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# Hypertrophic osteodystrophy in a Jindodog

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# 진도개에 발생한 비대성골이영양증

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**요 약 : 전지 파행을 호소하여 의뢰된 약 2개월령의 진도개 암캐에서 비대성골이영양증이 진단되었다. 내원 2일 전부터 파행과 침울 증상을 보인 이 개의 이학적인 검사 결과 grade II/IV 정도의 좌측 전지파행이 관찰되었으며, 좌측과 우측의 앞발목관절의 미약한 종대가 인정되었다. 좌측발목관절을 촉진한 결과 요골과 척골의 골단부위를 촉진할 때 심한 통증을 호소하였으며, 우측 앞발목관절의 촉진에서도 미약한 통증이 인정되었다. 좌측과 우측 앞발목관절의 x-ray 검사에서 양측 전지 원위 요골 및 척골의 성장절 바로 위쪽 골간단에 성장절과 나란히 존재하는 불규칙한 방사선투과성의 선이 관찰되었고, 이 부위의 바로 아래쪽과 위쪽에는 방사선투과성이 감소되어 있었다. 이상의 결과 비대성골이영양증으로 진단되었다. ketoprofen과 amoxicillin을 투여하고, 운동을 철저히 제한한 결과 이 개의 증상은 빠르게 호전되었으며, 치료 2일 후 파행증상이 완전히 소실되었고, 증상이 재발되지 않았다. 이 보고에서는 진도개에 발생한 비대성골이영양증을 처음으로 소개하면서 이 질환의 원인, 진단 및 치료에 대해 문헌적으로 고찰하였다.**

***Key words*** : hypertrophic osteodystrophy, radiographs, Jindodog

## Introduction

Hypertrophic osteodystrophy, which is also referred to as metaphyseal osteopathy, is a disease of young rapidly growing large and giant breed dogs[1,4,12]. The condition causes disruption of the metaphyseal trabeculae mainly in long bones, although metacarpal bone, scapula, ribs and mandible may also be affected[2,4]. The disease is characterized by lameness, and clinical signs may include metaphyseal swelling, signs of pain, and systemic illness with lethargy, pyrexia, and inappetence[1,4,6,14,15]. A significantly higher risk of hypertrophic ostepdystrophy has been reported in the Great Dane, Weimaraner, Boxer, Irish setter, and German shepherd, although the disease has been reported in many other breeds. The disease is most commonly affected 2 to 6 month of age[1,12].

The exact cause of hypertrophic osteodystrophy is unknown. Causes that have been proposed include Vitamin C deficiency[10], oversupplementation of minerals and vitamins, and copper deficiency[8], but these prior suggestions have not been substantiated[1,12,15]. It has recently been proposed that the infectious agents such as canine distemper virus[3,12] and a high calcium diet[5] may be involved in the etiopathogenesis of the disease, and some investigators think the disease has a multifactorial etiology[1,15]. Hypertrophic osteodystrophy should be considered for young growing dogs that have clinical signs of lameness, and differentiated from osteochondrosis dissecans, panostitis, retained cartilage cores, elbow dysplasia, septic or nonseptic polyarthritis, and nutritional secondary bone disease[2,6,12]. Hypertrophic osteodystrophy is diagnosed via radiography[7,12].

A familial or genetic predisposition to the susceptibility of the disease in the specific breed has been suggested[1,13,15]. To the authors knowledge, cases of

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hypertrophic osteodystrophy has not been reported in Jindodog. This report details a case of hypertrophic osteodystrophy in Jindodog, and possible etiologies of the disease are discussed.

## Case histories

A 2 month-old sexually intact female Jindodog was referred to Chonnam national university veterinary teaching hospital with a history of moderate forelimb lameness, reluctance to stand, and depression of 2 days' duration. Physical examination revealed a grade II/IV weight bearing lameness of the left forelimb. The dog had mild firm enlargement of the both right and left carpal joints. Palpation of the distal portion of the left radius and ulna and manipulation of the left carpal joint elicited signs of severe pain. Signs of very mild pain were elicited on palpation of the right carpal joint. The body temperature was 38.7℃, and the heart rate was 145 beats/min. No abnormal findings were found on a neurologic examination. Historical data revealed that this dog was vaccinated with a combined modified virus distemper-hepatitis-parvovirus-parainfluenza vaccine and leptospirosis bacterin about 10 days before present. The diet had consisted of a commercial puppy food.

