The Shoulder Joint and Common Abnormalities

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KEYWORDS

• Medial shoulder syndrome • Supraspinatus tendinopathy • Bicipital tendinopathy
• Arthroscopy • Osteochondrosis • Osteochondritis dissecans
• Infraspinatus contracture • Shoulder luxation

KEY POINTS

- The shoulder is a complex joint composed mostly of static and dynamic capsuloligamentous support structures.
- Thorough orthopedic and neurologic examination, radiographs, advanced imaging techniques (musculoskeletal ultrasound, computed tomography, and magnetic resonance imaging), and/or arthroscopy are used to obtain accurate diagnosis.
- Osteochondrosis/osteochondritis dissecans is an important developmental disease commonly affecting the caudal humeral head.
- Canine bicipital and supraspinatus tendinopathies show similarities to humans; however, accurate diagnosis and clinical significance are difficult to determine.
- Medial shoulder syndrome is one of the most common shoulder pathologies occurring in greater frequency than luxation.

INTRODUCTION

The shoulder is a diarthrodial joint capable of immense range of motion because it is composed primarily of capsuloligamentous supporting structures but mainly functions in flexion and extension.[1] Additionally, the shoulder is capable of abduction, adduction, and internal and external rotation. Both passive (static) and active (dynamic) mechanisms provide stability and counteract forces that otherwise would destabilize the joint.[2] Passive mechanisms require no muscle activity and allow a wide range of joint motion. Passive stabilizers include the limited joint volume, adhesion/cohesion mechanisms, concavity compression, and capsuloligamentous restraints (medial glenohumeral ligament [MGL], lateral glenohumeral ligament [LGL], joint capsule [JC], labrum, and origin of the biceps tendon).[3,4] Active stabilizers require coordinated muscle contraction and include the cuff muscles (supraspinatus, infraspinatus, teres

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minor, and subscapularis)[5,6] and, to a lesser extent, biceps brachii, long head of the triceps brachii, deltoideus, and teres major muscles.[7]

Shoulder pathology plays an important role in canine forelimb lameness. Historically, shoulder discomfort was generalized into large categories; however, imaging technique advances have allowed for emergence and greater understanding of various disorders. In addition to shoulder palpation, thorough investigation of the elbow and neurologic evaluation are necessary with forelimb lameness because elbow pathology, spinal abnormalities, and peripheral nerve sheath tumors are important differentials.[5] This article gives an updated review of common canine shoulder pathologies, including osteochondrosis, bicipital and supraspinatus tendinopathies, infraspinatus contracture, medial shoulder syndrome (MSS), and luxation.

OSTEOCHONDROSIS/OSTEOCHONDRITIS DISSECANS

Osteochondrosis (OC) is a developmental disease resulting from incomplete endochondral ossification. When progression leads to disruption of the articular surface and flap formation, osteochondritis dissecans (OCD) results (**Fig. 1**).[8,9] OC/OCD commonly affects young, large, and giant breed dogs and occurs bilaterally in 27% to 68% of dogs.[10,11] Although it overwhelmingly is a disease of canines, a single report of feline shoulder OCD has been documented.[12] The caudocentral and caudomedial humeral head are common sites affected[13] (see **Fig. 1**), with less favorable outcomes associated with caudocentral lesions.[14]

Clinical signs often occur at 4 months to 8 months of age.[8] Later presentations likely are secondary to osteoarthritis from a lesion missed in the immature dog. Mild to moderate lameness, worse upon rising and after activity, are described. Examination often

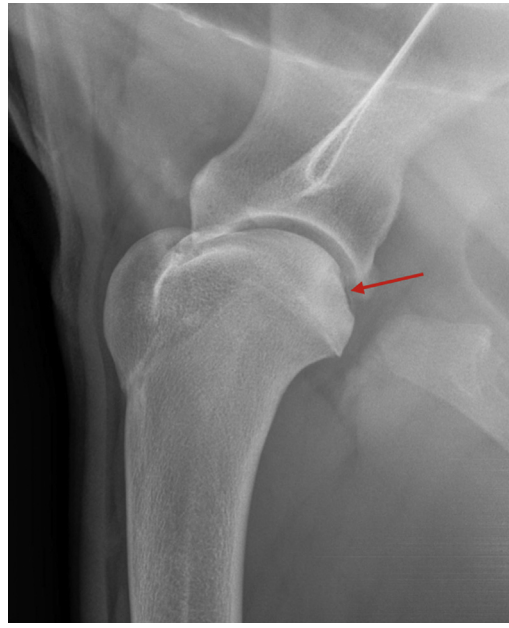

Fig. 1. Lateral shoulder radiograph revealing caudal humeral head OCD lesion. Note the subchondral defect (*arrow*) with flattening of the caudal humeral head and surrounding sclerosis.

reveals discomfort on deep shoulder flexion because this places direct pressure on the caudal humeral head.[8] Radiographs may appear normal or reveal a caudal humeral head subchondral defect (see **Fig. 1**).[15] When no defect is observed but high clinical suspicion exists, musculoskeletal ultrasound, arthrography, computed tomography (CT), and/or magnetic resonance imaging (MRI) may aid in diagnosis.[16,17] Conservative management is recommended only for those less than 6 months of age with mild lameness and no radiographic abnormalities. Surgery otherwise is standard of care and involves flap removal, surrounding unadhered cartilage excision, and encouraging local fibrocartilage formation through arthrotomy or arthroscopy (**Fig. 2**).[9,14] Larger defects or refractory cases can be addressed with osteochondral autografts[18] or synthetic plug implantation.[19] Activity restriction and rehabilitation are integral to a successful outcome.[20] Overall, return to normal or near normal function generally is good following surgery.[11]

