Canine Lameness Caused by Developmental Orthopedic Diseases: Fragmented Medial Coronoid Process and Ununited Anconeal Process*

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ABSTRACT: Fragmented medial coronoid process and ununited anconeal process are common developmental orthopedic diseases that affect the cubital joint in immature large-breed dogs. Several factors, including genetics, nutrition, and diet, have been implicated in the pathogenesis of both diseases. Lameness and joint pain are common clinical signs and may develop as early as 4 months of age. Early diagnosis allows initiation of proper medical or surgical treatment and may minimize the progression of osteoarthritis.

Fragmented medial coronoid process (FMCP) and ununited anconeal process (UAP) are among the many developmental orthopedic diseases that can cause lameness in immature dogs. A complete patient history and physical examination, including orthopedic and neurologic examination, are essential for developing a diagnosis of FMCP or UAP. The diagnosis is often confirmed radiographically, but magnetic resonance imaging or computed tomography (CT) may also provide valuable information. This article discusses the role of FMCP and UAP in lameness in juvenile dogs. The pathophysiology, diagnosis, treatment, postoperative care, and prognosis of each disease are discussed.

FRAGMENTED MEDIAL CORONOID PROCESS
Incidence and Pathophysiology

FMCP is a separation of the axial border of the medial coronoid process of the ulna. This typically involves a small fragment of the medial coronoid adjacent to the anconeal process. The anconeal process usually forms an osseous union with the proximal ulna by 20 weeks of age in most breeds.

Arthroscopic treatment of FMCP is preferred over traditional surgical techniques. Recent studies suggest that medical and surgical therapy may be equally effective in treating some dogs with FMCP.

the radial head. It is a common cause of lameness and degenerative joint disease (DJD) in large- and giant-breed dogs, including Bernese mountain dogs, rottweilers, Labrador and golden retrievers, Newfoundlands, German shepherds, and chow chows.\textsuperscript{1–5} FMCP more commonly affects male dogs and is usually bilateral.\textsuperscript{6}

The etiology and pathogenesis of this condition remain controversial and poorly understood.\textsuperscript{6} It is likely that FMCP is a multifactorial disease influenced by genetics, nutrition, and growth rate.\textsuperscript{7} Some researchers currently believe that osteochondrosis (OC) and elbow incongruity are associated with the development of focal areas of cartilage degeneration in forming joint surfaces. This leads to cartilage necrosis and subsequent fissure formation and fragmentation (Figure 1). Two distinct forms of elbow dysplasia have been described: trochlear notch dysplasia and asynchronous growth of the radius and ulna.

Olsson\textsuperscript{8,9} originally indicated that FMCP and osteochondritis dissecans (OCD) were manifestations of the OC complex. He concluded that any disturbance in the deep layers of the cartilage that formed joint surfaces (i.e., epiphyseal and metaphyseal physes of long bones) could lead to significant changes in the development of the articular cartilage and underlying subchondral bone. Because the medial coronoid process ossifies between 12 and 22 weeks,\textsuperscript{10} it is more vulnerable to both mechanical trauma and the development of OC during this period.

Grondalen\textsuperscript{11} observed fragmentation of the medial coronoid process in 85% and fissures in 41% of 120 dogs with elbow arthrosis. Fissures were believed to be lesions preceding fragmentation. Histologic examination was performed on 28 elbows, and thickened or degenerative hyaline cartilage was observed in only 18%. Grondalen suspected that the medial coronoid process could be exposed to significant forces that would exceed its weight-bearing capacity and lead to fissure formation or fragmentation. This study concluded that “the condition may be due to OC, to general or local mechanical overloading of normal tissue, or to a generalized weakness in the cartilage and the bone in combination with mechanical overloading.”\textsuperscript{11}

In 1986, Wind\textsuperscript{12} examined radiographic differences between normal canine elbow joints and those of Bernese mountain dogs that were clinically affected with elbow arthrosis. She observed incongruity of the elbow joint characterized by proximal displacement of the trochlear notch of the ulna, widening of the humeroradial and humeroulnar joints, and cranial displacement of the humeral condyle. Necropsy findings were similar to radiographic changes. Wind also postulated that during the period between 4 and 6 months of age when the developing coronoid process lies above the level of the radial head, the concomitant development of an ulnar trochlear notch having an abnormally small curvature could result in FMCP. Forces transmitted through the humeral condyle onto the underdeveloped elevated medial coronoid of the ulna would cause the medial coronoid process to fracture. She also concluded

