The Little Physis That Couldn't Scott McMullin Mississippi State University College of Veterinary Medicine class of 2019 Clinicopathologic Conference 6/8/2018

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## Introduction

Radial shortening is an orthopedic developmental condition in which the radius has a decreased longitudinal bone growth owing to an inappropriately shortened growth period relative to its ipsilateral ulna (11). Several different inciting factors can be responsible for this, all of which can result in a prematurely closed physis: osteochondrosis, nutritional oversupplementation, hypertrophic osteodystrophy, genetic predisposition in chondrodysplastic breeds, and trauma. The most common of these is trauma, either by a fracture involving the physis or a crushing insult to a portion or entirety of the physis. The resulting limb deformities following such a traumatic event depend on the extent of the physeal damage and premature physeal closure. If the premature closure is symmetric across the distal physis, possible deformities include carpal varus and a widening of the radiocarpal and radiohumeral joint spaces carpal varus, while the limb itself remains straight. If the physeal closure is asymmetric, halting growth of only one aspect of the distal radius, the accompanying deformities depend on which aspect of the distal radius is affected: premature closure of the medial aspect of the distal radius may result in carpal varus and internal rotation of the carpus, while premature closure of the lateral aspect of the distal radius, however, may result in carpal valgus and external rotation of the carpus. In both cases, radiocarpal and radiohumeral joint incongruities may be involved. If the proximal radial physis is affected, radiohumeral joint incongruities are the most likely deformities (9).

## **History and Presentation**

An approximately one-year-old intact female mixed breed Labrador Retriever was presented to the MSU-CVM surgery service on April 11th, 2018 for a lameness of unknown duration. Two days prior to presentation, her current owner discovered her and one other similar-looking dog roaming his property. After an unsuccessful attempt to locate their previous owner, they were both adopted as strays. It was at this time that her new owner recognized a prominent forelimb lameness and decided to bring her to MSU-CVM for evaluation, suspecting a fracture. Given that she was a stray just days prior to her presentation, all medical history, vaccination and parasite preventative status, and normal behaviors and attitude were unknown. Her owner was able to report, however, that since her arrival, her appetite was normal, and she appeared to be urinating and defecating normally.

On presentation to MSU-CVM, the patient was bright, alert, and responsive. She weighed 24.4 kg and was determined to have an ideal body condition score of 5/9. Her capillary refill time was less than two seconds, and her mucous membranes were pink and moist. Examination of her oral cavity showed mild dental calculus. Examination of her integument revealed the presence of three female ticks, a mild amount of scales on her dorsum and several areas of alopecia on the palmar aspect of her metatarsals. Her orthopedic exam revealed crepitus on manipulation of her left carpus, a carpal varus angular limb deformity, and a shortened left forelimb as compared to the right. Pain was not elicited during palpation of long bones of the limbs, nor during passive range of motion manipulation of these joints. No neurological deficits were noted. All other physical exam findings were within normal limits. A thorough scan with a microchip reader did not reveal a microchip.

# Pathophysiology

Traumatically induced radial shortening is ultimately a condition of disrupted juvenile skeletal development (4). Two primary tissues are involved in the skeletal system, cartilage and bone, and each tissue develops from distinct cell types of mesenchymal lineage, cartilage from chondrocytes and bone from osteoblasts. Bone development can also be divided into two primary categories, with flat bones like the skull forming in a process called intramembranous ossification and long bones of the appendicular system like the radius forming in a process called endochondral ossification. In endochondral ossification, mesenchymal cells differentiate into chondrocytes, mature through several phases to become hypertrophic, secrete a matrix which acts as a scaffolding for mineralization, die, and leave behind spaces or tunnels called lacunae. Vascularization occurs through these lacunae, bringing osteoblasts to refashion cartilage into bone and osteoclasts to remodel the bone (6).