BICIPITAL TENDINOPATHY

Bicipital tendinopathy (historically termed, tenosynovitis) once was thought to be a common pathologic finding. Advances in diagnostic capabilities through arthroscopy (**Fig. 3**), musculoskeletal ultrasound (**Fig. 4**), CT, and MRI, however, have led to identification of other soft tissue pathologies not previously recognized.[21,22] Bicipital tendinopathy affects middle-aged to older, medium to large breed dogs, often resulting in chronic, progressive lameness worse after exercise.[8,23,24] The pathogenesis largely is unknown but can be classified as primary or secondary tendinopathies.[25] Primary bicipital tendinopathy is thought to occur secondary to chronic microtrauma.[23,25] The biceps tendon origin is relatively hypovascular, which may predispose it to mechanical failure.[2,25] Secondary bicipital tendinopathy occurs as a result of trauma, impingement via joint mice or enlarged supraspinatus, and/or intraarticular pathology.[24]

Chronic intermittent or progressive weight-bearing lameness, which initially may be noticeable only upon rising or following activity and may or may not be responsive to

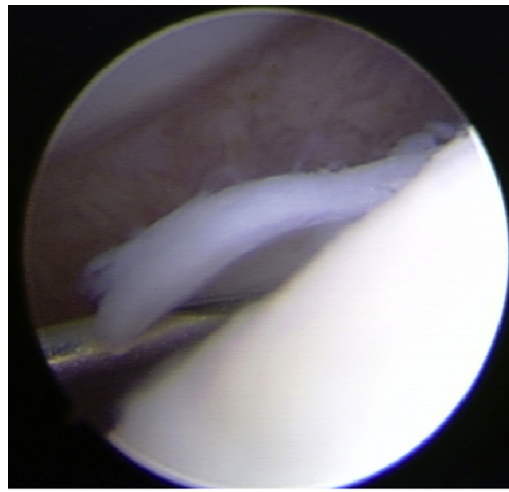

Fig. 2. Arthroscopic image of a caudal humeral head OCD flap. In this image, cranial is to the right. A hypodermic needle can be seen lifting the flap up.

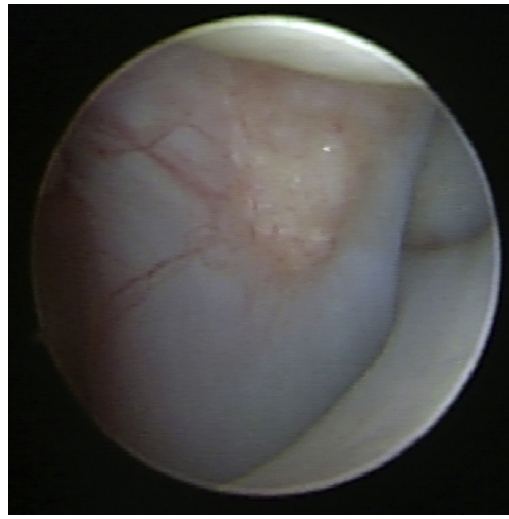

Fig. 3. Arthroscopic image of a normal biceps. The origin of the biceps from the supraglenoid tubercle is noted in the top of the image.

anti-inflammatories, is common. Diagnosis is obtained through examination abnormalities (discomfort on direct shoulder palpation, positive biceps stretch test, and so forth) and diagnostic imaging (radiographs, musculoskeletal ultrasound, MRI, and/or arthroscopy).[5,24,25] Tension upon shoulder flexion and discomfort during biceps stretch test (**Fig. 5**) may be present. Importantly, in contrast, those with a biceps tear have loss of end-feel. Radiographs largely are normal; however, tendon calcification may be present in chronic cases (**Fig. 6**). Given their close anatomic relationship, it may be difficult to distinguish calcification within the biceps versus supraspinatus. Skyline radiographic views are used to help identify calcification within the

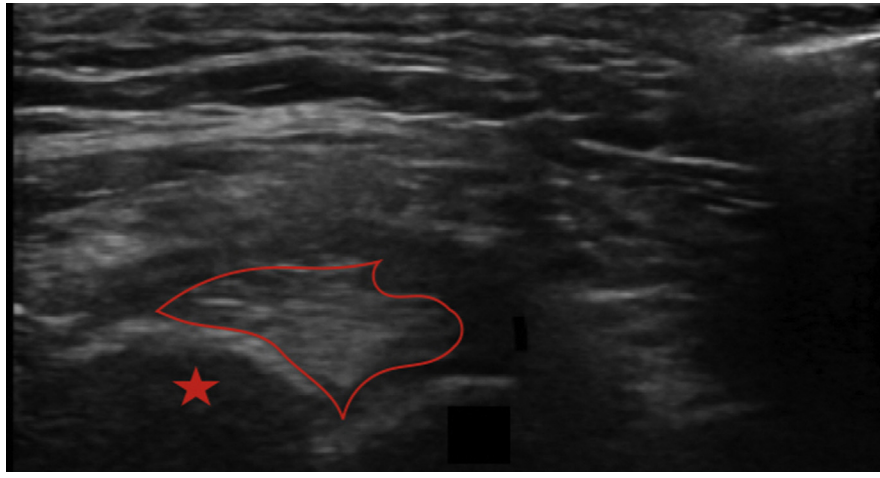

Fig. 4. Musculoskeletal ultrasound image of the normal origin of the biceps (*outlined*) arising from the supraglenoid tubercle (*star*). (*Courtesy of* D. Canapp, DVM, Annapolis Junction, MD.)