Figure 1—Histologic section of an FMCP from a 10-month-old Labrador retriever. Fissure formation (arrow) and cartilage erosion (arrowheads) can be seen.
that if the ulnar trochlear notch had remained free of arthrosis in a 6-month-old puppy, the medial coronoid process was sufficiently mature to sustain forces exerted by the humeral condyle and FMCP was less likely to occur.³

Wind and Packard¹⁴ examined 825 dogs of various breeds radiographically and made individual measurements of the radius and ulna at different phases of their growth (during the first year of life). There was a significant increase in the relative size of the proximal ulna in the medium- and large-breed dogs. This was necessary to allow the ulnar trochlear notch sufficient space to encompass the heavier humerus characteristic of these breeds. They again suggested that failure of the developing trochlear notch to achieve a sufficiently large arc of curvature to accommodate the humeral trochlea might be a contributing factor in the development of FMCP.

In a radiographic study of 77 Swiss mountain dogs, Bienz⁵ also found asynchronous growth of the radius and ulna and incongruity of the elbow joint. He found that Swiss mountain dogs with FMCP differed from unaffected members of the breed by having a proportionately shorter radius than its paired ulna, a proportionately longer proximal ulna, and an increased distance between the trochlear notch and the proximal radius.

Pool¹⁶ also has supporting evidence that elbow dysplasia associated with FMCP and OC of the medial condyle of the humerus results from asynchronous growth between the radius and ulna. His conclusion is based on unpublished data from dissections of 36 pairs of elbow joints of immature large-breed dogs in which he found a range of developmental stages of FMCP and/or OC of the medial condyle of the humerus. In this study, the radius and ulna were observed to lengthen independently of one another and move slightly past one another until a maturing interosseous membrane and ligament began to constrain the longitudinal movement of these long bones, apparently beginning at about 4 to 5 months of age, and stabilize longitudinal movement before skeletal maturity. The humeral condyle remained firmly seated in the trochlear notch of the ulna in these specimens.

Lesions of FMCP and OC of the medial condyle of the humerus occurred separately or, less frequently, together as “kissing lesions” in the same elbow joint. Both lesions appeared at a time that the disparity in lengths of the proximal radius and ulna elevated the medial coronoid process above the level of the articular surface of the proximal radius. At this time, the coronoid process was located 1 to 2 mm above the level of

Figure 2—Flexed mediolateral (A) and craniocaudal (B) radiographic views of a 6-month-old Bernese mountain dog with FMCP. Radiographic signs include sclerosis of the trochlear notch (arrowheads) and osteophyte formation on the medial epicondyle of the humerus and coronoid process (arrows).
the articular surface of the proximal radius. This joint incongruity forced the medial coronoid process to bear most of the weight transmitted to the ulna by the medial condyle of the humerus. After the fracture of the elevated medial coronoid process had occurred in many of the specimens, a period of increased longitudinal growth of the radius returned the proximal articular surface of the radius to its normal position at the base of the trochlear notch of the ulna, thereby restoring joint congruity. In these specimens, the FMCP lay below the level of the proximal articular surface of the radius, and in this location the lateral radiographic image of the FMCP is difficult to detect because of superimposition of the radial head.

Histopathologic specimens of FMCP fragments from several pups were examined in this study. In older pups with a more mature medial coronoid process, FMCP fracture fragments usually had an abrupt fracture line that passed through a relatively smooth articular surface. These fragments had a well-defined articular margin and a viable base of vertically oriented cancellous bone. In younger pups that still had a primarily cartilaginous medial coronoid process, the FMCP fracture fragment was misshapen, had an irregular fracture line, and was primarily composed of degenerative hyaline cartilage and crushed fragments of ischemic and fibrotic bony elements resembling crushed articular margins of juvenile horses that give rise to chip fractures.

Another report suggests that abnormalities in the extracellular matrix components of the coronoid process may be responsible for fragmentation. Whatever the cause, fissuring and fragmentation of the medial coronoid process create pain and lameness. The fragment is usually not the cause of osteoarthritis (OA) in the majority of cases. The severity of OA and clinical dysfunction are related to the degree of incongruity and subsequent loss of articular cartilage. In severe cases, the only structure with cartilage is often the FMCP; the remaining articular surface is devoid of articular cartilage.

Clinical Signs

The disease process starts early, with clinical signs reportedly occurring most often between 4 and 7 months of age. However, many patients are not presented for veterinary care until they are 7 to 9 months of age and already displaying signs of DJD. Clinical signs of FMCP are difficult to differentiate from OC of the elbow. The two disease processes have been reported to coexist in 37% of canine patients. The patient exhibits a forelimb lameness that becomes worse after exercise or minor trauma. Many dogs with FMCP stand with the elbow slightly adducted and the antibrachium rotated laterally. Pain is elicited on flexion and extension of the elbow, particularly if the paw is laterally rotated while flexing the elbow. Pain may also be elicited by direct palpation over the medial aspect of the elbow joint. Joint effusion, periarticular soft tissue swelling, muscle atrophy, and crepitus may be present.

