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JUVENILE RICKETS AND HYPERPARATHYROIDISM IN THE ARCTIC FOX [1]

GERALD J. CONLOGUE,[2] [3] WILLIAM J. FOREYT,[3] ALFE L. HANSON, and JOHN A. OGDEN [2]

Abstract: Three of seven Arctic fox kits, *Alopex lagopus,* trapped on St. Paul Island, Alaska, had evidence of rickets and hyperparathyroidism. Radiographic, morphologic and histologic examination confirmed the diagnosis. The disease was presumed to be a juvenile-onset disease due to calcium-deficient intake following weaning. The possibility of insufficient exposure to sunlight could not be determined. No other abnormalities (e.g., genitourinary) were found. Skeletal involvement was quite varied. All growth plates exhibited columnar hyperplasia compatible with rickets, while the metaphyses showed decreased trabeculation and cortical thinning compatible with osteodystrophia fibrosa. One fox had bilateral metaphyseal fractures through this osteodystrophic bone.

INTRODUCTION

While considerable experimental research in the various types of rickets and osteodystrophy has been conducted in small laboratory animals such as mice and rats, very little information is available in larger mammals. Certainly such data would be helpful in furthering the understanding of the disease, especially as compared to human rickets. Normal and abnormal patterns of bone growth by endochondral and membranous formation differ in smaller, faster-maturing species and may be misleading compared to the slower maturation rates and different ossification/remodeling patterns in large-boned mammals.[7]

Experimental rickets has been reported in captive foxes fed a high horsemeat ration,[3] and in a coyote pup reared on an artificial diet.[2] Dämmrich[1] described combined rickets and osteodystrophy in calves, goats, pigs, lions, apes, dogs and cats reared on artificial diets. He felt differing skeletal responses were due to species and age-related patterns of skeletal biology.

The following report appears to be the first concerning wild animals discovered to have spontaneous, presumably dietary, rickets.

CASE MATERIAL

Seven Arctic fox kits, *Alopex lagopus,* were live-trapped using Tomahawk traps on St. Paul Island, Alaska, from June to August, 1978. Blood and fecal samples were obtained from all the kits. Three had obvious limb deformities and were radiographed. One was released, but kits 3 and 5 died shortly following capture. The latter kit (5) was examined at necropsy and the skeleton fixed in 10% formalin for later examination. This kit, which certainly was representative of all three involved animals, will be described in greater detail.

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Kit 5, a 1268 g male, was initially observed dragging its hind legs while crossing the road near Sea Lion Neck on St. Paul Island,[1] August, 1978. He was trapped shortly thereafter. Initial examination revealed a prolapsed rectum infested with maggots. Both front legs demonstrated hyperextension and ulnar deviation at the carpal joint. Both proximal tibiae exhibited palpable fractures, and the physeal regions of the distal radius, ulna and distal tibia were palpably enlarged bilaterally. Roentgenograms taken at the Island hospital demonstrated bowing of the femora, humeri, tibiae and fibulae. Both proximal tibiae had recent fractures, with no evidence of fracture callus while the fibulae were bowed (Fig. 1). The distal ulnae were shortened, with markedly widened physes, and concomitant radial bowing.