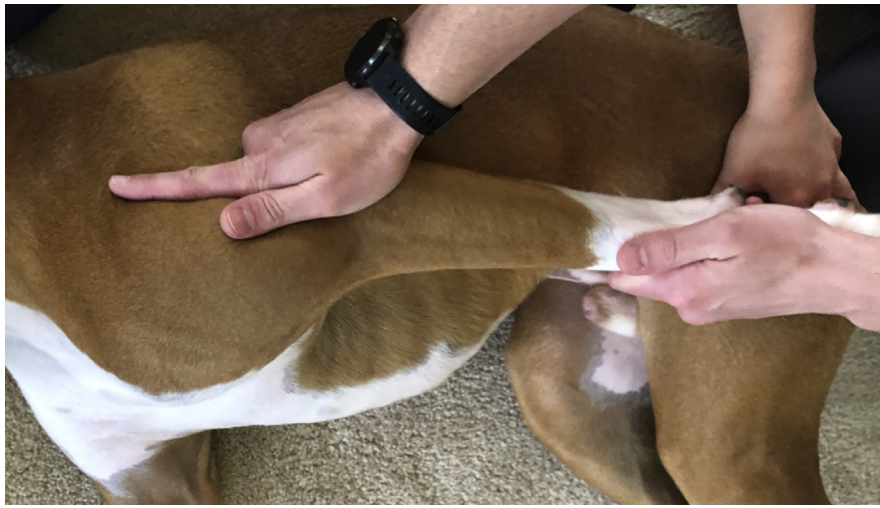

Fig. 5. Biceps stretch test is performed by full flexion of the shoulder and extension of the elbow. The dogs head is to the left.

intertubercular groove.[23] Ultrasound changes consistent with biceps tendinopathy include a sonolucent line around the tendon on the transverse view (**Fig. 7**), enlarged hypoechoic tendon with fiber pattern disruption, and/or bicipital groove irregularities.

Treatment is largely conservative with exercise restriction and formal rehabilitation to facilitate healing and improve tissue flexibility. For moderate or refractory cases, intra-articular therapies (corticosteroids, plasma products, and other biologics) are considered.[20,23,26–29] Should a patient not respond to any intervention, other pathologies should be ruled out prior to recommending surgery. Surgery consists of biceps release (tenotomy) (**Fig. 8**) or tenodesis.[25]

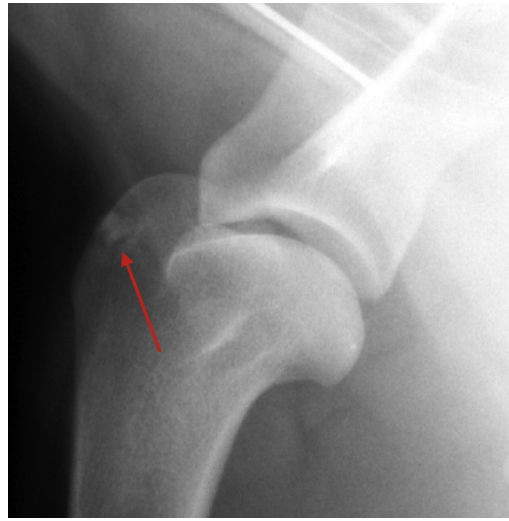

Fig. 6. Lateral shoulder radiograph revealing calcification (*arrow*) in the region of the biceps/supraspinatus.

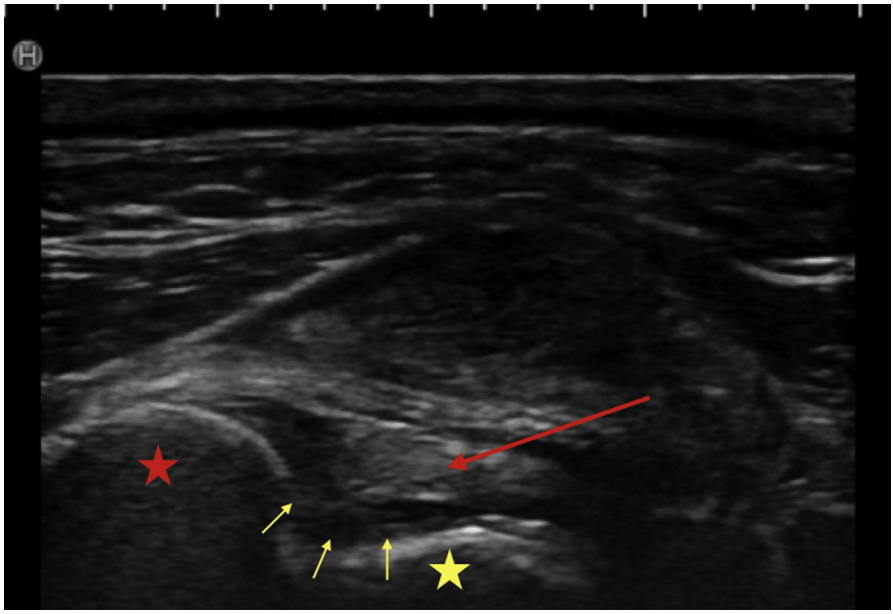

Fig. 7. Musculoskeletal ultrasound of biceps tendinopathy. The biceps (*red arrow*)has a hypoechoic fiber patter at its origin on the supraglenoid tubercle (*red star*). In addition, there is fluid (*yellow arrows*) surrounding the biceps tendon. The yellow star is the bicipital groove for reference. (*Courtesy of* D. Canapp, DVM, Annapolis Junction, MD.)