Diagnosis

Radiography can help diagnose FMCP in most cases. Radiographic views that should be obtained include a standard lateral, cranio-caudal, extended mediolateral, and flexed mediolateral. Extended and supinated caudomedial-cranio-lateral (Cd75M-CrLO) and cranio-lateral-caudomedial (Cr15L-CdMO) views are also useful in diagnosing FMCP. Recently, a mediiodistal to lateroproximal 30° oblique (MEDLAP) view was described that also allows visualization of the medial coronoid region. In many cases, however, a presumptive diagnosis of FMCP is often based on radiographic signs of OA.

Radiographic changes consistent with FMCP include loss of normal detail in the region of the coronoid process and secondary osteoarthritic changes of the elbow. The most consistent radiographic changes seen in patients with FMCP are sclerosis of the trochlear notch and periosteal proliferation of the dorsal anconeal ridge, proximal radius, and medial aspects of the humerus and ulna (Figure 2). In some cases, other diagnostic modalities, such as contrast arthrography, linear tomography, magnetic resonance imaging, CT, or arthroscopy, may be necessary to confirm a diagnosis of FMCP (Figure 3). Nuclear scintigraphy can help
identify the anatomic location of the site of inflammation. From this point, CT generally identifies the osteomalacic axial segment of the medial coronoid.

**Treatment**

Treatment of FMCP consists of medical therapy or surgery. Conservative therapy includes weight management, activity restrictions, and medication for pain and OA. Surgical therapy has several goals, including the removal of loose or free-floating cartilage or bone fragments, correction of articular incongruence, and alleviation of joint pain. Arthrotomy and arthroscopy are the current preferred methods of surgical treatment. In most cases, surgery is recommended for dogs younger than 12 months of age that have clinical or radiographic signs of FMCP. Surgery is also recommended in dogs up to 24 months of age with large lesions evident radiographically and significant clinical signs. Dogs with severe radiographic signs of OA are typically poor surgical candidates and are better managed with conservative therapy. Removal of the FMCP or large osteophytes may give temporary relief of clinical lameness, but long-term medical management will still be necessary. Surgical treatment seems to have a favorable outcome despite the progression of OA on follow-up radiographs.

In a study of 19 dogs comparing surgical and medical treatment, medical management resulted in a more rapid return to normal weight bearing than did surgical treatment. Medical management consisted of initial weekly SC injections of an NSAID (pentosan polysulfate) for a total of four doses followed by additional injections based on follow-up examinations. At a 9-month follow-up, no differences were detected between the medically and surgically treated dogs. However, it is important to note that most surgically treated patients in this study did not receive concurrent medical therapy, whereas medically managed cases did require long-term therapy. In a separate study of 22 dogs, little difference in the outcome was observed between dogs treated medically or surgically. Eleven of the dogs that returned for evaluation had progression of OA based on radiographs.

**Prognosis**

The prognosis for FMCP remains variable and depends primarily on the severity of clinical signs and radiographic changes as well as the treatment used. Age at the time of surgery and the surgical approach used did not affect the long-term prognosis. However, as with other developmental orthopedic diseases, the prognosis is worse if severe clinical dysfunction is present at an early age. Additional diagnostic tests, including CT and an assessment of the cartilage surface integrity as observed during arthrotomy or arthroscopy, can aid in determining the long-term prognosis. Early diagnosis and treatment with surgery can allow a more positive clinical outcome, but surgery is not curative and secondary OA usually progresses.

**UNUNITED ANCONEAL PROCESS**

**Incidence and Pathophysiology**

UAP affects several breeds and occurs when the anconeal process does not form an osseous union with the proximal ulnar metaphysis by 20 weeks of age. Although the condition varies among breeds, the anconeal process remains ununited if not attached to the ulna by 20 weeks. UAP primarily occurs in large- and giant-breed dogs, including German shepherds, Saint Bernards, Great Danes, and Labrador retrievers. It is also seen in chondrodystrophic breeds, including the basset hound. No sex predilection has been observed for UAP. The disease is reported bilaterally in 20% to 35% of affected dogs, with right and left elbows equally affected.

