Hypertrophic Osteodystrophy in a Dog

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비대성 골이형성증의 진단 영상

최지혜·조태현·김현목·박성민·최민철*·윤정희*¹ (주)해마루 소동물 임상 의학 연구소 *서울대학교 수의과대학

요 약: 2달령의 수컷 로트와일러가 4일간 설사와 통증, 보행 실조 등의 증상을 보여 (주)해 마루 소동물 임상 의학 연구소에 의뢰되었다. 흉부 방사선 검사에서 폐야 전반에 걸친 폐포성 침윤과 흥벽의 심한 비후가 관찰되어 흉막폐렴으로 진단하였으며 복강 초음파 검사에서는 상복부에서 소량의 복수가 관찰되었고 전반적인 간 echogenicity가 증가되어 있었다. 치료 후설사 증상은 사라지고 전신 상태가 다소 호전되어 내원 후 7일째에 흉부 방사선 검사를 재실시하였다. 흉막과 폐의 병면은 변화가 없었으나 상완골과 요골의 골간단 부분에 이상 소전이 발견되어 골격계 방사선 검사를 실시한 결과 상완골, 요골, 대퇴골 그리고 경골의 골간단에 경화성 변화와 무정형의 골막 반응이 관찰되었고 요골, 척골과 경골의 골간단 부분에 성장판 외의 방사선 투과성 선이 판찰되었다. 이상의 특이적인 방사선 소견을 바탕으로 비록본 질환의 정확한 원인은 밝힐 수 없었으나, 감염성 원인에 의한 비대성 골이형성증으로 진단하였다.

Kev words: 개, 비대성 골이형성증, 방사선 검사, 방사선 투과성 선

Introduction

Canine hypertrophic osteodystrophy, metaphyseal osteopathy, is a disease of young growing dogs and mainly affects large and giant breeds^{2,4,10}. The spontaneous disease is characterized by the clinical signs of pain, lameness, pyrexia, and by swelling and hyperthermia of the metaphysis of the long bones^{1,10}.

Hypertrophic osteodystrophy is an osteomalacia disease of unknown etiology, although numerous etiologies have been proposed like nutritional over supplement, vitamin C deficiency and infection^{2,5,10}.

Radiographic changes are limited usually to the ends of long bones, and lesions are symmetric bilaterally^{1,7}. Frequently there is lipping of the metaphysis of long

bones, and a line of increased density is observed next to the growth plate with an adjacent metaphyseal radiolucent zone^{7,10}. The specificity of the radiographic changes for this disease often is taken for granted and it is one of the bony diseases that can be confirmed only with radiographic sign without biopsy¹⁰.

The disease first was considered to be due to vitamin C deficiency because it resembled human infantile scurvy and large doses of ascorbic acid was used to cure the disease^{9,10}. Some investigators think the cause is related to overnutrition and rapid growth, results from mineral overload, or has a multi-factorial etiology¹⁰. Some investigators think it is from infection of respiratory system and recently there are some reports hypertrophic osteodystrophy is related with canine distemper virus infection^{4,5,610}.

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Case

An approximately 2-month-old male Rottweiler was referred to Haemaru Small Animal Clinical Research Institute, because of a 4-day history of diarrhea, depression, non-ambulatory and remained recumbent. The dog had have a good appetite with commercial dry food and only one episode of vaccination.

On physical examination, temperature, respiratory rate and pulse was normal but unknown pain was found.

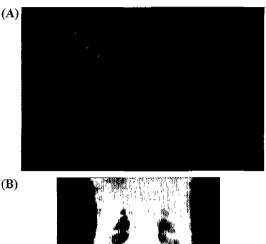
Result of the CBC was normal and serum biochemical abnormalities were found in ALT(341 U/L; normal range 10-75), ALP(380 U/L; normal range 20-200) and albumin (1.7 g/dl; normal range 2.1-4.0).

