Milo’s Misstep

Kasey A. Lucore

Mississippi State University

College of Veterinary Medicine

Class of 2021

Clinicopathologic Conference

24 July 2020

Advisor: Betsy Swanson, DVM, MS, DACVS-SA

Introduction:

Elbow dysplasia is the leading cause of forelimb lameness in canines. It has several components including: ununited anconeal process, fragmented medial coronoid process, osteochondrosis, and joint incongruity. The signalment is young, medium to large, and giant breed dogs with males more commonly having a fragmented medial coronoid process than females.[3,7,9,10] This disease is often biphasic, with clinical signs being seen around 6-8 months of age and then again at around 2 years of age. The presentation at the younger age is most likely due to pain from the fragmented medial coronoid process and the later presentation is most likely from osteoarthritis pain. It is a polygenic trait with hereditary and environmental factors.[1,3] It is common to see bilateral involvement and neither of the forelimbs have reports of being over-represented.[9] There are multiple treatment options depending on which disease process is occurring.

History and Presentation:

Milo is an approximately 2-year-old, neutered male, boxer that was presented to the Mississippi State University College of Veterinary Medicine Small Animal Surgery Service on July 31, 2019 for lameness of his forelimbs. Milo's owners reported that he had been lame since they adopted him at the age of five months, and they believed this lameness was progressively getting worse. Three months prior to his presentation at MSU-CVM, his referring veterinarian (rDVM) started him on carprofen (a non-steroidal anti-inflammatory medication). The owners believed this was beneficial for Milo. The rDVM diagnosed bilateral elbow dysplasia via radiographs on 7/16/2019 and then referred Milo to MSU-CVM.

On presentation, Milo was alert and responsive, but nervous. He weighed 27.4 kgs and had a body condition score of 5/9. His vital parameters were: temperature of 101.2°F, heart rate of 92 beats per minute, respiratory rate of 40 breaths per minute, capillary refill time was less than two seconds, and mucous membranes were pink and moist. Cardiopulmonary auscultation revealed no murmurs, arrhythmias, crackles or wheezes. Normal bronchovesicular sounds heard throughout all lung fields. His femoral pulses were strong and synchronous with his heartbeat. His eyes, ears, nose, and mouth were clear of debris and discharge. He was tense on abdominal palpation, but otherwise not reactive/painful with no organomegaly noted. He had decreased elbow flexion bilaterally but was very stoic and did not display any pain response during his orthopedic exam. The remainder of the orthopedic and general exam were within normal limits.

Diagnostic Approach and Considerations:

A typical diagnostic work up of elbow dysplasia starts with a good history collected from the owner and a thorough physical exam. The owner will most likely mention a forelimb lameness which might worsen after exercise or be stiff in the morning. Physical exam usually shows lameness in the forelimb(s), possibly an altered gait, decreased motion in the elbow joint, and pain on manipulation. One of the most effective ways to test for pain in this region is to flex the elbow and laterally rotate it and then feel along the medial compartment.[12] Joint effusion and swelling may also be felt in severe cases. Radiographs are initially performed to diagnose elbow dysplasia. A flexed and extended mediolateral, and an oblique craniolateral-caudomedial view is recommended. The extended mediolateral view is used to look for joint incongruity and to evaluate the humeral condyles for changes consistent with osteochondritis dissecans. The flexed mediolateral view is used to evaluate the anconeal process, look for osteophyte formation, to look for sclerosis of the semilunar notch, and to evaluate the height of the coronoids. The oblique

view is used to evaluate the medial coronoid process and medial humeral condyle as well as osteophyte reactions and osteochondral defects. Some other aspects to look for on radiographs are a “step” or increased space between the radius and ulna, an elliptical shape of the trochlear notch of the ulna, an increased joint space, a cranial displacement of the humeral head, and arthrosis. However, the method of plain radiography does not show all abnormalities and is not reliable for diagnosing subtle joint incongruity.[4,8]

