"The Cricket that couldn't jump"

A Case of Avascular Necrosis of the Femoral Head

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Class of 2020

Clinicopathological Presentation

March 13, 2020

Introduction:

Avascular necrosis of the femoral head is a non-inflammatory aseptic necrosis of the femoral head and neck. It is often due to an ischemic event and results in deformation and degenerative joint disease of the affected pelvic limb (2,5). The exact etiology of the disease is poorly understood, but the pathologic process of revascularization and bony remodeling suggests a vascular origin (2). This disease commonly occurs in young and small breed dogs such as terriers and toy breeds between the age of 3-13months, usually weighting less than <12kgs (4,5). Patients usually present for intermittent or acute pain, and intermittent or acute non-weight bearing lameness of the pelvic limb. On physical examination there is pain on palpation, abduction and extension of the affected hip and variable degree of muscle atrophy of the affected pelvic limb (7). The most common differential diagnoses include patellar luxation and cranial cruciate disease. The diagnosis is mainly based on radiographic changes, and cranio-caudal and flexed hip (frog view) are the preferred views. These radiographs are used to identify changes that consist flattening of and lucent areas on the femoral head, elongation and thickening of the femoral neck, and decreased opacity of the femoral epiphysis (3,4,6). The treatment of choice is a femoral head and neck ostectomy, which is considered a salvage procedure to control the pain, lameness and help the patient regain function of the limb (3). With proper surgical technique and adequate physical therapy, the prognosis for lameness and pain resolution is excellent (3).

History and presentation:

Cricket is a 9-month-old female spayed Yorkshire who presented to Mississippi State University Surgery Department on January 28th, 2019 for non-specific left hip changes and a possible torn cranial cruciate ligament. Cricket became painful and intermittently lame on the left hindlimb in October 2019, one week after a hiking trip in Gatlinburg, TN. The referring

veterinarian prescribed Deramaxx®, Metacam® and recommended rest. These medications alleviated the pain, but signs reoccurred after the medications were discontinued. Cricket presented to a different veterinarian where radiographs of the pelvic and left hindlimb were performed. She was diagnosed with non-specific hip changes and a possible torn cranial cruciate ligament. It was then recommended that Cricket have a CT performed to get a definitive diagnosis.

Upon initial presentation, Cricket was quiet, alert and responsive. She weighed 2.4kgs (5.3 lbs) and had a body condition score of 5/9 (with 5 being ideal). Her vitals were normal with a rectal temperature of 100.7* F, a heart rate of 156 bpm, and a respiratory rate of 32 rpm. Her mucous membranes were pink and moist with a CRT of less than 2 seconds, indicating an adequate hydration status. On cardiopulmonary auscultation, a normal sinus arrhythmia was appreciated, and no murmurs, crackles or wheezes were heard. There was no evidence of any nasal or ocular discharge, and there was no debris present in her ears. She had retained canines, but her teeth appeared otherwise clean and healthy. Her abdomen was tense on palpation. She was weight bearing lame on her left hind limb with a lameness score of 2/5. On orthopedic exam, she had decreased range of motion (ROM), and she was painful on the left hindlimb. She had mild to moderate atrophy on the left hip muscles and biceps femoris muscle. She has a grade I/V medial patellar luxation on the right hindlimb. All other physical exam and orthopedic exam findings were within normal limits.

Diagnosis:

For Cricket's case, cranio-caudal pelvic radiographs revealed moderate to severe flattening of the craniolateral aspect of the left femoral head. There was a step defect along the medial aspect of the left femoral head and neck. The left femoral neck was thickened, and there

were patchy regions of sclerosis and lucencies within the femoral neck and proximal femoral metaphysis/diaphysis. There was moderate remodeling of the left acetabular cup with smooth periosteal new bone formation along the medial aspect of the acetabular bone. There was widening of the left coxofemoral joint with less than 50% coverage of the femoral head by the acetabular rim. There was smooth periosteal proliferation along the medial aspect of the proximal aspect of the left femoral diaphysis. There was moderately decreased muscle mass of the left hindlimb due to disuse muscle atrophy. Consideration for the changes to the left coxofemoral joint and surrounding bony changes to the acetabulum and femur were aseptic necrosis of the femoral head or chronic healing fractures from prior trauma with a probable capital physeal fracture. The sclerotic regions within the femoral neck and proximal femoral metaphysis/diaphysis could have been due to bone infarction or endosteal reaction from prior fissure fractures. Due to the strong breed predisposition, physical examination findings and radiographic evidence avascular necrosis of femoral head was diagnosed. In preparation for surgery, a small animal anesthesia panel was performed, which did not reveal any hematologic abnormalities.