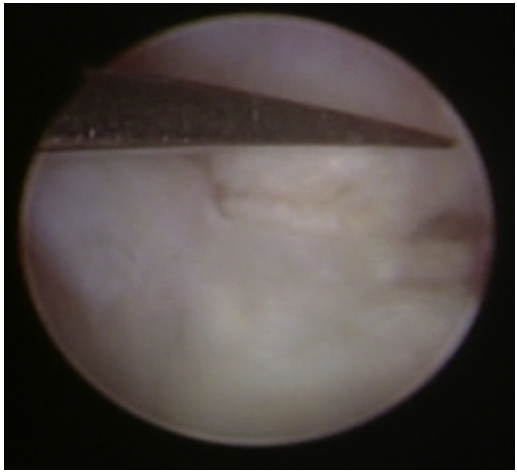

Fig. 8. Arthroscopic biceps tenotomy using arthroscopic scissors.

SUPRASPINATUS TENDINOPATHY

Supraspinatus tendinopathies are documented in humans and to lesser extent in canines with similarities that include the proposed pathogenesis (hypovascularity), clinical evaluation, potential effects on the biceps, and management strategies.[26,30–32] Supraspinatus tendinopathies also can have mineralization.[33] With pathogenesis largely unknown, several theories exist, including hypoxia, degenerative changes due to overuse or concurrent pathology, and metabolic disorders.[26,30] Although distinct areas of hypovascularity or avascularity are documented (similar to humans), the significance is unknown but may lead to fibrocartilaginous transformation.[30] Myxomatous degeneration and/or cartilaginous metaplasia are common histopathologic changes.[33] Similarly, the clinical implication of mineralization is unclear[26,31,33–35] and concurrent disease often is present.[36]

Supraspinatus tendinopathies occur in medium to large breeds, with Labrador retrievers and rottweilers overrepresented.[26,34] History and examination may be similar to biceps tendinopathy. Lameness may be unilateral even with bilateral disease.[33] Supraspinatus tendinopathies resulting from compression of the biceps often are more affected.[37] Examination may include discomfort and tension on shoulder flexion and/or direct supraspinatus insertion palpation.[38]

Similarly, radiographs largely are normal.[33] Ultrasound is a noninvasive tool for confirming supraspinatus tendinopathy and further investigating other periarticular structures (**Figs. 9** and **10**).[32] It can be helpful particularly in differentiating active inflammation within or surrounding the supraspinatus tendon from static mineralization.[32,34,36] Common ultrasound changes include increased tendon size, irregular fiber pattern, nonhomogeneous echogenicity, and/or calcifications (**Fig. 11**).[36] Mistieri and colleagues[32] proposed that MRI is more helpful for investigating concurrent biceps tendon impingement. Care should be used, however, because MRI supraspinatus

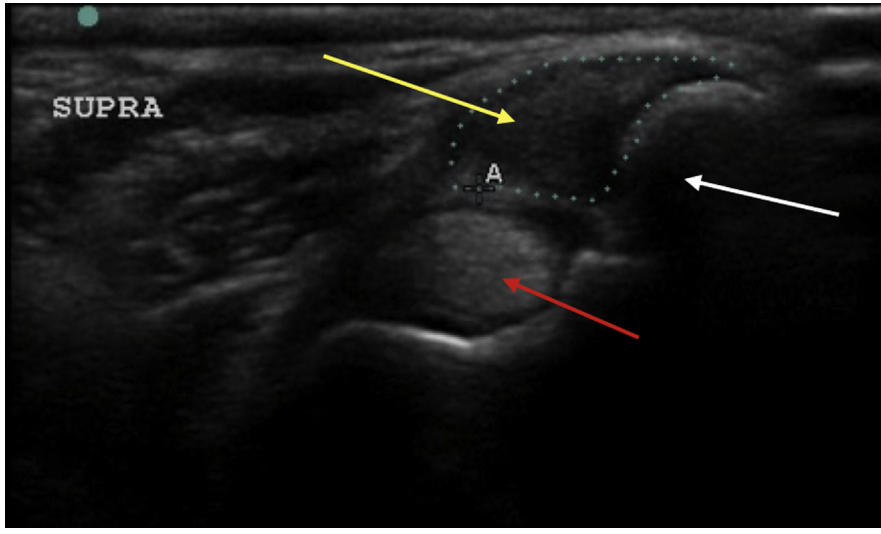

Fig. 9. Musculoskeletal ultrasound image of normal supraspinatus tendon (*yellow arrow*) and biceps tendon (*red arrow*). The white arrow is the greater tubercle of the humerus where the supraspinatus tendon inserts. (*Courtesy of* D. Canapp, DVM, Annapolis Junction, MD.)

