

Un-United Medial Epicondyle of the Humerus: Radiographic Prevalence and Association with Elbow Osteoarthritis in a Cohort of Labrador Retrievers

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Objective—To longitudinally characterize the radiographic appearance of un-united medial epicondyle (UME) of the humerus, evaluate UME association with osteoarthritis (OA) and consider its relevance to the elbow dysplasia complex.

Study Design—Longitudinal cohort study.

Animals—Labrador retrievers (n = 48) from 7 litters.

Methods—Forty-eight same-sex littermates were paired for this lifetime feeding study. One of each pair was control-fed; the pair mate was fed 25% less than the control each day. Elbows of 46 surviving dogs were radiographed at ages 6 and 8 years, and/or at end-of-life (EOL). Elbow histopathology was done EOL, although UME lesions were not evaluated histologically.

Results—Seven dogs (15%) had UME, representing 5 litters; 4 were control-fed, 3 diet-restricted. Six (86%) dogs had unilateral lesions; 1 was bilateral. UME was evident on craniocaudal (CrCd) radiographic projections by 8 years in all dogs. UME was detected in only 1 elbow by mediolateral radiographic projection. Elbow OA frequency in UME affected dogs was not significantly different from the remaining study population. Histopathologic lesions were bilateral in dogs with unilateral UME.

Conclusions—UME may be more common than previously thought. Most cases were unilateral and diet restriction had no effect on frequency. The CrCd view was critical for diagnosis. Elbow OA was not directly associated with UME.

Clinical Relevance—Infrequent diagnosis of UME could result from infrequent radiography and use of only the flexed lateral radiographic projection required by the Orthopedic Foundation for Animals for elbow screening. Like hip evaluations, screening for UME should be continued for life, until genetics are better understood. Lack of association between UME and elbow OA suggests that UME is not likely a component of elbow dysplasia.

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INTRODUCTION

UN-UNITED medial epicondyle (UME) was first described in 1966 and later defined as a failed fusion

of the medial epicondyle ossification center to the humerus.^{1,2} Since then, different classifications of this condition have been described based on the radiographic location and appearance of the bone fragments.³ Others

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have renamed the condition as osseous metaplasia in the flexor tendons.⁴ Regardless of the name and origin of the fragments, reports seem to agree that the Labrador retriever breed is most commonly affected, although other breeds, such as the German Shepherd dog and English Setter, also seem to be overrepresented based on existing reports.^{4,5} Dogs may have lameness and elbow pain, or not show clinical signs.^{4,5} Diagnosis is made by radiography, although the bony fragments may not be visible in both orthogonal projections.^{3,5,6} A few reports describe dogs with growth of bony fragments over time during adult life, and some dogs can develop fragment regrowth after surgical excision.^{5,7,8}

UME of the elbow is uncommon and poorly understood in dogs. It has been reported as a component of elbow dysplasia, but can exist alone.^{5,7,9} Elbow dysplasia is generally known to terminate in progressive osteoarthritis (OA). Not all reported cases of UME have associated OA^{6,10}; and in those dogs without OA, cause and effect relationships are often uncertain.^{5,7} Additional proposed causes for the UME appearance include dystrophic calcification of flexor tendon origins, traumatic flexor tendon avulsion, medial humeral condylar osteochondritis dissecans, or development of an aberrant ossification center.^{5,7,10} Despite case reports that discuss possible causality, UME has not been evaluated longitudinally, and its potential role as a component of elbow dysplasia has not been established. UME was an incidental observation in a lifetime study of effects of diet restriction in the dog. Our purposes are to characterize the longitudinal radiographic appearance of UME starting at 6 years of age, evaluate its association with OA, and to consider its relevance to the elbow dysplasia complex.

MATERIALS AND METHODS

Forty-eight puppies from 7 litters of Labrador retrievers were paired based on gender and weaning weight, and each pair mate was assigned at random to 1 of 2 feeding groups. Beginning at age 8 weeks, 1 group was control-fed (CF), whereas each diet-restricted (DR) pair mate received 75% of the amount of food that was consumed by the CF pair mate on each preceding day. Feeding and housing protocols for this lifetime study have been described.¹¹⁻¹³