Thoracic radiography revealed severe alveolar pulmonary infiltrate overall lung fields and thickness of pleural wall. There was interlobar fissure between right cranial and middle lung lobe and air bronchogram especially right lung field (Fig 1). Abdominal ultrasound revealed small amount fluid in cranial abdomen and general hyperechoic change of liver.

On the basis of the results of serum chemistry and radiographic examination it was diagnosed hepatic disease and pleuropneumonia. There was no respiratory sign but the lesion in thoracic radiography was severe, we decided to treat pleuropneumonia with cephradine 25 mg/kg BID IV and gentamicine nebulization twice a day.

After 2 days the clinical sign of diarrhea is resolved completely and serum chemical profiles including ALT, ALP and albumin were decreased in normal range. The dog was more active and could control his head but still remained lateral recumbent.

On day 7, additional radiographic examination was performed to investigate the change of pleuropneumonia. Thoracic radiography revealed that thickness is pleural wall, alveolar infiltrate overall lung fields and consolidation of caudal region of left cranial lobe similar to previous films, but incidental findings of musculoskeletal lesion were observed (Fig 2). Radiography revealed that sclerotic changes and lacy periosteal reactions of proximal and distal metaphysis of bilateral femur, tibia, radius and ulna and proximal metaphysis of bilateral humerus. New bone formation



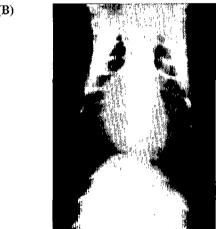


Fig 1. Thoracic radiography, lateral view (A) and ventrodorsal view (B) on day 1. Air bronchogram due to severe alveolar infiltrate at perihilar region and right lung field and interlobar fissure between right cranial and middle lung lobe were observed.

by periosteal reaction was found on shoulder joint. And costochondral junction changed irregular, thick and sclerotic changes (Fig 3). Especially, radiolucent line in addition to physeal cartilage was observed at distal metaphysis of radius and tibia. Hypertrophic osteodystrophy was diagnosed on the basis of these typical radiographic findings.

The dog remained lateral recumbent and appealed pain especially forelimbs with firm, swelling of elbow joint. The etiologies of hypertrophic osteodystrophy are obscure but some causes like nutritional oversupplementation, vitamine C deficiency and infection were proposed. In this case, on the basis of clinical sign like diarrhea and vomiting and pulmonary lesion like pleural thickness and alveolar infiltrate beside the bony lesion, the condition may have an infectious origin

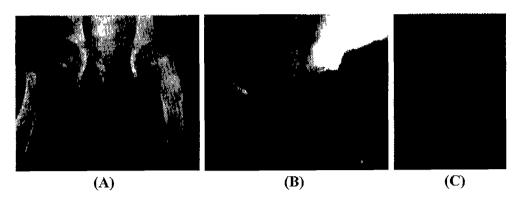


Fig 2. Radiography of femur (A), radius and ulna (B) on day 7. There were sclerotic change of proximal metaphysis (A) and distal metaphysis near the physeal cartilage in femur and distal metaphysis in tibia (B). Abnormal radiolucent line was observed in metaphysis of radius and ulna (C).

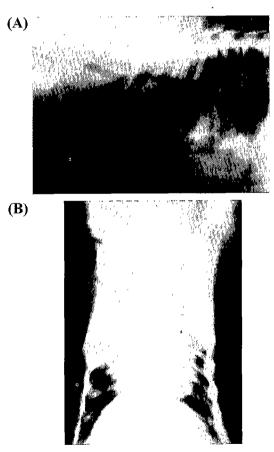


Fig 3. Lateral (A) and ventrodorsal (B) view of case on day 7. Costochondral junction changed thick, irregular and sclerotic. On ventrodorsal view severe alveolar infiltrate of left cranial lung lobe and right middle lung lobe was observed.

with systemic manifestation although an infectious agent could not be identified.

