur was shorter than the left and was slightly curved laterally (Fig. 3). His weight at 130 days of age was 8,630 gm.



3. Radiograph, taken 3 months after radiograph shown in Fig. 2, of healed fracture sites (arrows). Thickness of bone cortices is near normal.

DISCUSSION

The symptoms and lesions described in this case report are typical of those seen in domestic carnivores being fed calcium deficient diets. Personal experience in feeding the same diet as given the coyote pup to orphaned domestic dogs and cats on numerous occasions has not led to the development of juvenile osteomalacia. Clinical examination of other young wild carnivores (wolves and foxes) raised in captivity on various diets not supplemented with calcium has revealed lesions common to osteomalacia. These observations emphasize the importance of diet supplements when raising wild animals in captivity on artificial diets.

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