Tag, You're it

Kaitlyn N Junkin

Mississippi State University

College of Veterinary Medicine

Class of 2021

Clinicopathologic Conference

October 30th, 2020

Advisor:

Dr. Melody Whitney, VMD

Introduction

Osteochondrosis (OC) and Osteochondritis dissecans (OCD) is a multifactorial musculoskeletal disease found commonly among developing large to giant breed puppies around 4-8 months of age.[1] OC is described as a focal disruption in the endochondral ossification process within the articular-epiphyseal cartilage most commonly found on articular surfaces within the shoulder, stifle, hock, and elbow joints.[1,2,3,4,5] OCD is a further manifestation of OC when a cartilage flap forms and extends from the underlying subchondral bone to the surface articular cartilage.[2] Clinical symptoms of OC/OCD include chronic thoracic or hind limb lameness, pain upon hyperextension and hyperflexion of the affected joint, and development of degenerative joint disease. This developmental disease is often bilateral, and contralateral joints should always be evaluated.[6] Diagnostics typically include imaging modalities such as radiographs, ultrasonography, magnetic resonance imaging (MRI), and arthroscopy.[6] The preferred treatment is surgical intervention to remove the OCD lesions and to promote healing of the subchondral bone lesion to stimulate formation of fibrocartilage.[7] Prognosis is good to excellent for shoulder OC/OCD lesions with arthroscopic treatment and restricted activity post operatively to allow fibrocartilage to fill the defect in the subchondral bone.[5]

History and presentation

Tag was an approximately 8-month old intact male Border Collie that presented to Mississippi State University College of Veterinary Medicine (MSU CVM) Surgery Service on 6/17/2019. One month prior, Tag presented to his primary veterinarian for right thoracic limb lameness after falling out of a truck. His owner reported Tag was painful and reluctant to be active. Radiographs of his right thoracic limb were performed. An osteochondritis dissecans lesion in the right shoulder was diagnosed by his primary veterinarian at this time. He was

treated with Tramadol (5.6 mg/kg orally every 8-12 hours) for 2 weeks, Meloxicam (1.8 mg/kg orally every 24 hours) for 3 weeks, and Prednisone (0.28 mg/kg orally every 24 hours) as needed with the last dose given on 6/15/19. Over the past month, Tag had been reluctant to be active. When he was active, his owner noticed a non-weight bearing right thoracic limb lameness. In the few days prior to presenting to MSU CVM Surgery Service, Tag had become painful and lame in his left thoracic limb. Due to his chronic, persistent lameness in his right thoracic limb and acute lameness in his left thoracic limb, his owner elected to bring him to MSU CVM Surgery Service for further diagnostics and treatment. On presentation, Tag was no longer receiving the Tramadol or Meloxicam. At this time, Tag did not appear to be experiencing any adverse side effects from taking a steroid and non-steroidal anti-inflammatory (NSAID) at the same time.

On initial physical exam, Tag was bright, alert, and responsive. He weighed 17.7 kilograms. His vital parameters were within normal limits with a temperature of 102.1 °F, a heart rate of 120 beats per minute, and a respiratory rate that could not be obtained due to panting but had normal respiratory effort. He had pink mucus membranes with a capillary refill time of less than two seconds. All lymph nodes palpated normally and of appropriate texture and size. On cardiothoracic auscultation, no crackles, wheezes, murmurs, or arrhythmias were appreciated. The abdomen was non-painful, and no organomegaly or masses were detected. He had healed blisters on his right front metacarpal pad and 2nd digital pad and on his left front digital pads. On orthopedic exam, Tag was painful on flexion and extension of both of his shoulder joints, on palpation of the biceps brachii muscle on both front limbs, on extension and supination of both elbows, and on manipulation of both hip joints. The left side was worse than the right for all joints palpated.

Diagnostics

The gold standard for OC/OCD is arthroscopy as it can be diagnostic and simultaneously therapeutic and can also identify lesions that may be missed other imaging modalities such as radiographs, computerized tomography (CT) or MRI.[1] Imaging modalities can be useful in confirming diagnosis OC/OCD before arthroscopy is performed. One study evaluated the sensitivity and specificity of ultrasonography, radiographs, and MRI when compared to arthroscopy and detecting OC/OCD lesions. Overall, MRI was the most sensitive of the imaging modalities with a sensitivity of 96% (compared to radiograph's sensitivity of 88.5%).[6] A benefit of MRI and CT is the absence of superimposition that radiographs have. Without superimposition of other structures, an OC/OCD lesion can more easily be identified.[8] Other diagnostic such as bloodwork and urinalysis do not show changes specific for OC/OCD lesions but are useful tools for overall health screening.[6,7]

For Tag, a complete blood count was performed demonstrating a mild monocytopenia of 167 cells/ul (175-1700 cells/ul), which can be normal for Tag and was not clinically significant. A chemistry panel was performed demonstrating a mild hypercalcemia 11.5 mg/dl (8.8-11.2 mg/dl) and moderate hyperphosphatemia 8.1 mg/dl (2.5-5.0 mg/dl), which are contributed to his juvenile status and clinically insignificant.