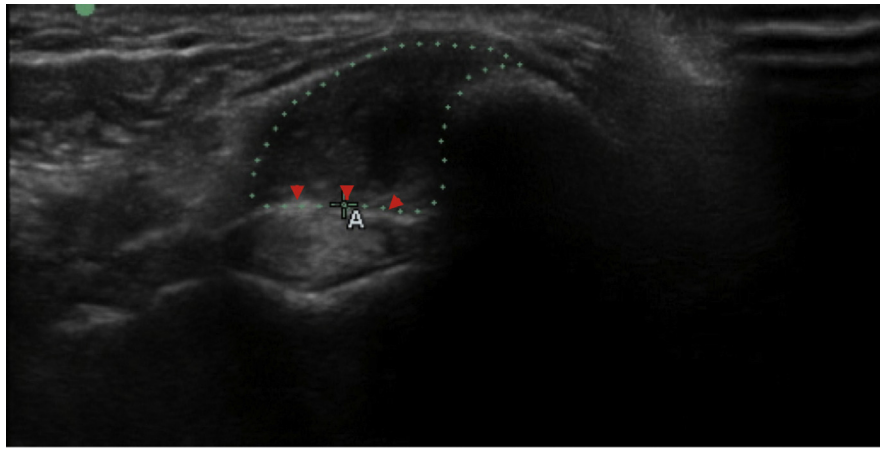

Fig. 10. Musculoskeletal ultrasound image of supraspinatus tendinopathy (*outlined by dots*). The enlarged supraspinatus is compressing the biceps (*arrowheads*) contributing to secondary tendinopathy. (*Courtesy of* D. Canapp, DVM, Annapolis Junction, MD.)

description reveals a normal trilaminar appearance on sagittal and transverse images and should not be mistaken as evidence of a tendinopathy.[39]

Management largely is conservative with exercise restriction, formal rehabilitation, and/or intralesional injections considered. Two studies reported ultrasound appearance and gait analysis improvement after ultrasound-guided injections of adipose-derived progenitor cells and platelet-rich plasma (PRP) or bone marrow aspirate

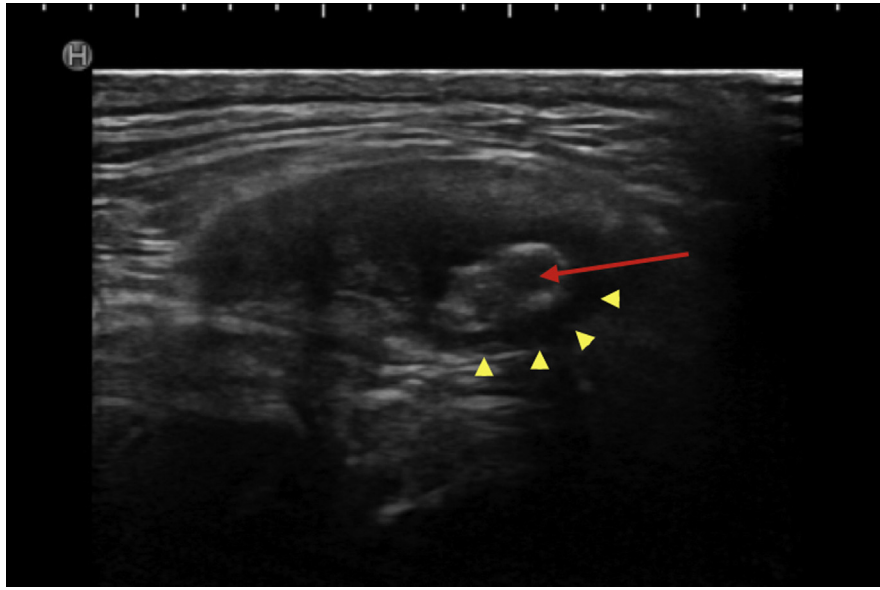

Fig. 11. Musculoskeletal ultrasound of calcified supraspinatus tendinopathy (*arrow*). An anechoic halo (*arrowheads*) surrounds the calcification.

concentrate and PRP.[40,41] Another study found subjective improvement with a single PRP injection in 10 dogs.[42] Additional success with extracorporeal shock wave therapy has been observed.[43] Surgical treatment is controversial, with varying success rates,[31,33,34] and consists of mineralization removal and/or tendon debulking to relieve pressure on the biceps.[26,33,34] Mineralization may recur but may not lead to clinical disease.[34]

INFRASPINATUS CONTRACTURE

The infraspinatus' main function is abduction and rotation of the humerus and to a lesser extent flexion and extension of the joint.[44] The infraspinatus traverses the shoulder laterally, acting as a dynamic stabilizer.[44] Infraspinatus contracture is an uncommon condition overall but is associated with active, medium to large breed dogs, specifically working, hunting, and sporting dogs.[8] Most are affected unilaterally; however, bilateral cases have been documented.[45–47] Although the etiology is unknown, based on histopathology and electromyography testing, it is thought to be a primary muscle disorder resulting from repetitive microtrauma during vigorous exercise rather than an acute trauma or neuropathy.[8,45,48]

History often reveals abrupt forelimb lameness, shoulder discomfort, and/or swelling of the infraspinatus musculature after strenuous activity.[49] Within 2 weeks to 6 weeks, the lameness and discomfort resolve. Fibrosis and contracture, however, result in a nonpainful, characteristic circumducted gait abnormality.[8,44,45,48–50] Range of motion often is limited in pronation and abduction. The affected limb is positioned with the elbow adducted and paw abducted (**Fig. 12**).