Discussion

Canine hypertrophic osteodystrophy, metaphyseal osteopathy, is considered to be caused by some etiologies like nutritional oversupplementation of dietary calcium, vitamin C deficiency and infection but precise etiology is unknown3,1,8,10. The disease first was considered to be due to vitamin C deficiency because it resembled humane infantile scurvy¹⁰. Many veterinary practitioners use large dose of ascorbic acid to cure of this disease10. Some investigators think the cause is related to overnutrition and rapid growth, results from mineral overload, or has a multifactorial etiology10. Recently, there were many reports that canine distemper virus infection is related with occurrence of hypertrophic osteodystrophy^{4,5,6,7}. The investigators have examined the effects of CDV on the formation of multinucleated, tartrate resistant acid phosphatase positive, calcitonin receptor positive, osteoclast like cells in cultures of canine bone marrow mononuclear cells, and shown that both in vitro and in vivo infection with CDV produced a dose dependent increase in the number and size of osteoclast like cells4.5,6. They have extended these results to show that CDV infection induces interleukin-6 and c-Fos mRNA in these cells, and these results further support the hypothesis that CDV might be involved in the etiopathogenesis

of hypertrophic osteodystrophy^{4,5,6}. They used the technique of in situ hybridization and have shown that CDV is detectable in the bone cells within the metaphysis of the affected dogs⁷. So, they decided that the detection of CDV RNA within the bone cells of dogs with metaphyseal osteopathy suggests that thus virus may be a cause of the disease⁷.

The pathogenesis is obscure, but an apparent disturbance of metaphyseal blood supply leads to changes in the physis and adjacent metaphyseal bone, resulting in delayed ossification of physeal hypertrophic zone³. The acute phase of this disease lasts about 7 to 10 days³. This case showed clinical signs about 30 days.

On physical examination, primarily the metaphysis of long bones are swollen, warm and painful. In this case, the dog's joint especially elbows and hook joint were swollen and painful but there was no warmth. Hypertrophic osteodystrophy can cause abnormal change of long bone mainly but it can also cause change in the mandible, the cranium and the ribs^{4,7}. We examined all bones of the dog, however, there was no lesion of mandible and cranium. But radiography demonstrated that costochondral junction showed increased in density and thickeness. There is one study3 that a history of recent diarrhea may precede the onset of lameness like this case. And other clinical signs of lameness, lethergy, and refusal to rise, which can be shown in hypertrophic osteodystrophy, were correspond with those of this case.

Radiographic metaphyseal densities, which are used to make a clinical diagnosis of hypertrophic osteodystrophy, were found to result from elongation of the calcified cartilage lattice of the primary spongiosa¹⁰. Intertrabecular acute inflammation was associated with necrosis, failure to deposit osseous tissue on the calcified-cartilage lattice, and trabecular microfractures¹⁰. This process led to metaphyseal infraction and separation of the epiphysis¹⁰. Radiographic changes are usually bilaterally symmetrical and most severe in the distal radius and ulnar metaphyseal regions^{1,3,4,10}. Abnormal radiolucent line within the metaphysis and metaphyseal flaring and sclerosis may occur^{3,4,10}. Paraperiosteal new bone formation may develop later in the long bone metaphysis^{3,4,10}. The flaring and

irregularity are symmetrical and are most obvious in the distal radius and ulnar metaphysis^{5,4,10}. The costochondral junction is enlarged with increased radiodensity¹⁰. In this case radiolucent line in the metaphysis, sclerotic change and amorphous periosteal reaction were shown at long bones and costochondral junction was thickened and increased radiodensity. Therefore, on the basis of these typical signs hypertrophic osteodystrophy was diagnosed although an infectious agent could not be identified, this condition may have an infectious cause.

Conclusion

Hypertrophic osteodystrophy can be diagnosed on the basis of history, clinical sign and radiographic examination in a dog. In this case typical radiographic findings were observed at long bone and costochondral junction. There were also severe pulmonary lesion. Although the cause of hypertrophic osteopathy has not been determined, infectious cause has been suggested.

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