Pathophysiology of the disease:

Avascular necrosis of the femoral has many synonyms; Legg-Calvé-Perthes Disease, aseptic necrosis of the femoral head, coxa plana, osteochondritis, and osteochondrosis, among others. The initial phase of this orthopedic disease is developed due to a non-inflammatory ischemic necrosis of the femoral head that occurs before closure of the capital femoral physis (3). The blood supply to the femoral head while the physis are open comes solely from the epiphyseal vessels (3). The exact etiology is unknown but anatomical conformation, increased

intracapsular pressure, infarction of the femoral head, hormonal influences, steroid use, and genetic predisposition are suspected (1,2,3,4). Hereditary factors are suspected due to the strong breed predisposition, and it is thought to be an autosomal recessive trait (2,4). The compromise to the blood supply causes necrosis of subchondral bone of the femoral head epiphysis. The fragmentation phase occurs due to the loss of mechanical integrity caused by osteonecrosis. Subsequent bone weakening and normal weight-bearing forces can make the bone prone to small microfractures, fragmentation, flattening, and collapse of the subchondral bone of the femoral head and neck, making the animal prone to develop acute/sharp pain, and lameness (1). The fragmentation phase and collapse can overlap with the healing phase which consists of re-ossification and revascularization. The epiphysis will begin to revascularize in order to repair and remove the weak necrotic areas (2). If the growth plate is disrupted, necrosis and revascularization of the metaphysis can also be seen (2). Osteogenesis in the femoral head and neck will lead to enlargement, malformation and incongruence with the acetabulum, leading to degenerative joint disease, osteophyte formation, progressive lameness and muscle atrophy (1,2,5). Granulation tissue will be laid in the affected area, which contributes to the misshaped femoral head. The flattening of the femoral head seems to be a combination of mechanical collapse, asymmetrical growth and disturbances in endochondral ossification (2,7). The femoral head usually flattens after 4 months and collapses within 6 months of the initial ischemic event (7).

This disease usually affects small or toy breed dogs weighing less than 12 kgs (5). The age of onset is ranged from 3-13 months, most commonly occurring between 4-11 months of age (2,3,4). There is no sex predisposition, but there are multiple breed predispositions including,

Terriers, Miniature Poodles, Chihuahuas, and Dachshunds, among others (4,5). This disease also happens in multiple species including rats, rabbits, and humans.

Patients usually present for acute or chronic pain, and/or unilateral lameness that can range from mild intermittent to acute or chronic non-weight bearing. There is a 12-16.5% incidence of bilateral involvement, with a low incidence that both femurs will be affected simultaneously (2,5).

During orthopedic examination crepitus, discomfort or pain can be elicited on manipulation of the hip joint, especially during pelvic abduction or extension (3,7). The thigh muscles are often atrophied due to chronic disuse of the affected pelvic limb. The disease can be subclinical, without evident lameness or pain. Thorough orthopedic examination, and monitoring should be done during the first 13 months of age in predisposed breeds. In moderate to severe cases, the lameness can range from mild intermittent to acute non-weight bearing lameness. During orthopedic examination patella luxation, and cranial cruciate ligament rupture/tear should be ruled out; as these are the two most common differential diagnoses (2,3,5). Other differential diagnosis includes juvenile hip dysplasia, traumatic fracture to the proximal femur, pelvic trauma, infection, or neoplasia (2,3,4).

Diagnosis is based on a combination of signalment, history, physical examination, pelvic radiographs and is confirmed with histopathological evaluation. Other diagnostic tools consist of magnetic resonance imaging (MRI) or commuted tomography (CT). The radiographic findings depend on the severity and stage of the disease. Ventro-dorsal hip extended view is most frequently used to visualize the changes, but the flexed ventrodorsal view of the hip joint (frog-legged view) is preferred to visualize femoral head changes (2,4,5). With mild disease, the acetabulum and contour of the femoral neck can appear normal. However, there can be a wider

joint space and multifocal to focal areas of decreased density within the affected areas of the subchondral bone of the femoral head, femoral epiphysis and the metaphysis (2,7). In the retrospective study made by Borges Cardoso, the radiographic changes were graded in relation of severity, which ranged from grade 1 through 5. Grade 1: consists of decreased density of the femoral head, neck or epiphysis. Grade 2: includes, flattening of the femoral head and presence of density foci of decreased density. Grade 3: indicates irregularity of the articular surface of the femoral head and presence of multiple low-density foci or cavitation's, and a spur on the acetabular rim. Grade 4: is characterized by loss of normal shape of the femoral head, increased prevalence of the areas with decreased density. Finally, Grade 5 consists of fragmentation of the femoral head and discontinuity of the articular surface (7). Sometimes the bone can develop a moth-eaten appearance and the femoral neck can be elongated with a "football" appearance (5).

During early or mild stages of the disease, the animal might not be painful, and lack of evidence of the bony changes may make it hard to identify the disease (4,5). The disease is usually diagnosed after fragmentation, collapse or degenerative joint disease is present (4). If identified early or only mild disease is present, medical management can be attempted to control the pain and disease progression. Medical management consists of weight-reduction, anti-inflammatories, activity restriction and non-weight bearing exercises during the revascularization phase to decrease the chances of fragmentation and collapse (4,6). For most cases, surgical therapy by performing a femoral head and neck ostectomy (FHO) is the treatment of choice (4). There is no evidence that total hip arthroplasty has any post-operative benefits over FHO. However, there has been success in small-breed dogs (<10kgs) and in dogs whose pain did not resolved with an FHO (4). With proper surgical technique and physical rehabilitation, the prognosis for normal activity and pain relief is excellent. Some of the common surgical

complications include infection, decreased range of motion, and a decrease in length of the limb. It is important to review the relationship of the gluteal muscles, iliopsoas, the position of the sciatic nerve and the internal pudendal nerve and artery (4). Damage to these structures can affect the limb integrity and cause significant damage to the architecture of the limb.