Bilateral shoulder radiography was performed. His radiographs demonstrated classic lesions that are seen with OC/OCD, which includes an irregular, radiolucent subchondral defect that is surrounded by sclerosis.[1] On Tag's radiographs of the right shoulder joint, a 6.5 mm concave defect within the caudal aspect of the humeral head with a faint region of sclerosis surrounding this defect was found. This finding was consistent with OC of the right shoulder joint. On radiographs of the left shoulder joint, an 8 mm concave defect within the caudal aspect

of the left humeral head with a faint region of sclerosis surrounding this defect was found. Just caudal to this defect, a smoothly marginated 7.2 mm curvilinear mineral opaque structure was present. These findings were consistent with OCD of the left shoulder joint. Radiographs of both the right and left elbow joints and both the right and left coxofemoral joints were normal.[9]

Pathophysiology

The development of OC/OCD is multifactorial with a variety of suspected inciting causes such as genetics, nutrition, rapid growth, trauma, and ischemia.[2,3] This condition is only found in young, growing animals because it is a defect in the epiphyseal cartilage of long bones, which is not present in adult animals.[2] Although all large to giant breed dogs are at an increased risk of developing OC, studies have shown that the Labrador Retriever, Great Dane, Newfoundland, and Rottweiler are at the highest risk, especially for elbow OC.[3,10] For the predilection sites with the exception of the tarsus, there is a gender predisposition for males due to their tendency to grow faster and larger than females.[10]

In a healthy animal, epiphyseal cartilage ossifies by endochondral ossification and facilitates longitudinal growth of long bones.[2] For normal endochondral ossification to occur, the zones of maturation (resting, proliferative, hypertrophic, and calcifying) must be in the correct temporal and spatial order. The deepest zone of the epiphyseal cartilage is infiltrated with blood vessels and osteoprogenitor cells to produce osteoid and lay down new bone on the surface of the extracellular matrix. As an animal gets older and increases in weight, the blood vessels within the epiphyseal cartilage become smaller and less important.[2] Overtime, all the blood vessel canals will close off and either be replaced with cartilage or anastomose with the epiphyseal bone marrow vessels.[7] In an adult animal, synovial fluid is the nutrition source for articular cartilage.[2]

The primary basis for OC is a focal disruption in endochondral ossification of the articular-epiphyseal cartilage. [1,2,3,4,5] In the dog, the predilection sites for lesions are the caudal humeral head, the medial or lateral femoral condyles, the medial or lateral trochlear ridge of the talus, and the medial aspect of the humeral condyle.[3] With this disruption from trauma, ischemia, or some other event, blood vessels cannot penetrate the deepest zone of the cartilage, and osteoid cannot be laid down. As a result, cartilage is retained and starts to become necrotic due to loss of blood supply. The necrotic, retained cartilage is weaker and more prone to trauma. Additionally, the overlying articular cartilage is not supported by subchondral bone. A cleft can form that extends from the articular surface down to the necrotic, retained cartilage. Normal physiologic loading or abnormal trauma forces can cause a flap to form, which is considered OCD. The flap can remain attached to the underlying bone, or it can break off and form an osteochondral loose body (also known as a joint mouse) that freely floats within the joint cavity. The exposure of subchondral bone to synovial fluid induces synovitis. Possible sequalae are angular limb deformities (if the physeal cartilage becomes damaged), subchondral bone cysts (if necrotic, retained cartilage persists), pathologic fractures (if the growth plate becomes too thick), and severe degenerative joint disease (if synovitis continues).[2,7]

Prognosis is dependent upon type and location of the lesion. There are two different types of lesions that affect the articular-epiphyseal cartilage. Type 1 lesions occur in the avascular, central articular surface and include the following sites: caudo-central humeral head, medial aspect of the humeral condyle, and lateral femoral condyle. Type 2 lesions occur in the vascular joint margin and include the following sites: caudo-medial humeral head and medial or lateral trochlear ridges of the talus. Type 1 lesions produce flaps that either detach or may remain attached to the subchondral bone, which prevents healing of the underlying bone defect.