3-D imaging, such as CT or MRI, should also be performed as they are the most reliable diagnostic imaging for diagnosing elbow dysplasia. CT allows for better visualization of non-mineralized cartilage fragments which are common. An MRI allows for better visualization of a fragmented medial coronoid process and any osteochondritis dissecans lesions. Linear tomography or positive contrast arthrography can assist in the diagnosis of elbow dysplasia. Arthroscopy is the most specific and sensitive method used to diagnose elbow dysplasia and can be used as a form of treatment simultaneously.[4,8,11] Infrared imaging is another method that may be utilized for a diagnosis of elbow dysplasia. One study demonstrated that it was able to detect thermal changes to the elbow before changes in radiographs were noted and was correct up to 100% of the time.[8]

Milo was originally diagnosed with elbow dysplasia by his rDVM using radiographs. The radiograph of his right elbow showed a rounded medial coronoid process, osteophyte formation on cranial aspect of the radial head, and smoothly marginated osteophyte formation on the anconeal process. His left elbow also displayed degenerative joint disease with osteophyte formation on the anconeal process and on the medial coronoid process as well.

Since the radiographs of Milo were consistent with elbow dysplasia, repeat radiographs were not needed. Therefore, the computed tomography (CT) was the modality of choice at MSU-

CVM. On the right elbow it revealed: a blunted medial coronoid process, a 9.3 x 4 x 9.1mm mineral attenuating structure (a fragment) adjacent to the medial coronoid process, and a lucent line that showed incomplete fusion of the anconeal process. There was new bone formation in the following regions: the medial and lateral aspects of the humeral condyle, medial and cranial aspect of the radial head, medial coronoid process, anconeal process, and the proximolateral aspect of the ulna. The articular surface of the lateral aspect of the ulna had an irregularly marginated lucent region which was surrounded by sclerosis.

The CT of the left elbow showed: a blunted medial coronoid process, a 5.3 x 3.2 x 7.0mm mineral attenuating structure adjacent to the medial coronoid process, and another mineral structure in the supratrochlear formation that measured 6.4 x 7.5 x 6.7mm. Periarticular new bone formation was found along the medial and lateral aspect of the humeral condyle, anconeal process, medial coronoid process, and the cranial and lateral aspect of the head of the radius. There was also an irregularly marginated lucent region that measured 8.6 x 2.3mm along the lateral articular surface of the ulna which had sclerosis around this site. At this point, Milo was diagnosed with severe bilateral degenerative osteoarthrosis of the cubital joints, fragmented medial coronoid processes and ununited anconeal processes of the cubital joints bilaterally, and elbow joint incongruity secondary to elbow dysplasia bilaterally.

With Milo's diagnosis of bilateral elbow dysplasia, surgical correction in the form of arthroscopy was planned. Arthroscopy is the gold standard for treatment of this disease, especially if performed before extensive secondary degenerative changes are present. If fragmented medial coronoid is treated conservatively, then there will most likely be continued cartilage damage and pain.[3,4,6] An arthrotomy would have been acceptable, but studies show that it can cause a higher number of cases of lameness and septic arthritis postoperatively.[6]

Pathophysiology

Elbow dysplasia can be broken down into four different parts, fragmented medial coronoid process, ununited anconeal process, osteochondrosis, and joint incongruity. Multiple components of this disease are usually present. Fragmentation of the medial coronoid processes has multiple etiological theories but is mainly related to other aspects of this disease. One theory is that there is joint incongruity which results from radioulnar mismatch.[3,10] A short radius transfers extra weight to the medial coronoid process which leads to damage of the subchondral bone. This extra weightbearing force causes destruction of the cartilage of the medial humeral condyle and leads to fragmentation of the medial coronoid process.[5] A short ulna displaces the humeral head proximally (relative to the ulna), which puts extra load on the anconeal process which interferes with the union of the anconeal process.[10,11] This radioulnar mismatch can also be caused by trauma of the growth plate, hypertrophic osteodystrophy, or a persistent cartilage core in the ulnar growth plate.[11]

Another theory is that an osteochondrosis lesion can disrupt endochondral ossification of the coronoid process of the ulna. Osteochondritis dissecans (OCD) lesions are commonly seen with a fragmented medial coronoid process. In the case of OCD, a thickened cartilage flap will be seen over a deep subchondral bone defect. Osteoarthritis is also commonly seen with OCD on the adjacent humeral trochlea or the opposing ulnar surface.[5] Osteochondrosis can destroy the chondrocytes that are deep in the cartilage which can lead to cartilage damage, necrosis, and fissures. Fissures of the coronoid cause pain and can lead to fragmentation. If fragmentation occurs, degenerative joint disease and osteochondritis dissecans can occur by the erosion of cartilage on the medial humeral condyle by the fragment. This most commonly occurs in the medial portion of the joint and can lead to medial compartment disease.[3,4] Medial compartment

disease is the term for the grouping of fragmented medial coronoid process, osteochondrosis and/or OCD lesions.