The etiology of UAP is undetermined and is likely multifactorial. Nutrition, genetics, growth rate, trauma, and OC have all been implicated as causes of UAP. At approximately 12 weeks of age, one to several small centers of ossification develop within the cartilage model of the anconeal process. Multiple centers of ossification begin to coalesce to form a single center separated from the spongiosa of the proximal ulna by a thin cartilage plate resembling a growth plate. In most cases of UAP, the anconeal process may remain in situ, where it is loosely attached to the ulna by fibrous tissue or fibrocartilage. In other cases, the anconeal process is either loose in the joint or wedged in the olecranon fossa of the distal humerus.

The proposed theory that UAP is part of the OC complex of diseases suggests that the anconeus fails to attach to the ulna because of abnormal endochondral ossification within this separate center of ossification. Currently, no reports indicate the presence of degenerative cartilage in the ossification center of the anconeal process before its separation from the ulna. Therefore, there is no unequivocal support for the role of OC in the pathogenesis of UAP. Although it is theoretically possible that OC resulting in focal defects in the cartilage model could underlie the various lesions of elbow dysplasia (i.e., elbow joint incongruity, UAP, FMCP, OCD of the humeral condyle), there is no compelling scientific documentation that supports this theory. Interestingly, in a large study of 500 clinical cases conducted by proponents of the OC theory, only one case was found in which UAP occurred concurrently with OCD of the humeral condyle and FMCP.
Canine elbow dysplasia is a term used to describe abnormal development of the elbow joint and its several manifestations, including UAP, FMCP, OC of the medial portion of the humeral condyle, and elbow incongruity. The definitive cause of elbow dysplasia is unknown and remains controversial.

Other authors feel that UAP is caused by asynchronous growth of the radius and ulna. Joint incongruence may develop within the elbow joint, resulting in abnormal forces that may prevent normal attachment of the anconeus. If radial growth is increased relative to ulnar growth, a proximally directed force is applied to the humeral condyles. This force may be transmitted to the anconeal process and interfere with its normal attachment. Support for this theory is evident in recent studies of elbow joint incongruity and by the fact that the anconeal process will often unite after ulnar osteotomy.

Clinical Signs
The clinical signs of UAP include forelimb lameness, stiffness, and pain and crepitus on palpation of the elbow joints. Clinical signs can occur as young as 4 months of age, with lameness that is intermittent and potentially exacerbated by exercise or prolonged rest. Most dogs are presented for lameness when they are 5 to 12 months of age.

Diagnosis
The diagnosis of UAP is based on radiographic evaluation of both elbows. Typical radiographic views include standard lateral and anteriopalmar as well as flexed lateral (Figure 4). Most cases of UAP are readily diagnosed from the flexed mediolateral radiographic projection. The typical radiographic finding is a radiolucent cleavage line between the anconeal process and the ulna, sclerosis along the margin of the cleavage line, and DJD of the elbow. A definitive diagnosis should not be made before a dog is 20 weeks of age. Secondary degenerative changes of the elbow may be observed and can be of assistance in determining a patient’s prognosis.

Treatment
The options for treating UAP include medical therapy or surgery. Medical management has been less successful than surgery, with secondary OA progressing more rapidly. Spontaneous fusion has been reported by several authors. Surgery is the recommended treatment of choice but is only part of a long-term plan that includes weight management, controlled exercise, and appropriate medications. Several surgical options have been described, including removal of the UAP, surgical reattachment of the anconeal process to the ulna, and osteotomy or ostectomy of the ulna with or without surgical fixation of the anconeal process (Figure 5).

Removal of the UAP is historically the most common procedure used in dogs. The UAP can be excised through either a caudomedial or caudolateral arthrotomy. Long-term results in 23 dogs with UAP were good to excellent after removal of the anconeal process. Arthroscopic removal of the anconeal process has also been documented.

Surgical reattachment of the UAP is attempted using lag-screw or pin fixation, with the implants placed either from the anconeal process into the ulna or from the ulna into the anconeal process. Reattachment is usually attempted before 24 weeks of age. After 24 weeks, reattachment is unlikely and removal of the UAP is usually recommended. One study of lag-screw fixation techniques on 10 elbows showed encouraging results, although additional long-term studies are warranted. If the anconeal process fails to unite after fixation, surgical excision may be required to eliminate joint pain.