However, it is possible for the flap to reattach and heal via fibrous tissue. Type 2 lesion defects are more likely to heal with fibrocartilage.[7] For shoulder OC/OCD, type 1 lesions (caudo-central) are more likely to result in lameness post-operatively due to the caudal articular cartilage of the glenoid cavity of the scapula being directly exposed to the subchondral bone where the flap was removed. In type 2 lesions (caudo-medial), only a small portion of the lateral aspect of the subchondral bone comes in contact with the articular surface of the glenoid cavity and favors a better long-term prognosis for return to normal overall function.[5] Prognosis for shoulder OC/OCD is generally good to excellent with most dogs returning to the normal function with no to mild lameness present. Bilateral cases are associated with more long-term persistent lameness.[5] Owners should be warned about the future potential to develop of osteoarthritis despite absence of lameness. However, for OC/OCD found in the stifle, elbow, or hock, prognosis is generally fair to poor due to progressive osteoarthritis, loss of articular cartilage, and persistent intermittent lameness.[8]

Proposed etiologies are trauma, genetics, rapid growth, nutrition, and ischemia. Trauma is unlikely to be the sole contributing factor to developing OC; however, it can contribute to developing OCD later in the disease process as the necrotic cartilage weakens.[2] Nutrition and rapid growth are usually seen together when contributing to OC/OCD. Excess calcium and energy intake in large to giant breed dogs have been noted to be inciting factors. Dogs that have increased energy intake typically grow at a faster rate in addition to having effects on hormones that facilitate in regulating growth such as growth hormone, insulin-like growth factor-1 (IgF-1), thyroid hormones (triiodothyronine and thyroxine), and insulin. Authors Richardson and Zentek proposed that excess calcium in the diet stimulates the release of calcitonin, which increases osteoblastic activity, decreases osteoclastic activity, decreases bone resorption, and increases the

risk of developing OC.[3] This has only been reported once in a paper that performed a study on Great Dane puppies and has not been repeated. The true relationship between nutrition, rapid growth, and OC/OCD is unclear.[7] Lastly, ischemia and subsequent necrosis of cartilage is a rising suspected etiology of OC. The focal ischemia that occurs is more commonly present in the axial aspect where the epiphyseal cartilage becomes avascular first. The vessel pattern within the epiphyseal cartilage is similar within different dog breeds and can explain why there are specific predilection sites among dog breeds and why it is commonly a bilateral developmental condition.[2]

Treatment

OC can be medically or surgically managed; however, surgical management via arthroscopy or arthrotomy is the treatment of choice. Surgery entails removing the flap if present and promote healing of the subchondral bone lesion to stimulate formation of fibrocartilage.[7] Instead of allowing the defect to form fibrocartilage, the defect can be filled with an osteochondral autograft transfer (OAT) or a synthetic implant. OAT is made of articular cartilage from non-weight bearing aspects of the stifle joint.[1] The benefits of OAT are to provide a more accurate biochemical and biomechanical environment within the humeral subchondral defect. These benefits include creating a barrier between the synovial fluid and subchondral bone, providing a more anatomically correct articular contour to the humeral head, and supplying hyaline or hyaline-like cartilage to the defect.[7] The synthetic implant is made of polycarbonate urethane and bone ingrowth material and also provides a more superior biomechanical property to the defect and overall joint that fibrocartilage lacks.[1] Debridement reparative techniques have a primary goal of accessing the bone marrow to provide progenitor cells to the healing subchondral defect. Some common debridement reparative techniques

include curettage, abrasion arthroplasty, subchondral drilling or forage, and microfracture. When curetting the rim of the lesion, the necrotic cartilage is carefully debrided away to expose healthy subchondral bone. However, curettage should not be performed if fibrocartilage is already present within the defect. Another method is abrasion arthroplasty, which consists of using curettes or motorized burrs to expose healthy underlying subchondral bone and vasculature. This procedure is usually performed until adequate subchondral bone bleeding is noted by the surgeon. Subchondral drilling and microfracture are similar procedures, and both result in introducing holes into the subchondral bone until adequate bleeding is encountered.[7]

Due to radiographic evidence of OC in his right shoulder and OCD in his left shoulder, surgery was determined to be the best treatment plan. Tag underwent a bilateral glenohumeral joint arthroscopy on 6/18/19. Due to marked extravasation of fluids, the arthroscopy was coveted to open arthrotomy. The same arthroscopy with subsequent arthrotomy procedure was performed for the right joint space. Large cartilage flaps with necrotic subchondral bone were identified bilaterally and debrided. The subchondral bone was curetted until adequate bleeding was noted. Cefazolin 22 mg/kg was administered intravenously every 90 minutes throughout surgery. Recovery from anesthesia was uneventful.