This process of cartilage proliferation and bone remodeling is obviously not completed at birth, and a consequence of this is that a zone of proliferating cartilage is left sequestered between the metaphyses and epiphyses of long bones. This zone is referred to as the physis or growth plate, and it becomes the locus for longitudinal bone growth until skeletal maturity when all physeal chondrocytes have terminally differentiated and been replaced by bone. Injury or disruption of the chondrocytes while the physis is still proliferative or open can result in premature closure and arrested bone development. A handful of different mechanisms exist by which atraumatic disruption of the physis can occur: chrondysplastic breeds like the Basset Hound have a heritable disruption of growth potential (6); "hyena disease" in dairy heifers, a disease in which the hind limbs suffer more stunted growth relative to the forelimbs, results from excess dietary vitamin A administered as treatment for scours; and others (3). Traumatic injury to the physis also has the potential to disrupt further bone development. Fractures of the physis are categorized by the Salter-Harris classification system according to the location of the fracture and prognosis for recovery (1). Types I and II typically affect only hypertrophied chondrocytes and carry the best prognosis. Types III and IV affect a portion of the proliferating layer of chondrocytes, which are most critical to continued bone growth. These are also articular

fractures, predisposing the animal to osteoarthritis. Types V and VI crush and damage a greater proportion of the proliferating chondrocytes and their nourishing blood supply, carrying as a consequence the worst prognosis for continued normal bone growth. Of all these causes, either atraumatic or traumatic, Salter-Harris type V crushing injuries are the most likely to result in premature physeal closure and subsequent radial shortening (11).

Traumatic injury to the antebrachium is most likely to cause injury to, in order of likelihood, the distal ulna, distal radius, and proximal radius (8). Each of these physes is responsible for different proportions of growth potential for their respective bones: 85% of ulnar length is contributed by the distal ulnar physis, 60% of the radius by the distal radial physis, and 40% of the radius by the proximal radial physis (4). They each also normally reach maturity and close at different times: the distal ulnar physis closes between 6 - 12 months of age, the distal radial physis also between 6 - 12 months, and the proximal radial physis between 5 - 11 months. Growth is not even throughout development, typically accelerating during months 4 - 6 and slowing down during months 9 - 10 (2). A consequence of all this is that the degree of limb deformity depends greatly on which physis is injured and when during development this occurs. Potential comorbidities associated with radial shortening are carpal varus or valgus, recurvatum, internal or external carpal rotation, joint incongruities at the carpus and elbow, and fragmented coronoid processes impacted by a shift in load-sharing at the elbow. The long-term effects of these deformities are abnormal force applications through the carpal and elbow joints, leading to osteoarthritis. Animals suffering antebrachial injury can also develop concurrent conditions that, while sharing the same traumatic event, do not form as a direct result of radial shortening, with carpal subluxation being one such example (11).

## **Diagnostic Approach**

As with most other orthopedic conditions, an accurate diagnosis of radial shortening and its comorbidities begins with a thorough history, including signalment, and a physical exam, complete with a full orthopedic exam. The orthopedic exam begins by observing the animal in the search for both signs of lameness and localization of any lameness identified. While owners are often able to recognize lameness in their animals, it is not uncommon for them to mistakenly localize the lameness to the wrong limb, which is one reason this initial observation can be so useful. Following this, a neurologic screening exam should be performed to rule out the possibility of lameness due to neurologic disease. Assuming no neurologic deficits are detected, the rest of the orthopedic exam involves palpation of each limb to assess swelling, effusion, joint crepitus, range of motion, and pain response (4).

Diagnostic imaging follows next, of which three primary modalities are typically employed: radiography, computed tomography (CT), and arthroscopy. Radiography has been reported to be useful in diagnosing elbow joint incongruity when the extent of radial shortening is significant, with 90% sensitivity being achievable with at least 1.5 to 4.0 mm of shortening. Radiographs can also show evidence of osteoarthritis, varus and valgus deformities, and carpal subluxations. CT is more sensitive for smaller degrees of shortening, particularly when being used to print a three-dimensional model of the affected limb. It is also more sensitive in diagnosing associated conditions like fragmented coronoid process. Overall, comparing these two imaging modalities, CT is useful in eliminating bone superimposition and in creating threedimensional models that can be used for surgical planning, while radiography can provide superior spatial resolution (8). Arthroscopy has been shown to be the most sensitive in diagnosing radial shortening and can also identify lesions of the articular cartilage that CT and radiography would miss. It also has the added benefit of allowing joint debridement should that be indicated (4). None of these diagnostic modalities, unfortunately, are able to diagnose a Salter-Harris type V crushing injury until 2 - 4 weeks after the traumatic event, when physeal closure and ossification has already begun (8).

## **Treatment and Management**

Medical management of radial shortening only comes in the form of post-operative care; the only definitive form of treatment of such growth deformities is surgical (4). Assuming the animal has already reached skeletal maturity, surgical treatment is aimed at reestablishing joint congruity at the carpus and elbow and correcting any other comorbidities, with the goal of intervention being to halt as much as possible any further development of degenerative joint disease. As the radius is shortened relative to the ulna, correction of joint incongruities requires either shortening the ulna or lengthening the radius (10).