Ulnar osteotomy or ostectomy without surgical fixation of the anconeal process has also been performed.
The procedure is intended to correct elbow incongruity caused by discrepancy in the length of the radius and ulna and encourage union between the anconeal process and ulna. In a study using ulnar osteotomy alone, Turner et al. reported good clinical results in 17 dogs but noted that anconeal fusion was not achieved consistently. Sjostrom et al. reported favorable clinical results with ulnar osteotomy alone compared with excision in 22 elbows that were evaluated. Meyer-Lindenberg et al. showed good results in eight dogs treated with ulnar ostectomy without primary fixation of the anconeal process. In chondrodystrophic breeds, such as the basset hound and dachshund, a distal ulnar osteotomy is recommended because proximal and central osteotomies carry a higher risk of painful nonunion. In nonchondrodystrophic dogs, a proximal ulnar osteotomy is preferred for stabilizing joint incongruity but may be associated with a higher morbidity.

Ulnar osteotomy or ostectomy with surgical fixation of the anconeal process has also been performed. The procedure is intended to simultaneously stabilize the ununited fragment and correct elbow incongruity. In a report of four dogs in which lag-screw fixation and ulnar osteotomy were performed concurrently, 23- to 40-month follow-up evaluations showed excellent clinical outcomes with no evidence of pain, crepitus, or joint effusion and minimal progression of OA. Another report of 35 dogs treated with ulnar osteotomy and fixation of the anconeal process found no lameness and only minor increase in radiographic evidence of
OA. These studies suggest that ulnar osteotomy with lag-screw fixation of the anconeal process may be the preferred method of treatment. However, additional controlled studies are needed to confirm this finding.

Prognosis

The prognosis for dogs with UAP is generally favorable provided that treatment is initiated before severe degenerative changes occur. Breeding of affected dogs is generally not recommended.

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**ARTICLE #3 CE TEST**

The article you have read qualifies for 1.5 contact hours of Continuing Education Credit from the Auburn University College of Veterinary Medicine. Choose the best answer to each of the following questions; then mark your answers on the postage-paid envelope inserted in *Compendium*.

1. Which of the following radiographic views is helpful in diagnosing FMCP?
   a. cranio-caudal
   b. extended mediolateral
   c. mediolateral-lateroproximal 30˚ oblique (MEDLAP)
   d. caudomedial-cranialateral (Cd75M-CrLO)
   e. all of the above

2. Which statement regarding FMCP is true?
   a. It is more common in female dogs.
   b. It is usually unilateral.
   c. It is most likely influenced by genetics, nutrition, and growth rate.
   d. Pain is generally not elicited on range of motion.
   e. Affected dogs often stand with the elbow slightly abducted and the antebrachium rotated medially.

3. Surgery goals for FMCP include
   a. correction of articular incongruence.
   b. removal of loose or free-floating fragments.
   c. alleviation of joint pain.
   d. all of the above
   e. none of the above

4. Which statement concerning the treatment of FMCP is false?
   a. Arthrotomy and arthroscopy are the current recommended surgical options.
   b. Dogs with very mild radiographic signs of OA are typically poor surgical candidates.
   c. Dogs younger than 12 months of age that have clinical and/or radiographic signs of FMCP are good surgical candidates.
   d. Dogs with very mild radiographic signs of OA are typically poor surgical candidates.
   e. all of the above
d. Dogs with clinical signs and large lesions or osteophytes and that are younger than 24 months of age are good surgical candidates.
e. Long-term medical management is still likely following surgery.

5. Long-term prognosis does not appear to be affected by
a. age at the time of surgery.
b. severity of clinical signs.
c. radiographic changes.
d. all of the above

e. none of the above

6. Definitive diagnosis of UAP should not be made before the patient is ______ of age.
   a. 90 days
   b. 20 weeks
   c. 40 weeks
   d. 8 months
   e. 1 year

7. Which statement concerning UAP is false?
   a. The etiology is unknown but likely multifactorial, involving genetics, growth rate, and trauma.
   b. A pathogenesis similar to that of OC is suspected.
   c. It has been reported to occur most commonly in Great Danes.
   d. Male dogs are affected twice as much as female dogs.
   e. The disease is more commonly reported to be unilateral.

8. Which of the following is not a common surgical option for UAP?
   a. ulnar ostectomy
   b. proximal ulnar osteotomy
   c. humeral corrective osteotomy
   d. lag-screw fixation
   e. excision

9. Which statement regarding treatment of UAP is true?
   a. Medical therapy is recommended if both elbows are affected.
   b. Spontaneous fusion does not occur.
   c. Medical therapy alone has been less successful than surgery.
   d. Medical therapy should consist of limb immobilization to encourage fusion of the anconeal of the anconeal process.
   e. none of the above

10. Which of the following radiographic views allows the best diagnosis of UAP?
    a. flexed mediolateral
    b. extended and supinated caudomedial-cranialateral (Cd75M-CrLO)
    c. craniocaudal
    d. standard mediolateral
    e. mediodistal-lateroproximal 30° oblique (MEDLAP)