Postoperatively, Tag was treated with hydromorphone (0.1 mg/kg intravenously every 4 hours), omeprazole (1 mg/kg orally every 12 hours), carprofen (4.4 mg/kg subcutaneously once), and trazodone (5 mg/kg orally every 8 hours). After receiving 2 consecutive doses of hydromorphone, Tag appeared to be extremely painful by screaming and rolling around in his cage at the slightest touch. A ketamine (0.6 mg/kg intravenous) bolus was given and followed by a ketamine (0.6 mg/kg/hr) constant rate infusion (CRI) was administered overnight in addition to hydromorphone. His pain scores and subjective comfort improved markedly with the addition

of ketamine. Tag did well overnight and became more comfortable in his kennel. The next morning on 6/19/19, his pain score was a 1 out of 20 on the Glasgow Composite Pain Scale, and he was bright, alert, and responsive. He was transitioned to oral carprofen (1.9 mg/kg orally every 12 hours) and Tylenol 4 (1.5 mg/kg orally every 8 hours) as well as gabapentin (10 mg/kg orally every 8 hours). The ketamine CRI was tapered over the course of the afternoon. He remained in the ICU overnight to ensure his comfort level remained adequate, and his pain could be maintained on oral medications.

Case Outcome

The next morning 6/20/19, Tag appeared to be feeling back to his normal self and was not sensitive to touch nor painful when walking. He was transferred from ICU into surgery wards, and maintained on trazodone, gabapentin, Tylenol 4, and carprofen orally as previously described. Tag was discharged later that afternoon with 10 days of Tylenol 4, 5 days of carprofen, 30 days of trazodone, and 14 days of gabapentin at the same previously mentioned doses. Discharge instructions were given for strict cage rest with gradual increase in daily activity over an 8-week timeframe. Physical therapy, which consisted of passive range of motion at home 2-3 times a day, was prescribed. On 7/25/19, Tag came back for a 6 week recheck appointment and was found to be healing adequately with a good to excellent prognosis for return to normal function. His owner reported that he was not painful at home and was adjusting to cage rest with the help of oral acepromazine prescribed by his primary veterinarian. Tag was given the all clear to increase his daily activity level and return to normal over the course of a few weeks. On 10/15/20, Tag's owner was contacted for an update. Tag is back to normal with no lameness reported in the past 18 months since he had surgery. He is a farm dog and works

cattle for a living. His owner reports that he works hard all day and never seems to be in pain from his strenuous, active lifestyle.

References

1. Danielski A, Farrell M. Use of Synthetic Osteochondral Implants to Treat Bilateral Shoulder Osteochondritis Dissecans in a Dog. Vet Comp Orthop Traumatol. 2018 Sep;31(5):385-389. doi: 10.1055/s-0038-1651517. Epub 2018 Jun 20. PMID: 29925093.
2. Ekman S, Carlson CS. The pathophysiology of osteochondrosis. Vet Clin North Am Small Anim Pract. 1998 Jan;28(1):17-32. doi: 10.1016/s0195-5616(98)50002-2. PMID: 9463856.
3. Richardson DC, Zentek J. Nutrition and osteochondrosis. Vet Clin North Am Small Anim Pract. 1998 Jan;28(1):115-35. doi: 10.1016/s0195-5616(98)50008-3. PMID: 9463862.
4. Mathis KR, Havlicek M, Beck JB, Eaton-Wells RD, Park FM. Sacral osteochondrosis in two German Shepherd Dogs. Aust Vet J. 2009 Jun;87(6):249-52. doi: 10.1111/j.1751-0813.2009.00418.x. PMID: 19489784.
5. Olivieri M, Ciliberto E, Hulse DA, Vezzoni A, Ingravalle F, Peirone B. Arthroscopic treatment of osteochondritis dissecans of the shoulder in 126 dogs. Vet Comp Orthop Traumatol. 2007;20(1):65-9. PMID: 17364099.
6. Wall CR, Cook CR, Cook JL. Diagnostic sensitivity of radiography, ultrasonography, and magnetic resonance imaging for detecting shoulder osteochondrosis/osteochondritis dissecans in dogs. Vet Radiol Ultrasound. 2015 Jan-Feb;56(1):3-11. doi: 10.1111/vru.12179. Epub 2014 May 21. PMID: 24844132.
7. Tobias, K. (2012). Veterinary Surgery Small Animal. In *Elsevier Inc*.
8. Fossum, Theresa Welch. *Small Animal Surgery*. St. Louis, Mo. : Elsevier Mosby, [2013], 2013.
9. Mississippi State University. Diagnostic Imaging Service. Reports.

10. Quinn R, Preston C. Arthroscopic assessment of osteochondrosis of the medial humeral condyle treated with debridement and sliding humeral osteotomy. Vet Surg. 2014 Oct;43(7):814-8. doi: 10.1111/j.1532-950X.2014.12260.x. Epub 2014 Aug 11. PMID: 25109907.