At necropsy all organs, with the exception of the parathyroids, were grossly

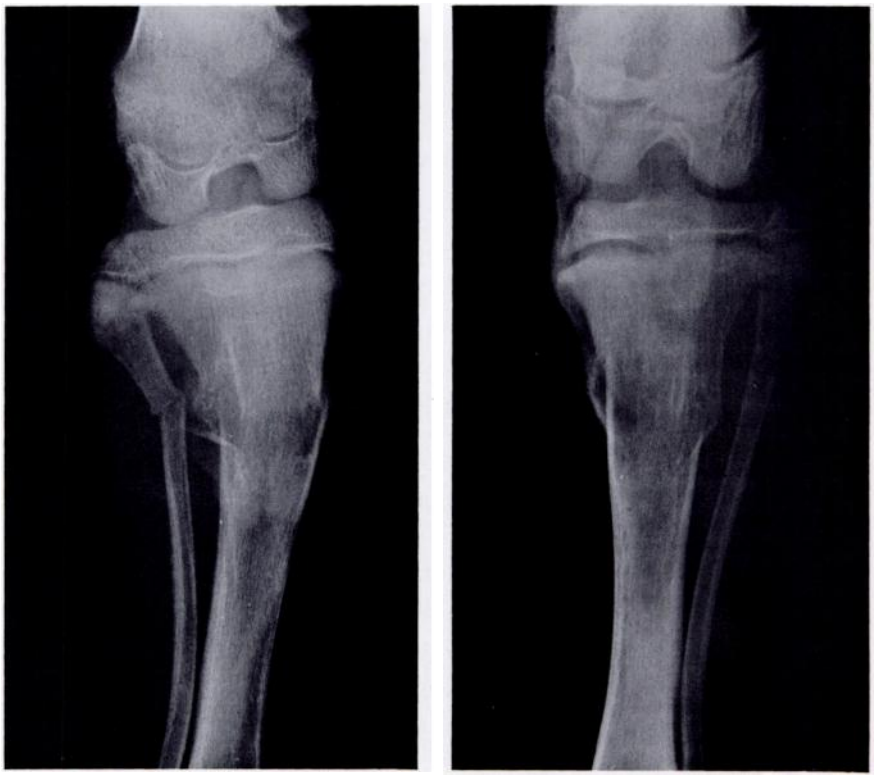


FIGURE 1: Right (A) and left (B) tibiofibular units showing recent metaphyseal fractures. On the right tensile failure has led to disruption of cortical integrity of both the tibia and fibula, with production of a valgus deformity. In contrast, on the left the tibial cortex is intact, having sustained a compression pattern of failure manifest as a torus (buckle) fracture, while the fibula has undergone plastic deformation (bowing) without cortical disruption. This has produced a mild varus deformity on this side. The valgus/varus combination is not unlike the characteristic "windblown" knee deformity seen in human rickets.

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normal. Particularly, no genitourinary or renal deformities were noted. The parathyroids were enlarged, and histology demonstrated diffuse hyperplasia. The most notable findings were in the skeleton. The skull was thin and almost transparent. The costochondral junctions were enlarged in a fashion compatible with the "rachitic rosary" seen in humans (Fig. 2). The skeletal components were variably affected, with the most significant areas being the distal radius and ulna, distal tibia and proximal humerus (Fig. 3). The distal ulna showed a markedly widened physis, with grossly evident intercolumnar separations. The radial physis was less involved. Because of differential growth rates the radius was bowed relative to the pathologically shortened ulna. Histology confirmed the diagnosis of rickets, with greatly increased length to the cell columns, poor formation of primary spongiosa, and disorganized remodeling in the metaphyseal secondary spongiosa. The diaphyseal cortices were histologically relatively normal, while the metaphyseal cortices were thin. Fractures were evident in both proximal tibial metaphyses, and the fibulae had compensatory bowing. Within the endosteal bone there was marrow fibrosis and trabecular thinning. This was most evident in the metaphyseal region, but was also present in the diaphyseal marrow cavity.

DISCUSSION

Fox kits trapped at other locations on the island appeared to be normal. Only the three kits from two specific locations presented these rachitic deformities. Carcass remains found in the dens in both areas lacked bird remains or seal bones. One area was several hundred yards

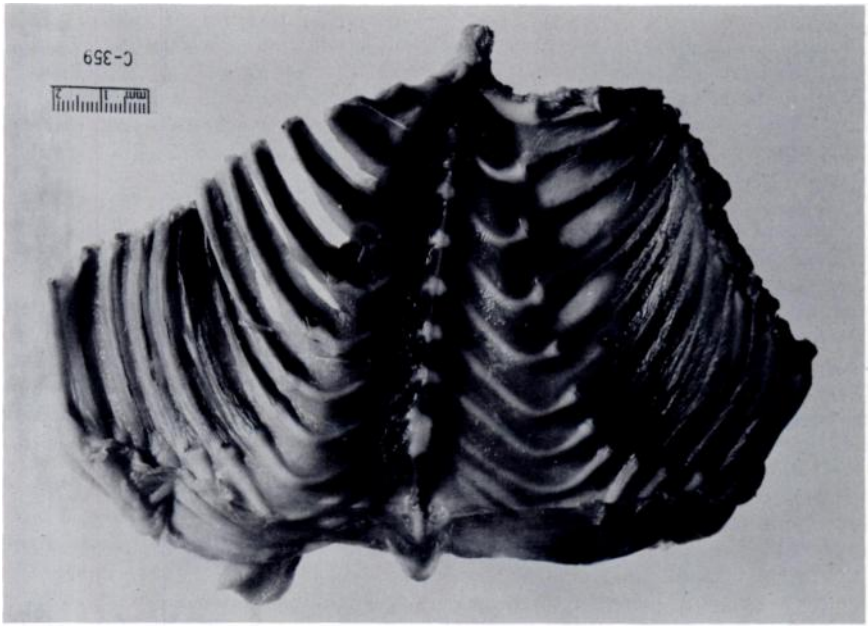


FIGURE 2: Sternum, ribs and costochondral cartilage. Note the fusiform swelling of the costochondral junctions.