Fragmented medial coronoid process may also occur from tension of the biceps compressing the craniolateral segment of the medial coronoid process against the radial head.[3,5,10] This force can lead to fragmentation of the medial coronoid.[5] This fragmentation can also lead to kissing lesions which are cartilage wear caused from the displaced medial coronoid process. Kissing lesions occur most commonly on the medial humeral condyle and can also be painful.

An ununited anconeal process most commonly occurs when the ulna is too short relative to the radius. This is commonly caused by damage to the distal ulnar physis. This length disparity causes the radial head to be proximal to the medial coronoid process and causes repeated trauma to the center of ossification, leading to failure of union. The anconeal process ordinarily fuses to the metaphyseal region of the proximal ulna around 5 months of age. If the anconeal process does not fuse as the dog continues to grow, it will press against the humeral trochlea and cause a shearing force which will separate the anconeal process from the ulnar metaphysis. This area will have cartilage loss, possible exposed subchondral bone, and possibly some fibrocartilage. This leads to elbow instability and osteoarthritis.[5,12]

Treatment and Management Options

Due to the multi-faceted nature of the disease, there are multiple documented treatment options. It is common for multiple treatment options to be performed in order to correct elbow dysplasia. However, arthroscopy is the most effective treatment for elbow dysplasia.

In regard to disease of the medial aspect of the coronoid process, a subtotal coronoid ostectomy can be considered. This is typically performed when the disease affects the deeper regions of the coronoid. This can be performed via arthroscopy or by exposing the medial joint compartment via arthrotomy. Another treatment option for disease of the coronoid process includes removal of free fragments and debridement of the diseased portion of bone.[2,5]

A biceps ulnar release procedure can be performed to correct rotational instability causing an excess force on the medial coronoid process. Releasing this muscle will reduce pressure on the medial aspect of the joint and can help prevent fragmentation of the medial coronoid process. This procedure may best be used on dogs with focal subchondral pathology in the region of the radial incisure. This release is performed where the biceps tendon inserts onto the ridge caudal to the abaxial portion of the medial coronoid process.[2,5]

Osteochondritis dissecans is another aspect of the disease that needs to be treated. OCD is a failure of osteochondral ossification which results in a flap of dead cartilage and commonly occurs on the lateral aspect of the humeral condyle. If the lesions are small, curettage may be used to stimulate fibrocartilage growth. However, in large lesions, fibrocartilage may not be enough long-term to prevent recurrence. Fibrocartilage is also unlikely to be able to have an appropriate load-bearing contour again. In these cases, an osteochondral autograft transfer can be performed. This procedure restores contour and creates a hyaline cartilage articular surface.[2,9] With OCD, future osteoarthritis is still common, even with early surgical treatment.[9]

There are a few surgical options for the treatment of the ununited anconeal process. One option is the removal of the ununited anconeal process which is commonly done via arthrotomy. This method has good to excellent long-term function. Another option is to reattach the ununited anconeal process via a lag screw placed from the ulna into the anconeal process. However, this

method is best used if the dog is 24 weeks of age or younger. It is possible for the ununited anconeal process to fuse back, but this is extremely rare.[2]

In cases involving elbow incongruity an ulnar or radial osteotomy can be performed to provide less stress on the elbow. An osteotomy of the ulna or radius can be performed to reduce abnormal forces on the anconeal process, if the anconeal process is stable. An oblique proximal osteotomy allows the proximal part of the ulna to move proximally and tilt cranially. This relieves abnormal pressure and can restore congruity which can allow for the anconeal process to fuse. An oblique osteotomy prevents extreme tilting of the ulna and reduces the mobility of the proximal part of the ulna. Early surgical intervention is important because once malformation of the trochlear notch of the ulna or humeral condyle occurs, surgical treatment may be ineffective.[2,9,11]