Radiographs were obtained of both forelimbs. Radiographic abnormalities of the skeleton include irregular metaphyseal radiolucencies with adjacent areas of sclerosis proximal to the distal radius and ulna physis in both forelimbs (Fig 1). A diagnosis of hypertrophic osteodystrophy was made. The dog was treated with ketoprofen (Rhone; France, 2 mg/kg of body weight, IM, every 12 hours) and amoxicillin (20 mg/kg, PO, every 12 hours), and activity was restricted. The dog responded significantly to the therapy, and was clinically improved. At examination 2 days after treatment, signs of pain and forelimb lameness were not evident. Ketoprofen and amoxicillin were discontinued on the next day, and typical activity allowed to resume.

The dog was represented to the veterinary hospital for second vaccination about 3 weeks after discharge, and was reevaluated and no abnormalities was found clinically, and no evidence of pain on palpation of the previously affected metaphysis. Radiographs of the both carpus were taken again, and the distal metaphysis of the radius and ulna of the both forelimbs

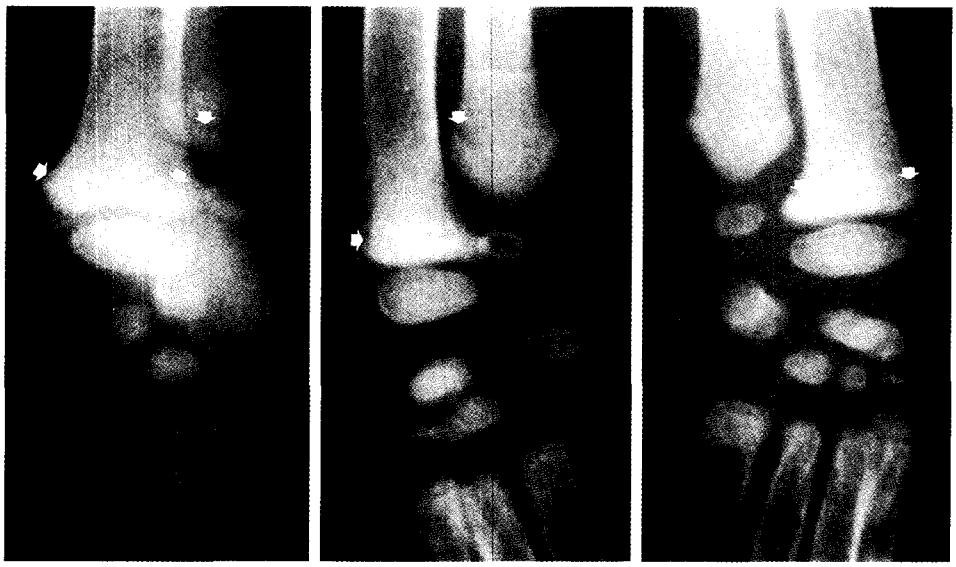

**Fig 1.** Craniocaudal (a) and oblique (b) radiographic views of the left carpus, and craniocaudal radiograph of the right carpus (c). Irregular radiolucent zones are seen in the radial and ulnar metaphysis (arrows), proximal to the physis, and there are regions of increased opacity proximal to the lucent zones (open arrows).

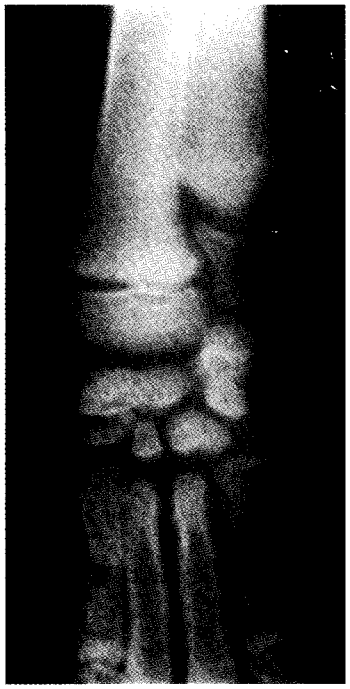

**Fig 2.** Craniocaudal radiograph of the left carpus 3 weeks after treatment. The radial and ulnar metaphysis are normal. There are not irregular radiolucent zones and regions of increased opacity proximal to the physis.

appeared normal (Fig 2). Eight months after discharge, the owner reported no noticeable abnormalities.