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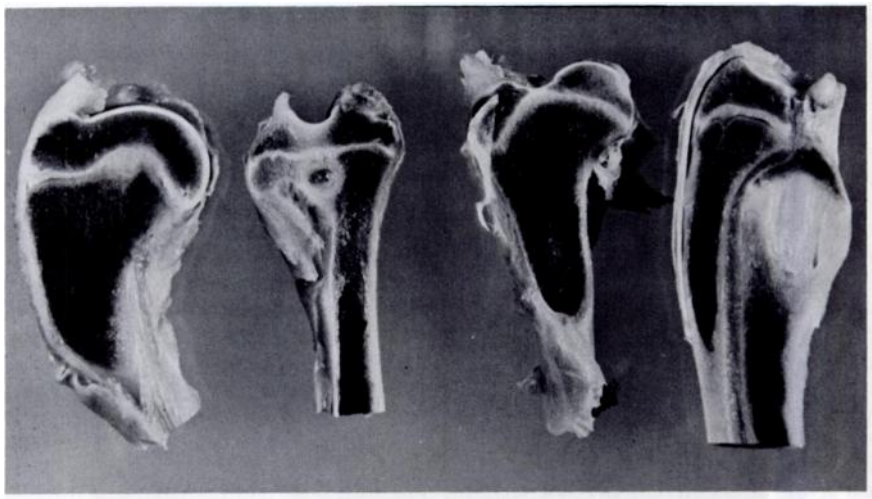


FIGURE 3: From left to right, proximal humerus, distal humerus, proximal femur and distal radius and ulna. There is widening of the mid-portion of the proximal humeral physis, relatively minimal involvement of distal humerus and proximal femur, and marked involvement of the distal radius and ulna. The ulna showed massive widening of the physis. The radius was less involved, but was easily separated from the adjacent metaphysis.

from the seal carcass dump. The den sampled there was littered with meat chunks, but no osseous fragments. The other area was close to the seal rookery, and the remains of numerous placentae were found around the den.

Under controlled conditions fox kits preferentially fed high meat diets developed similar deformities, although with a greater prevalence of fibrous osteodystrophy and facial skeletal effects.[3] Calcium-deficient artificial diet also caused rachitic changes in a related species, the coyote *(Canis latrans)*, quickly corrected by calcium supplementation to the diet.[2]

Interestingly, the diaphyseal bone laid down prenatally and postnatally, when presumably the kits were relying on the mothers for calcium intake, was relatively normal. Only the more recently formed metaphyseal spongiosa and actively forming physis of the juvenile kits was primarily involved, implying that once weaned, these animals were unable to ingest sufficient calcium.

Maltz *et al.*,[5] described a child with rickets postulated to be due to deficient calcium intake, but they did not substantiate whether the vitamin D intake was normal. More recently Kooh and associates[4] described an infant with proven rickets consequent to insufficient calcium intake, with normal vitamin D levels. They felt that the critical factor in the development of the rickets in their case was the rapid growth and metabolic demand for calcium in the fifth to twelfth months. A similar mechanism probably occurred in these fox kits.

It has been established that adequate intake of calcium is necessary for normal skeletal growth and mineralization. However, absolute dietary requirements, whether in the human or other animals, are hard to define. The case reported by Kooh[4] is certainly the first to definitely support the concept that an inadequate

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calcium intake may cause osteoporosis or rickets.

Kooh *et al.*,[4] postulated the following mechanism: protracted calcium deficiency → hypocalcemia → secondary hyperparathyroidism → hyperphosphaturia and generalized aminoaciduria → hypophosphatemia. Whether the initiating event was vitamin D or calcium deficiency, the eventual biochemical manifestations and skeletal lesions are similar. In the fox kits the hyperplasia was diffuse and not due to a discernible adenoma, supporting the likelihood these animals had secondary hyperparathyroidism. Unfortunately it was not possible to adequately assess blood and urine biochemically.

In human rickets one of the major areas of physeal overgrowth (widening) is the distal radius.[7] In contrast, the distal ulna was much more involved in these fox kits. This is not totally unexpected, since the distal ulna is a more important functional unit in quadrupeds and is traumatically injured more frequently than the radius.[6] Again, the reverse susceptibility to trauma is seen in skeletally immature humans.

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