Bilateral elbow radiographs (craniocaudal and extreme flexed mediolateral projections) of surviving dogs were made while the dogs were anesthetized, at ages 6 and 8 years, and/or at end-of-life (EOL). The radiograph series for each dog was reviewed sequentially by a board-certified radiologist (D.N.B.), who was unaware of the feeding assignments of the dogs. All radiographs were examined for evidence of UME, other structural abnormalities (such as the presence of fragmented medial coronoid process, un-united anconeal process, osteochondritis dissecans, and gross elbow incongruity), and OA. Elbow OA was graded as mild to severe based on criteria

defined by the International Elbow Working Group (<http://www.iewg-vet.org/>). Based on the experimental protocol, histopathologic examination of major diarthrodial joints (specifically synovium, articular cartilage, and chondrosesous metaplasia) was performed on elbows after necropsy at EOL. In a systematic fashion, starting externally with the joint capsule, anatomic parts of each joint were examined grossly. The thickness of the joint capsule and presence of chondrosesous metaplasia (osteophytosis), pattern and extent of articular cartilage and subchondral bone loss or deformation, and abnormal joint configuration were assessed in each dog. For histopathologic examination, full-thickness sections were taken with the use of a high speed band saw from the following areas: (1) 20 mm long and 8 mm wide sections of joint capsule from the lateral, medial, and cranial aspects and (2) 5 mm sagittal and transverse sections from both proximal and distal articular surfaces through the level of the joint capsule attachment. The severity (mild, moderate, severe), distribution (focal, multifocal, focally disseminated and diffuse), and character (inflammatory, fibrosing, eburnation, fibrillation, etc.) of any lesion(s) present were noted and a diagnostic, morphologic description was given for each lesion noted. Periarticular structures, including UME fragments, were not evaluated histopathologically.

Statistical comparisons between the subset of dogs with UME and the entire study population, when appropriate, were performed using the Fisher's exact test. Statistical significance was set at $P < .05$.

RESULTS

Forty-six of 48 dogs survived to 6 years of age, and had elbow radiographs evaluated. Further elbow radiographs were evaluated at 8 years in 42 dogs and at EOL in all 46 dogs. UME of the elbow was diagnosed radiographically in 7 of 46 dogs (15%), involving 5 of the 7 litters, with 2 affected dogs in each of 2 litters and 1 affected dog in each of 3 litters. Four CF and 3 DR dogs were affected. UME was diagnosed in 5 right and 3 left elbows. Two affected dogs were male and 5 were female, compared with a 3:5 male:female ratio in the study population. UME was unilateral in 6 dogs (86%) and bilateral in 1 dog (14%). UME fragments of the single bilaterally affected dog grew in size during the study but there was not progression in fragment size in the other 6 dogs.

Craniocaudal radiographic projections of the elbow supported diagnosis of UME at 6 years of age in all 7 dogs; however, 1 elbow of the bilaterally affected dog was not confirmed until age 8 years. Flexed mediolateral radiographs, however, revealed UME in only 1 affected elbow among all affected dogs. Radiographic elbow OA was present in 4 of the 7 UME dogs (57%) at EOL, compared with 13 of 38 (34%) in the remaining study population ($P > .05$). The single bilaterally affected dog had severe radiographic OA in both elbows by EOL, characterized by osteophytes on the anconeal process,

Table 1. Summary Radiographic and Morphologic Data for 7 Dogs with Un-United Medial Epicondyle of the Humerus (UME) at End-of-Life (EOL)

Dog	UME Elbow	Radiographic OA Severity	Morphologic Findings
1	R, L	Severe bilaterally	R, L: severe, bridging fibrillation and eburnation on the articular surfaces of the medial humeral condyle and ulnar trochlear notch; moderate periarticular osteophytosis; mild to moderate fibrosing synovioathy
2	L	Severe	R, L: mild to moderate cartilage fibrillation over the ulnar trochlear notch, moderate cartilage eburnation of the medial humeral condyle; severe, fibrosing synovitis; associated periarticular osteophytosis
3	R	Mild	L: mild cartilage fibrillation involving the medial humeral condyle, ulna, trochlear notch, and coronoid; mild osteophytosis; mild to moderate, fibrosing synovitis R: same as L except worse severity of articular cartilage fibrillation on the medial humeral condyle, trochlear notch, and coronoid process
4	R	Mild	R, L: mild to moderate articular cartilage fibrillation over the medial humeral condyle and trochlear notch; moderate fibrotic synovial thickening
5	L	None	R, L: severe articular cartilage fibrillation and eburnation over the medial humeral condyle and trochlear notch; moderate cartilage fibrillation and eburnation of humerus, ulna, radius
6	R	None	R, L: mild to moderate fibrosing synovitis; associated mild humeral osteophytosis
7	R	None	R, L: no significant pathology

R, right; L, left; OA, osteoarthritis.

radial head, and medial humeral epicondyle, bilaterally. The other 3 dogs with radiographic elbow OA at EOL had mild (2 dogs) and severe (1 dog) radiographic OA, respectively (Table 1). All 3 of these dogs had visible osteophytes on the distal humerus on the craniocaudal projection. On the lateral projection, 1 of the dogs with mild elbow OA also had osteophytes on the anconeal process; the dog with severe OA also had osteophytes on the anconeal process and radial head.