Treatment and Management

Cricket underwent an FHO on January 30th, 2019. Once she was under general anesthesia, Cricket was placed in right lateral recumbency. Her left hindlimb was clipped, aseptically prepped with 4% chlorhexidine scrub, and draped for surgery. Using a #10 scalpel blade, a 5cm incision was made from the cranial border of the greater trochanter to the proximal diaphysis of the left femur. Hemostasis was maintained with monopolar cautery throughout surgery. Metzenbaum scissors were used to dissect through the subcutaneous tissues. The tensor fascia lata, superficial gluteal, and middle gluteal muscles were retracted using Senn retractors and 2 Gelpi retractors. The ligament of the deep gluteal muscle was partially incised using a #15 scalpel blade. The superficial leaf of the fascia lata was incised at the cranial border of the biceps femoris and retracted. Freer and AO periosteal elevators were used to elevate the vastus lateralis muscle from the proximal femur and externally rotate the leg. Metzenbaum scissors were used to release the joint capsule, and the ligament of the head of the femur from the femoral head and neck. A sagittal saw was used to perform a femoral head and neck ostectomy. The cut surface was smooth with no irregularities, so rasping of the cut end was not required. The head of the femur was preserved and submitted for biopsy. The ligament of the deep gluteal was closed using an interlocking loop pattern with 3-0 PDS. The subcutaneous layer was closed using 3-0 Monocryl suture in a simple continuous pattern. The skin was closed using an intradermal pattern with 4-0 Monocryl. Post-operative radiographs were performed while Cricket was still

under anesthesia. The 2 retained upper canines were removed under anesthesia in the induction room. A periodontal elevator was used to separate the periodontal ligament from the canine and was carefully removed with Rongeurs. She recovered from anesthesia with mild hypothermia and no other complications. Post-operative pelvic radiographs revealed that the head and neck of the femur were completely resected, and the cut surface was smooth with no irregularities. Post-operative management included Tylenol 3, carprofen, methadone and trazodone. The incision was iced every 6 hours and passive range of motion was begun the next day.

Cricket was discharged on February 1st, 2019 with trazodone for anxiety and activity restriction, Tylenol 3® and carprofen for pain control. She had a 5 cm incision on the craniolateral aspect of the left hip and femur area. She was painful in extension of the hip and was mainly holding the leg up at discharge. Cricket's owners were instructed to ice the incision for the next 2 days and to start warm packing the incision area after the cold packing was discontinued. Also, they were directed to keep Cricket confined for the next two weeks (the time for incisional healing) and slowly increase her activity level until she could return to normal activity by the next 6 weeks. Her owners were instructed that passive range of motion and rehabilitation were critical for Cricket's recovery. At the time of discharge, Cricket had no radiographic or clinical signs of avascular necrosis of the femoral head on the right hip; since the disease has a 12 to 16.5 % of bilateral involvement it was instructed to monitor for future occurrence.

On February 25th, histopathology results from the femoral head revealed epiphyseal osteonecrosis with locally extensive epiphyseal collapse. The lesions were most consistent with Legg-Calves Perthes.

Case outcome:

At her 2-weeks recheck, Cricket's incision site appeared red and inflamed. The referring veterinarian prescribed cefaclor for 10 days to prevent a major infection of the incision site. Cricket returned to MSU-CVM for a 6 week recheck appointment. At that time, she had undergone 3 sessions of physical rehabilitation at Memphis Vet Specialists. Her owners reported that she was still stiff and painful, which was managed with Deramaxx®. She was still intermittently non-weight bearing; she would run and walk but would hold her leg up while standing. She had mild to moderate muscle atrophy on the left hip muscles and biceps femoralis muscle. There was no crepitus on manipulation of the left hip, but she had limited extension of the hip joint and was painful when extending the hip. Discussion with the owners at that time was that, the scar tissue around Cricket's left hip joint was tightening up and was preventing her from fully extending her hip, possibly resulting in discomfort and decreased use of that limb. We recommended to continue at home physical therapy and in addition, we strongly recommended that she continued intense rehabilitation 3 days a week (minimum 2 days a week) at a rehabilitation center, to improve her overall outcome and comfort in the left hip. The focus of the rehabilitation was to stretch the scar tissue around her left hip joint, improve hip range of motion (especially in extension), to improve her ability to use the left hind limb, and to increase muscle mass in the left hind limb.

Cricket underwent 6 weeks of physical rehabilitation, which helped her regain her range of motion, loosen some of the scar tissue and build up muscle mass. Today, Cricket is completely healthy and doing well at home. Her lameness and muscle atrophy have completely resolved, and she continues to enjoy frequent hiking trips with Mom and Dad.

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