A fragmented medial coronoid process is another abnormality that may need to be addressed. Treatment is the removal of the fragment and is best performed arthroscopically. Early detection of this disease, before osteoarthritis is severe, provides the best outcomes. Medical management will still be needed after surgical treatment as surgery can slow the progression of osteoarthritis but will not prevent it.[9]

Other treatment options exist, but they are mostly used as salvage procedures when there is end stage medial compartment disease which can be identified by an extreme loss of cartilage. These include, sliding humeral osteotomy, elbow replacement, elbow arthrodesis (if unilateral disease), and amputation (if unilateral disease). Sliding humeral osteotomy is done to transfer the weight from medial to lateral on the joint. It is done by performing a mid-diaphyseal humeral osteotomy and applying a sliding humeral osteotomy plate. However, this procedure has a lot of complications and although improvements are being made, it still needs more research and

improvement in the technique. An elbow replacement is a viable option as the newer implants have lower complication rates. Elbow arthrodesis will provide pain relief but can only be performed if the disease is unilateral because it will result in severe gait abnormality.[5]

Regardless of which elements of elbow dysplasia are involved, medical management is always a good choice for treatment in addition to surgery. This includes weight control, medical management, activity restrictions, and joint supplements. These will help reduce some of the unnecessary weight and load on the elbow joints. Hydrotherapy and physiotherapy may also be good additions to the treatment regimen. If the disease has progressed too far, or if surgical management has been previously attempted and has failed, medical management may be the best option and provide the best results. Prevention is also suggested through the use of early radiographs to diagnose early onset. Weight control and slower growth in large dogs is also suggested to help prevent the disease.[5,11]

As with any surgery, there are risks and complications. One complication that owners should be aware of is the failure to make a diagnosis by arthroscopy. This was found to be the most common complication overall at a rate of 6.4%. Another complication of an arthroscopy is the need to convert to an arthrotomy which increases the risk of joint sepsis. Arthroscopy is also more painful post-operatively and may lead to more arthritis due to the increased inflammation associated with a larger incision in the joint capsule. Iatrogenic cartilage lesions are another complication that varies greatly by the surgeon's experience and the choice of instruments. Pieces of fragments may also be left behind. Continued lameness can be difficult to monitor post-operatively as a complication due to the progression of osteoarthritis.[6]

Case outcome

On August 1st, 2019, Milo underwent a bilateral elbow arthroscopy to remove the fragmented medial coronoid processes. On the right elbow, there was one large fragment and marked eburnation of the medial humeral condyle. Eburnation is the conversion of the sub-chondral bone to a hard, dense surface at the site of cartilage erosion. The cartilage is eroded away, and the surface is left with a polished appearance. The arthroscopy turned into an arthrotomy due to the fragment being too large to remove arthroscopically. On the left elbow, there was a smaller fragment, several joint mice (pieces of the cartilage flap that have broken off into the joint) and marked eburnation of the medial humeral condyle. There was also soft, necrotic bone on the medial coronoid, and it was debrided using a curette. During surgery he was given cefazolin at a dose of 22mg/kg intravenously every 90 minutes. Postoperatively, he was transferred to ICU for the night where he was managed with hydromorphone at 0.1 mg/kg intravenously every 4 hours and lactated ringer's solution (LRS) at maintenance rate. Milo was moved out of ICU the next day on August 2nd and spent the night in surgery wards. He was placed on carprofen at 2.2mg/kg orally every 12 hours and acetaminophen with codeine at 2 mg/kg orally every 8 hours. On August 3rd, 2019, Milo was discharged with instructions to keep him strictly confined for two weeks. On weeks three and four he could begin with short 10-15-minute walks. Ice packing the incisions for three days and then warm packing for five days was instructed in addition to keeping an Elizabethan collar on and monitoring the incision.

Milo returned on September 12, 2019 for a six week recheck. The owners stated that Milo is doing well and is no longer showing any lameness. They say he is slightly stiff upon

rising from sleep, but quickly walks it off. No orthopedic abnormalities were found at this time and Milo was released to full activity.

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