Histopathologic elbow abnormalities in each UME-affected dog generally were identical bilaterally in type and severity of pathology; 1 dog with unilateral UME (dog 3) was the only exception (Table 1). Two of 3 dogs without radiographic OA at EOL had gross and histopathologic changes at necropsy. None of the dogs had radiographic or histopathologic evidence of un-united anconeal process, fragmented coronoid process, or osteochondritis dissecans.

DISCUSSION

Diet restriction and gender had no influence on UME in this population, although diet restriction did, in an earlier report, affect development of radiographic elbow OA.¹² With 15% prevalence, our results suggest that UME may be more common than previously thought.^{5,14} Moreover, our findings support an earlier suggestion that Labrador retrievers may be an overrepresented breed.⁵ However, it is also possible that UME is less often detected in the general pet population because pet dogs without clinical signs are not often screened with elbow radiographs, except for breeding purposes. In addition, the bony fragments characteristic of UME are not readily visible on the extreme flexed mediolateral radiographic projection that is used for elbow screening for breeding. It

is possible, as well, that UME fragments are not yet visible when younger dogs are screened, although this could not be evaluated in our study. All of these factors would underestimate actual prevalence.

In our study, the craniocaudal projection was much more useful for recognition of UME than the flexed lateral view, confirming that the humerus and radius can obscure observation of UME fragments (Fig 1).^{5,6} Currently, only the flexed lateral radiographic projection is required for elbow screening by the Orthopedic Foundation for Animals (OFA).¹⁵ This presents an important concern as a number of dogs with UME may be scored as “normal.”

Previous reports suggest that UME is more often bilateral and unrelated to trauma, while some suggest an association with osteochondritis dissecans. With these, a genetic cause was proposed.^{1,5,7,9,10} By contrast, we noted more unilaterally affected dogs (6 of 7). Previous reports also have suggested that UME may result from avulsion of the flexor tendons in young dogs secondary to

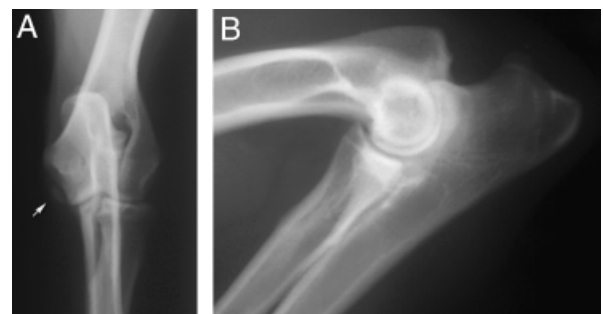


Fig 1. Craniocaudal (A) and extreme flexed mediolateral (B) radiographic projections of an elbow in a dog with un-united medial epicondyle (UME) is shown. The UME fragment is seen on the craniocaudal, but not the flexed mediolateral projection.

trauma.⁸ The activity of the dogs in our study, although not specifically controlled, was limited to a 2 m × 19 m indoor-outdoor kennel run for each dog pair, and none of the dogs had history of thoracic limb trauma.

It is well known that UME can occur concurrently with known causative factors of elbow OA, such as joint incongruity, fragmented medial coronoid process, and osteochondritis dissecans.^{5,7} In contrast, dogs in our study lacked radiographic or histopathologic evidence of other components of elbow dysplasia, indicating that UME can exist alone. Fifty-seven percent of dogs with UME in our study developed radiographic OA by EOL. This prevalence was not significantly different from the remaining study population. It is interesting that both elbows of dogs with unilateral UME had the same histopathologic abnormalities and severity of lesions, with 1 exception. These observations suggest that UME did not predispose dogs to OA in this population, and that the UME fragments are likely solely periarticular. Evidence from this study, therefore, does not support UME as a component of elbow dysplasia, although studies with larger sample sizes are needed for confirmation. Rather, it is possible that the underlying cause of elbow OA may be explained by joint growth regulation though specific trait loci, as has been shown for OA in the canine hip.¹⁶ Genetic studies are beginning to elucidate the genetic aspects of canine OA, but the genetics of elbow disease have not yet been evaluated by quantitative trait analysis.