Shortening of the ulna is achieved by performing an ostectomy proximal to the interosseous ligament connecting the radius and ulna, allowing the trochlear notch of the ulna to be pulled distally. Transecting the ulna distal to the interosseous ligament instead will leave the proximal ulna fixed to the radius and unable to shift into congruity with the radial head (10). After appropriate ostectomy and alignment, the ulna can then be stabilized with either an intramedullary pin or bone plate. As with all bone plates, three screws both proximal and distal to the transected site is ideal (4).

More options are available for lengthening the radius, and they fall into two general categories: gradual or acute lengthening. The process of gradual lengthening is accomplished by distraction osteogenesis, in which a transverse osteotomy is made across the bone and several

pins, ideally three, are applied above and below the osteotomy. These pins are then connected to an external device and gradually stretched over a period of time (10). The optimal rate of daily distraction has been determined to be 1 mm, divided into several episodes per day (4). Using this method, one case of a 4-month-old Labrador retriever has reported being able to elongate the radius 50% over its original length, until the ulna stopped growing and joint congruity was restored (7). Gradual lengthening offers an advantage over acute in the cases of skeletally immature animals, as the radius can be continually readjusted as the animals grows without additional surgery (10).

There are several reported approaches for acute radial lengthening. A transverse osteotomy in the proximal third of the radius allows the surgeon to lengthen the radius by creating a gap large enough to account for the discrepancy in length that can then be filled with either a bone or fat graft. Alternatively, an ostectomy in the ipsilateral ulna can provide a segment of bone to fill the gap. A bone plate or external fixator is then applied to stabilize the radius. Instead of a transverse osteotomy, an oblique osteotomy may be performed, allowing the radius to slide along itself before being stabilized in a similar manner. This method eliminates the need for a gap the be filled (4). A final method discussed in the literature is a sagittal sliding technique that involves a "stairstep" osteotomy that again keeps the radius in contact with itself. This can be fixed with lag screws internally and possibly an external fixator if indicated (10).

Correction of any associated joint disease can be accomplished with either arthroscopy or arthrotomy, with arthroscopy being preferred for its superior visualization and magnification diagnostically and the lower incidence of morbidity due to its reduced invasiveness. A fragmented coronoid process can be removed in this manner, or, if suspected, an incompletely fragmented process may be removed by subtotal coronoidectomy. Cartilage fragments, sometimes referred to as joint mice, can be also be removed thusly, and the articular surface debrided if indicated (4).

As discussed earlier, trauma to the distal radius may also cause sufficient injury to the carpus that a carpal subluxation may result. If mild enough, arthroscopy can be used here to remove cartilage fragments (4). However, carpal subluxation is often best treated with pancarpal arthrodesis. In this technique, the articular surfaces of all carpal bones are thoroughly debrided to facilitate arthrodesis of the joint spaces. Bone graft material can be placed in these joint spaces to aid healing. The joints are then stabilized by placement of a dorsal bone plate extending from three plate holes in the distal radius to three plate holes over, usually, the third metacarpal bone (5). Where bone purchase is available over the carpal bones, additional bone screws should be placed. A 10-degree angle of extension has been determined to be the ideal contour for a carpal arthrodesis plate (4).

The prognosis for correction of radial shortening and its comorbidities depends heavily upon the degree of deformity and osteoarthritis at the time of surgical intervention. Without surgery, lameness and osteoarthritis will steadily progress. With mild enough deformity and osteoarthritis, however, prognosis for a normally functioning limb following surgery is good. It is not uncommon for non-steroidal anti-inflammatory drugs (NSAIDs) to be eventually necessary for management of joint pain (4).

# **Case Outcome**

Pre-operative radiographs of the elbow revealed elbow dysplasia, degenerative osteoarthrosis, a possible fragmented coronoid process, an osseous fragment in the humeroradial joint, and a widened humeroradial joint space. The carpus showed subluxation of the

antebrachiocarpal and middle carpal joints, medial subluxation of the intermediate carpal bone, and a mineral fragment proximal to the fifth metacarpal bone. Sufficient disease was evident that no further imaging was deemed necessary.