Diagnosis is made through history, gait evaluation, and examination. If there is uncertainty, imaging can be completed. Radiographs often are unremarkable; however, calcification may be present.[46,51] Musculoskeletal ultrasound is more useful, especially when additional supraspinatus pathology is suspected.[46,52] Although less common, supraspinatus contracture has been reported and is important to differentiate.[8,49]

Because most cases are found in the contracted phase, the treatment of choice is infraspinatus tenotomy.[8] Following transection, immediate tissue release with characteristic popping sound and full shoulder range of motion should be appreciated.[8] Tenotomy carries a good prognosis for return to full function, including sport and work.[49] Early rehabilitation postoperatively, however, is critical for successful outcome.[20,49]

MEDIAL SHOULDER SYNDROME

The MGL, LGL, and JC comprise the static stabilizers because they do not respond to changes in joint position. The dynamic stabilizers contract and relax in response to changes in joint position and are composed of the periarticular cuff muscles, as described previously.[1,5,7,53,54]

MSS is an important cause of canine forelimb lameness, occurring with much greater frequency than luxations.[5] MSS is defined by abnormal motion or translation of the humeral head within the glenoid fossa.[5,55] Pathology commonly occurs medially (80% of cases)[56]; however, lateral instability and multidirectional instability have been reported.[5,57–60] Pathology occurs when 1 or more stabilizer is affected, the MGL being most common (**Fig. 13**).[57,58] It is unknown, however, which stabilizers and what severity produce clinical signs.[61]

Although MSS is becoming recognized more commonly, diagnosis is difficult to obtain and often achieved through careful examination and diagnostic imaging. MSS occurs in middle-aged, medium to large breeds. Acute, traumatic events have

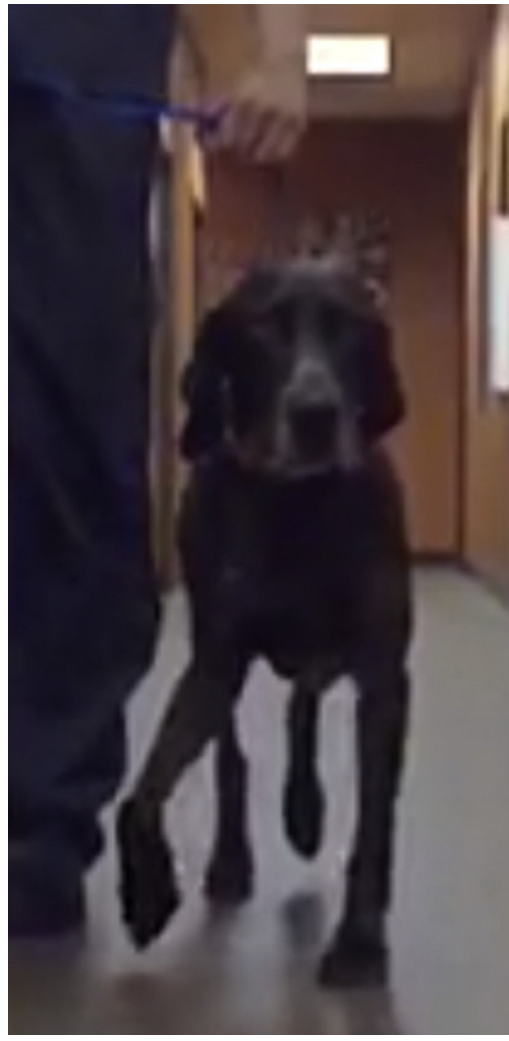

Fig. 12. Characteristic gait of an infraspinatus contracture patient. Note the external rotation of the humerus with elbow adduction and paw abduction during swing phase.

been documented but chronic overuse injuries are suspected of playing a larger role.[5,57,58,62] Chronic forelimb lameness, ranging from subtle and intermittent to severe and continuous (depending on the severity and structures affected), often is described.

Although nonspecific, muscle atrophy and/or pain on manipulation of the shoulder may be appreciated.[5] Various palpation techniques to confirm medial pathology have been postulated. The drawer test was described first; however, it can be technically challenging for unexperienced evaluators, and sedation is required.[5] Additionally, a hyperabduction test was described by Cook and colleagues.[57] This test technically is easier and a good alternative to the drawer test; however, interpretation should be made cautiously between breeds and individuals.

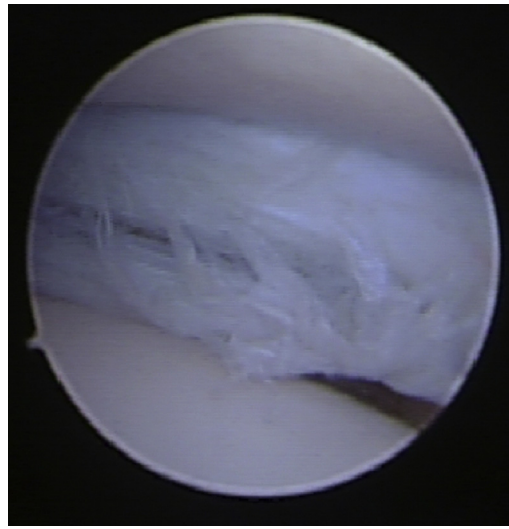

Fig. 13. Arthroscopic image of midbody MGL fraying. For reference, cranial is to the left.