There are 2 limitations to our reporting of this incidental finding. First, the longitudinal series of elbow radiographs began at 6 years of age. The primary study originated to examine the effects of diet restriction on hip dysplasia; based on clinical observations during the first 5 years, radiographic elbow, shoulder, and spinal data were collected from year 6. For this reason, assertions about age of onset and progression of fragment size before this time would be speculative. Second, histopathology of periarticular structures, including UME fragments, was not examined. Rather, the sole objective of the histopathologic data was to examine for evidence of OA. It is possible that histopathologic evaluation of the UME fragments may have helped to shed light on the underlying cause. Even so, the underlying cause has remained the subject of dispute in other studies, in which histopathology results were available,^{6-8,10} and one source indicated that fragment histopathology does not elucidate the pathogenesis.⁵

At present, canine UME remains poorly understood, and perhaps even the term itself is a misnomer. It seems, based on present knowledge, that characterization of UME as a component of elbow dysplasia should be reconsidered. However, pending availability of genetic studies, it seems prudent to avoid breeding UME-affected dogs. Our observations suggest that the prevalence of

UME may be underestimated in the United States. Elbow screening using at least 2 radiographic projections, along with serial elbow radiography of breeding dogs into later life, may be recommended.

REFERENCES

1. Ljunggren G, Cawley AJ: The elbow dysplasias in the dog. *J Am Vet Med Assoc* 148:887–891, 1966
2. Stevens DR, Sande RD: An elbow dysplasia syndrome in the dog. *J Am Vet Med Assoc* 165:1065–1069, 1974
3. Rorvik AM, Grondalen J: Bony fragments in the soft tissue of the medial and caudal aspect of the elbow joint in the dog: a preliminary report from a radiographic study of 155 elbows in 138 dogs, in EAVDI/ECVDI Annual Meeting, Paris, France, July 18–21, p 36
4. Meyer-Lindenberg A, Heinen V, Hewicker-Trautwein M, et al: Incidence and treatment of metaplasia in the flexor tendons attached to the medial humeral epicondyle in the dog. *Tierärztliche Praxis Kleintiere* 32:276–285, 2004
5. Piermattei DL, Flo GL, DeCamp CE: Fractures and orthopedic conditions of the forelimb, in *Handbook of Small Animal Orthopedics and Fracture Repair* (ed 4). St. Louis, MO, Saunders Elsevier, 2006, pp 339, 352–354
6. Price CJ, King SC: Elbow lameness in a young dog caused by an ossified disc in the joint capsule. *Vet Rec* 100:566, 1977
7. Zontine WJ, Weitkamp RA, Lippincott CL: Redefined type of elbow dysplasia involving calcified flexor tendons attached to the medial humeral epicondyle in three dogs. *J Am Vet Med Assoc* 194:1082–1085, 1989
8. Walker TM: A redefined type of elbow dysplasia in the dog—2 cases. *Can Vet J* 39:573–575, 1998
9. Fox SM, Bloomberg MS, Bright RM: Developmental anomalies of the canine elbow. *J Am Anim Hosp Assoc* 19:605–614, 1983
10. Grondalen J, Braut T: Lameness in two young dogs caused by a calcified body in the joint capsule of the elbow. *J Small Anim Pract* 17:681–684, 1976
11. Kealy RD, Olsson SE, Monti KL, et al: Effects of limited food consumption on the incidence of hip dysplasia in growing dogs. *J Am Vet Med Assoc* 201:857–863, 1992
12. Kealy RD, Lawler DF, Ballam JM, et al: Evaluation of the effect of limited food consumption on radiographic evidence of osteoarthritis in dogs. *J Am Vet Med Assoc* 217:1678–1680, 2000
13. Smith GK, Paster ER, Powers MY, et al: Lifelong diet restriction and radiographic evidence of osteoarthritis of the hip joint in dogs. *J Am Vet Med Assoc* 229:690–693, 2006
14. Harasen: Greg: the mysterious dysplastic elbow. *Can Vet J* 44:673–674, 2003
15. Keller GG, Kreeger JM, Mann FA, et al: Correlation of radiographic, necropsy and histologic findings in 8 dogs with elbow dysplasia. *Vet Radiol Ultrasound* 38:272–276, 1997
16. Chase K, Lawler DF, Adler FR, et al: Bilaterally asymmetric effects of Quantitative Trait Loci (QTLs): QTLs that affect laxity in the right versus left coxofemoral (hip) joints of the dog (*Canis familiaris*). *Am J Med Genet* 124A:239–247, 2004