Avascular Necrosis of the Femoral head and Neck in Miniature Poodle: A Case Report

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Keyword: miniature poodle, Legg Calve Perthes Disease

Introduction

Legg Calve Perthes disease (LCPD) or avascular necrosis is a developmental disorder in pediatric dogs. It is usually found in young small breed dogs (4) between 5-9 months old and mostly occurs bilaterally. The exact caused was unknown but there were many theories such as hereditary, the anatomical conformation, the increase intracapsular pressure, infarction of the femoral head and hormonal influence (7,9). However, the most realiabletheory was the disruption of the femoral blood flow. Dogs with Legg Calve Perthes often show lameness of their hindlimbs, pain or non-weight bearing of the effected leg. Radiographic diagnosis is one of the most effective diagnostic tools for evaluation of the clinical signs. Surgical treatment is preferred with good to excellent prognosis (9).

Materials and Methods

Nine months old, intact male, miniature poodle, named “It”, has visited The Small Animal Teaching Hospital, Faculty of Veterinary Science, Chulalongkorn University with the chief complain of lameness. There was no history of trauma, lameness and pain of the left hip joint was detected by the owner 2 weeks prior to the first hospital visit. The dog was mostly indoor dog and raise with homemade diet. Physical examination showed the lameness score 3/5 and partial weight bearing of the left hindlimb. Crepitation of the hip and stifle was not observed. Severe pain of the hip joints was observed during palpation. Radiographic diagnosis at the first visit showed non-remarkable lesion of both coxofemoral joints but muscular atrophy of the left hindlimb was observed.Blood profiles were all the normal range.Anti-inflammatory drugs(2.2 mg/kg of carprofen twice a day for the next 2 weeks) were given. After a month of medical treatment, weight bearing of the left hindlimb was improved but pain was still presented in the same level especially during flexion or gaining pressure to the left hip joint. On the second hospital visited, decrease bone density of left femoral neck was shown in the radiographic diagnosis.

One month later after the first visit, moth eaten like lesion was observed in the radiographic study of the left femoral head with subchondral bone sclerosis and periosteal osteophyte formation(Fig. 1) with the same degree of muscle wasting. After one month of medical treatment and no signs of improvement was noted, left femoral head and neck excision (FHNE) was performed. At the day of surgery, the joint capsules’ surface was slightly irregular, the articular cartilage of the femoral neck was detached and there was an erosive cavity in the femoral head and neck. Fungal and bacterial culture was performed and no growth of fungal was observed but coagulase negative Staphylococcus was found from the bacterial culture test.

The head and neck of the left femur was fixed in 10% formalin for 2 weeks then was changed to decalcification solution for another 2 weeks. The section was stained with hematoxylin and eosins. Microscopic finding showed a cleft in the trabecular bone surrounding with focally extensive of necrotic tissue, fibrosis and chondroid metaplasia in subchondral area. The articular surface was irregularly thickened and ulceration without chondrocyte necrosis was found. The fibrous tissue was infiltrated to the base of the ulceration and erosion lesion with multifocalchondrocyte proliferations. Osteocytes were absent from the lacuna at some areas of the necrotic subchondral bone tissue. Vessel proliferation was observed, osteoclasts were present at edge of the matrix and necrotic bone tissues (Fig. 2). Some lipofuscin engulfed by macrophages were observed in the distorted marrow cavity adjacent to the necrotic area. However, about 95% of the bone marrow at the femoral head remained intact.

There were no complications at the surgical site, stitches were removed at the 10th day after the operation. Rehabilitation (passive range of motion) was performed at home by the owner along with coldcompression. Lameness remained for 2 weeks after the operation. Pain and lameness score decreased
after electrical stimulation and ultrasonographic massage were performed. After a certain period post-operation, swimming for about 20-30 minutes once every week was suggested for the dog. The dog began to use all of its leg in the water with decrease of range of motion at the left hindlimb at the beginning. After one month of rehabilitation, 95% weight bearing at the left hindlimb was noted with a trend of progress prognosis after the completion of rehabilitation course.

Results and Discussion

The clinical signs of “It” was occurred before it could be detected by radiographic diagnosis. So, the diagnosis of LCPD should be combined between various diagnostic tools, such as signalment, history taking, clinical signs and various imaging diagnostic tools (8). However, blood profile may not be an appropriate tool for the diagnostic of this disease. From the bacterial culture result, coagulase negative Staphylococcus was found, it could be opportunistic bacteria in the skin of dogs and it is less likely to be virulent (2, 6). It could be found in either normal dogs and dogs with pyoderma or otitis externa. If there was a staphylococcal infection, systemic signs of infection or complication of the surgical site should be presented. In this case, no signs of infection was presented, so, it could be a contamination during the sample collection process.

The most reliable pathogenesis of LCPD is an ischemic necrosis of femoral head, but the actual pathogenesis and cause still remains unclear. Thromboemboli is also one of the suspected pathogenesis (1). However, neither thrombus nor emboli was presented in the tissue sections in this case. Other etiology of the disease should be in concerned. Further studies could be beneficial to find out the actual cause, early detection of even the prevention of LCPD in the future.

Acknowledgements

I would like to thank, the pathology division for the specimen processing Dr. Supaporn Komute, surgeon, and veterinarians at the surgery clinic and rehabilitation unit for their help in the case study and data collection.

References
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