Studies investigating the accuracy and repeatability of the hyperabduction test are conflicting.[7,57,59,62,63] Initially, dogs with medial shoulder pathology had higher abduction angles (>50°) compared with normal shoulder joints; however, arthroscopic shoulder evaluation was not used to define normal/abnormal.[57] Later studies showed similar results of larger abduction angle in affected dogs[57,62]; however, hyperabduction was observed with medial and lateral instability.[62] Additionally, sedation and muscle atrophy can affect measurements.[7] Divitt and colleagues[59] found that both techniques had minimal effects on arthroscopic medial compartment changes. Negative hyperabduction test, however, was 7 times more likely to occur in those without medial compartment changes. Given this, it can be concluded that it is relatively sensitive but lacks specificity.[59] An important aspect when performing the abduction test is the maintenance of shoulder extension. Because joint laxity increases with flexion, inability to maintain extension could elevate the angle falsely.[64] Comparison of contralateral limb angle may be helpful in unilateral disease.[57,63,64] A recent report noted the abduction test has poor interobserver variability, with increasing accuracy achieved by more experienced observers.[64]

Radiographs are performed to rule out boney abnormalities and assess degenerative joint disease (DJD), a majority showing no abnormalities. In the absence of OCD lesion, all shoulder joints with DJD should be investigated for MSS.[5,56] Stress radiography can be helpful; however, results may be similar to hyperabduction tests.[63] Although musculoskeletal ultrasound cannot identify direct pathology to the MGL, LGL, and subscapularis, it may be useful in investigating periarticular pathology.[21,57,62] In contrast, MRI can identify the MGL and subscapularis tendons; however, disease severity often is underestimated compared with arthroscopy.[21] Arthroscopy commonly is used because it allows direct visualization of the intraarticular components (see **Fig. 13**) and dynamic evaluation via probing and range of motion.[5]

MSS management is a topic of debate. To date, no literature supports superiority of surgery over conservative management. For those moderately to severely affected or with medial or multidirectional instability, however, surgery is recommended.[60,65]

Common techniques include open tendon transposition, open subscapularis imbrication, open or arthroscopic-assisted prosthetic repair, and radiofrequency-induced thermal capsulorrhaphy (RITC) (Fig. 14) or arthroscopic thermal capsulorrhaphy.[56,58,65–68]

Tendon transpositions were among the first described for medial stabilization and involve transposition of the biceps brachii tendon of origin or supraspinatus tendon (less common).[6,66,67] Their major drawback is the alteration of joint biomechanics leading to temporary or permanent functional gait abnormality and osteoarthritis.[6,66,67] For cases of intact tendons and mild laxity, subscapularis imbrication alone may be effective.[68] Alternatively, prosthetic repair techniques have shown promise in restoring a more biomechanically similar joint. One study described improved clinical results and minimal gait abnormalities in 9/10 dogs that underwent prosthetic MGL repair using bone tunnels, suture anchors, and monofilament suture.[69] Additionally, an arthroscopic assisted approach using a prosthetic ligament was described in 39 cases of varying severity.[70] Overall complication rate was 15% with no catastrophic complications and 77% return to normal function.[70]

Management of milder pathology largely is conservative with exercise restriction and formal rehabilitation. Some individuals utilize a hobbles vest to prevent shoulder abduction. Intra-articular biologics (platelet products) and extracorporal shock wave therapy (ECSWT) can be used adjunctively. Ideally, ECSWT is utilized initially and again 4 weeks to 6 weeks later.[71] The use and effectiveness of RITC are controversial and adopted from human medicine. With RITC, thermal energy is applied to lax ligaments and JC, causing collagen bundle shrinkage and tightening.[58,72] Although immediate tightening is observed, strict activity restriction and owner compliance are essential because these tissues lose their mechanical properties 2 weeks to 4 weeks after treatment.[58,72,73] Therefore, a non–weight-bearing sling (velpeau) or hobbles to prevent abduction is crucial. Afterward, a strengthening and conditioning program should be implemented to allow controlled stimulus while fibroblast infiltration and proliferation restore mechanical properties and improve muscle mass.[58,72] Subjectively, improved function is expected at 12 weeks to 16 weeks, with optimal improvement 5 months to 6 months following treatment. Initial reports showed 93% good and 79% excellent clinical improvement.[58] More recently, an 80% success rate was

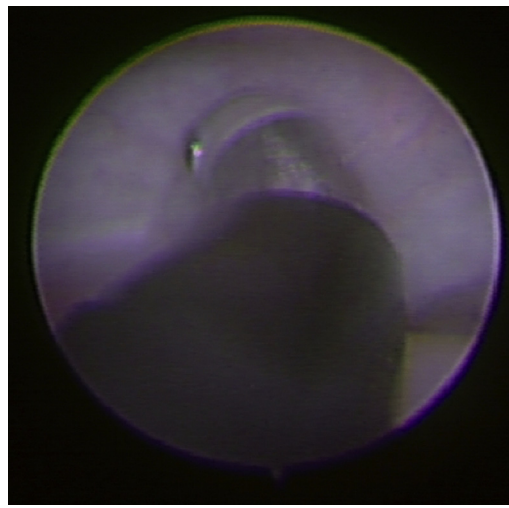

Fig. 14. Arthroscopic MGL RITC using a radiofrequency probe.

described but was not significantly better than nonsurgical management.[60] Contraindications for RITC include lateral, multidirectional, or bilateral instability, overt DJD, complete MGL tears, and neurologic dysfunction.[58] Although RITC has shown some success, based on the data, it cannot be considered superior to other surgical options. Therefore, RITC should be reserved for patients without overt instability that have failed conservative management whereas reconstruction is recommended if gross instability exists.[20]