On April 12th, 2018, the patient underwent general anesthesia. A left elbow arthroscopy was performed. In the left elbow joint, full thickness cartilage erosions of the medial aspect of the humeral condyle and medial coronoid process were noted associated with the radioulnar incongruity. Limited debridement of adjacent abnormal cartilage was performed. A caudal approach to the ulna was performed. Monopolar electrocautery was used for hemostasis. Metzenbaum scissors were used to dissect the subcutaneous tissue and fascia along the same plane. Gelpi retractors were used to improve access to the ostectomy site. An oscillating bone saw was used to make a complete transverse osteotomy through the proximal aspect of the ulnar diaphysis and 4 mm of the ulna was removed. The connective tissues between the ulna and the radius were dissected. The ulna was elevated using a periosteal elevator and clam shell bone reduction forceps. The ostectomy site was reduced and a compression plate was placed with 3 screws proximal and 3 screws distal to the ostectomy site. The exposed musculature was then flushed thoroughly, and then each tissue layer was closed separately. A 10 cm incision was made with a #10 scalpel blade on the dorsal aspect of the carpus extending from the distal radius to mid-metacarpal region. Blunt and sharp dissection was used to expose the distal radius, the carpus, and the metacarpals of the left forelimb, and gelpi retractors were used to improve visualization of the joints. The distal end of the radius and the radiocarpal, middle carpal, and carpometacarpal joints were debrided of articular cartilage to stimulate blood flow and facilitate arthrodesis. Cancellous bone autograft was harvested from the ipsilateral proximal humeral head with a curette and deposited around the debrided bones of the carpus. A 2.7mm - 3.5 mm

pancarpal arthrodesis plate angled approximately 10-12 degrees was placed in compressive fashion on the dorsal surface of the carpus and secured with 9 cortical screws extending from the distal radius to the fourth metacarpal. Each tissue layer was closed separately.

Post-operative radiographs were obtained that revealed appropriate positioning of the implant and alignment of the bones and a residual 15-degree carpal varus.

Post-operative recovery was uneventful. The patient was then medically managed with 2.2 mg/kg carprofen given orally every 12 hours for 7 days, 2.4 mg/kg Tylenol 4 given orally every 8 hours for 14 days, and 8.2 mg/kg cefpodoxime given orally once daily for 10 days. She became anxious during her extended stay at the hospital, which was managed with 2.2 mg/kg trazadone given orally every 8 hours or as needed.

On May 8th, 2018, she was sedated with 5 mcg/kg dexmedetomidine IV and 0.2 mg/kg butorphanol IV, and recheck radiographs were obtained. These revealed no evidence of osteomyelitis or implant failure, periosteal proliferation likely due to reactive bone, and some possible disuse osteopenia of the cuboidal carpal bones.

# References

- Chadwick, C. J., and George Bentley. "The Classification and Prognosis of Epiphyseal Injuries." Injury, vol. 18, no. 3, 1987, pp. 157–168.
- Dennis, Ruth. Handbook of Small Animal Radiological Differential Diagnosis. Saunders, 2005.
- Dukes, H. H., and William O. Reece. Dukes' Physiology of Domestic Animals. Comstock Pub. Associates, 2004.
- 4. Fossum, Theresa Welch. Small Animal Surgery. Elsevier Health Sciences, 2013.
- Guilliard, M. J., and A. K. Mayo. "Subluxation/Luxation of the Second Carpal Bone in Two Racing Greyhounds and a Staffordshire Bull Terrier." Journal of Small Animal Practice, vol. 42, no. 7, 2001, pp. 356–359.
- Martinez, Simon, et al. "Histopathologic Study of Long-Bone Growth Plates Confirms the Basset Hound as an Osteochondrodysplastic Breed." Canadian Journal of Veterinary Research, vol. 71, no. 1, Jan. 2007, pp. 66–69.
- Preston, Ca. "Distraction Osteogenesis to Treat Premature Distal Radial Growth Plate Closure in a Dog." Australian Veterinary Journal, vol. 78, no. 6, 2000, pp. 387–391.
- Thrall, Donald E. Textbook of Veterinary Diagnostic Radiology. Saunders Elsevier, 2007.
- Tilley, Lawrence P., and Francis W. K. Smith. Blackwell's Five-Minute Veterinary Consult. John Wiley and Sons, Inc., 2016.
- 10. Tobias, Karen M. Veterinary Surgery: Small Animal. Elsevier Saunders, 2012.
- 11. Zachary, James F. Pathologic Basis of Veterinary Disease. Elsevier, 2012.