## Discussion

Hypertophic osteodystrophy is a developmental disease that primarily affects the metaphyseal area of the long bones, scapula, and ribs of immature large breed dogs[4,10]. The disease is also referred to as skeletal scurvy, canine scurvy, metaphyseal osteopathy, metaphyseal dysplasia, osteodystrophy Types I and II, Moeller-Barlow disease, and hypovitaminosis C8. The condition has been described in Bouvier Des Flandres, Great Dane, Irish Wolfhound, Saint Bernard, Borzoi, Boxer, Dalmatian, Irish Setter, Weimaraner, German short-haired Pointer, German Shepherd, Labrador Retriever, Collie, Greyhound, Basset hound, and some terriers, which are large and giant breeds[2-4,6,10]. However, this can also affect small breed dogs like Maltese[4], although it is infrequent. Clinical signs of the condition may include systemic illness with pyrexia, anorexia, depression. Bacteremia with Escherichia coli, gastrointestinal, respiratory, and neurological signs, ocular discharge, tonsillitis, and hyperkeratosis of the foot pads have been reported in affected dogs[1,2,4,5,7]. Hypertrophic osteodystrophy is usually sporadic but can affect entire litters[1,2]. It has been though that the forelimbs are more often affected than the hindlimbs, and the distal metaphysis of the radius and ulna are the most commonly affected region[4,6], probably because dogs carry more their weight on the forelimbs, and clinicians tend to radiograph the forelimbs more often when the disease is suspected. Abeles et all described, however, that radiographic signs suggested the hindlimbs and forelimbs were equally affected. The lesions are usually bilaterally symmetrical.

The etiology of the disease is unclear. It has been suggested that nutritional disturbances[3,8] and vitamin C deficiency[9] may contribute to its etiopathology, but it have not been proven to cause the condition. Inadequate nutritional vitamin C has been ruled out as a cause of the disease[2,7], but inappropriate metabolism of vitamin C does remain a potential etiology. It has recently been proposed that hypertrophic osteodystrophy could be influenced by feeding a high calcium diet[12]. An infectious cause is also suggested by the clinical and hematological abnormalities such as leucocytosis with neutrophilia and monocytosis[1,3,7]. These type of tissue response is more consistent with bacterial infection than viral infection[2]. It is not uncommon for the patients to develop diarrhea or respiratory signs before they become lame[1,4,7]. However, no bacterial agent has been isolated from the bones of affected dogs[1,2].

Recent studies of hypertrophic osteodystrophy report that canine distemper virus (CDV) may be involved in etiopathogenesis of the condition. Baumg rtner et al[3] reports that CDV may infect all bone cell type (hematopoietic marrow cells, osteoclast, osteoblast, osteocyte) in some dogs experimentally. Bone lesions, restricted to the metaphysis of long bones, were observed in young dogs with systemic distemper following experimental and spontaneous infec-

tion, and CDV-RNA has been detected within osteoclasts and osteoblast in the metaphysis of dogs with hypertrophic osteodystrophy[1,15]. Transmission of hypertrophic osteodystrophy to susceptible dogs has not yet been documented via a bone marrow inoculum from affected dogs or a CDV isolated[2]. The role of CDV in the pathogenesis of the disease is still uncertain[1]. Recent vaccination with live CDV vaccine also be suspected as a etiopathogenesis of the disease[1,2].

Diagnosis of the disease usually based on clinical and radiographic signs. In early stage of the disease, affected bone demonstrate an uneven metaphyseal radiolucent zone parallel to the physis, with a narrow zone of increased radiopacity immediately adjacent to the physis, radiographically. In later stage, radiolucent metaphyseal zones adjacent to the physis still be evident, and periosteal new bone and soft tissue mineralization adjacent to the metaphyseal cortex can be seen. In inactive stage, spiculated periosteal exostoses and diaphyseal deformity of the affected bone can be result[2,3,8]. The dog of this report was in early stage, and radiographic changes were mild.

Although hypertrophic osteodystrophy is considered to be a self-limiting disease in mildly affected dogs[2], severely debilitated animals require fluid therapy, and enteral nutrition. There is no specific treatment in the disease, and treatment is supportive. Many clinicians recommend to use non-steroidal anti-inflammatory drugs[1-3,6-8]. Mineral and vitamin supplementation is controversial[2] and should be avoided. Corticosteroid medication is also controversial[1,2,4,6], but it seems to be effective to the disease.

## Conclusion

Hypertrophic osteodystrophy was diagnosed in a 2 month-old sexually intact female Jindodog. The dog was presented with a two-day history with moderate forelimb lameness and depression. Physical examination revealed a grade II/IV weight bearing lameness of the left forelimb, and mild firm enlargement of the both right and left carpal joints. Palpation of the distal portion of the left radius and ulna, and carpal joint elicited signs of severe pain. Radiographs of both forelimbs demonstrate uneven metaphyseal radiolucent zone parallel to the physis, with a narrow zone of increased radiodensity immediately adjacent to the physis of the distal radius and ulna. The dog was treated with ketoprofen and amoxicillin, and activity was restricted. The dog responded significantly to the therapy, and was clinically improved. To the authors knowledge, cases of hypertrophic osteodystrophy have not been reported in Jindodog. This report details a case of hypertrophic osteodystrophy in Jindodog, and the possible etiologies for the condition are reviewed.

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