SHOULDER LUXATION

Shoulder luxation is uncommon, can be congenital or traumatic, and may be acute or chronic in nature. A majority occur medially or laterally, with cranial and caudal luxation occurring with much less frequency. Congenital luxation typically occurs in toy and small breeds, whereas traumatic luxation is common in larger breed dogs.[8,66,74–76] A weight-bearing or non–weight-bearing lameness may be present, depending on the underlying cause, severity, and chronicity. With traumatic luxations, notable localized swelling, bruising, and discomfort may be present. With both, a palpable difference in the distance from the acromion to the greater tubercle may be noted. With medial luxation, the elbow tends to be flexed and adducted with the distal limb abducted and supinated. Lateral luxation shows a similar appearance; however, the distal limb is adducted.[8] Diagnosis is obtained using orthogonal radiographs and help rule out concurrent fractures.

Traumatic Luxation

Although traumatic luxation often occurs medially, large breeds also develop lateral luxation.[66] Prompt diagnosis, reduction, and stabilization are key to a successful outcome regardless of whether conservative management (manual reduction and coaptation) or surgical intervention is pursued. In the acute phase, luxations are relatively easy to manually reduce under general anesthesia, but residual instability may be present due to damage of the supporting structures.[66] Following reduction, coaptation is warranted for continued stabilization wherein a velpo sling is indicated for medial luxation whereas a spica splint is applied for lateral luxations.[76] Following at least 2 weeks of coaptation, continued activity restriction and rehabilitation is indicated for an additional 2 weeks to 6 weeks.[8,20] Those relatively stable after reduction tend to have successful outcomes. Surgery is indicated with unsuccessful manual reduction, failed closed reduction, gross instability after closed reduction, and/or chronic luxation.[8,66,75]

Surgical stabilization typically involves reconstruction or augmentation of the JC and glenohumeral ligamants.[66] Current described techniques include medial or lateral biceps brachii tendon transposition for medial, lateral, and cranial luxations; supraspinatus tendon transposition for medial luxation; and suture augmentation techniques.[66,67,70,76–80] Suture augmentation is preferred because tendon transpositions have a higher tendency of temporary or permanent joint incongruence and subsequent DJD.[6,66,67] With concurrent (peri)articular fractures, surgery also must include fracture reduction and rigid fixation.[81] In a single canine with severe shoulder instability and contralateral elbow luxation, ligament reconstruction and temporary transarticular bridging locking plate was successful.[82] Additionally, augmented repair with a woven poly-L-lactide device has shown experimental promise.[83] Transarticular pinning and lateral capsular reefing are historical techniques and no longer recommended.[66] Salvage procedures include excisional arthroplasty (glenoid and humeral head resection resulting in pseudojoint) and arthrodesis. These procedures may be

indicated with excessive DJD, failed previous stabilization attempts, chronic luxations, and/or when adequate stabilization cannot be accomplished.[8,66,67,76,84]

Full functional recovery is achievable, with those addressed in the acute phase (with conservative management or surgery) generally carrying a good prognosis.[76] Conversely, those undergoing excisional arthroplasty or arthrodesis experience a functional gait abnormality and decreased range of motion.[8,66,75,84]

Congenital Luxation

Congenital luxation occurs in toy and small breeds between 3 months and 10 months of age[66,85] and typically is unilateral; however, bilateral cases are documented. Although much less common, some luxations occur in adults with minor trauma. Luxation almost always is medial.[66,85] Closed reduction often is not successful given the inherent glenoid and/or humeral head abnormalities. Therefore, surgical stabilization with the previously described techniques often are warranted. Additionally, affected animals should undergo sterilization because it may be heritable.[66]

SUMMARY

Shoulder pathologies are a common cause of canine forelimb lameness. Obtaining early and exact diagnosis is critical. Due to the shoulder's complex anatomy and biomechanics, orthopedic and neurologic examination, radiographs, advanced imaging (musculoskeletal ultrasound, CT, and MRI), and/or arthroscopy often are used. In the immature dog, OC/OCD is considered a top differential for forelimb lameness with shoulder discomfort. In the mature dog, diagnostics are used to differentiate biceps and/or supraspinatus tendinopathy and MSS. For mild cases, conservative management with exercise restriction and guided rehabilitation plan[20] should be implemented. Intra-articular and ultrasound-guided injections are considered for mild, nonresponsive cases. Surgery is reserved for moderate to severe or refractory cases. Prompt luxation reduction is key to a successful outcome. Prognosis is good for conservatively managed, acute, traumatic luxation with mild disruption and prompt reduction. Surgery is indicated, however, for severe disruption and chronic or congenital luxation.

CLINICS CARE POINTS

- In young, large to giant breed dogs with shoulder discomfort, a high clinical suspicion for shoulder OC/OCD should exist.
- During biceps stretch test, biceps tendinopathy elicits discomfort and moderate tension whereas biceps tears have a loss of end-feel.
- Biceps and/or supraspinatus tendon mineralization should be interpreted with caution because it does not always correlate with an active problem.
- Infraspinatus contracture elicits characteristic circumducting gait and examination abnormalities (elbow adduction, paw abduction, limited shoulder abduction, and pronation).
- During hyperabduction test, maintaining shoulder extension and comparing contralateral angles increase accuracy.
- Prompt luxation reduction and stabilization are crucial for a successful outcome.

DISCLOSURE

The authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest or nonfinancial interest in the subject matter or materials discussed in this article.

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