medical news for vets

The Benchmark

OCTOBER-DECEMBER 2019

Canine Shoulder Injury: A Difficult Diagnosis to Make

VCA Alameda East Veterinary Hospital

Medical Director's Cut

Dr. Amber Hopkins, DVM, cVMA, DACVAA

Welcome to the one year anniversary of The Benchmark! We hope the material we have been providing has been interesting and educational.

A lot of changes have occurred at Alameda East over the last year. We have hired several ER doctors, allowing us to have staff ER doctors in hospital 24/7. We have hired a new surgeon, Dr. Mandy Rollins, who received her undergraduate degree from the University of Pennsylvania, with a major in psychology and a minor in art history. Dr. Rollins then attended veterinary school in Ireland, receiving her veterinary degree (MVB) from the University College Dublin School of Veterinary Medicine. Following graduation, she completed a one-year rotating internship at the Angell Animal Medical Center in Boston and then a one-year surgical internship at VCA Animal Specialty & Emergency Center in Los Angeles. She then completed a three-year residency in Small Animal Surgery at The Animal Medical Center in New York City and now joins our team with the hope of continually growing the surgical services and availability to our referring community and clients.

We have also added two Criticalists this year, Dr. Christine Guenther and Dr. Sarah Deitschel. Dr. Guenther just celebrated one year with us. Hopefully you have had a chance to chat with her. Dr. Deitschel joined us last month. She completed her undergraduate degree at Augustana College and attended veterinary school at the University of Illinois. This was followed by a one-year small animal rotating internship in San Diego. Her internship was followed by a three-year residency in Emergency and Critical Care at the University of Missouri.

In addition to new staffing, we have recently upgraded our phone system. You may have noticed some hiccups but we think those have been resolved. If you continue to have any issues, please email Savannah.tajon@vca.com.

Lastly, as fall arrives, so does the opening of our new GP building. Coming soon (hopefully mid to late October) all of our General Practice doctors will be moving to a new building located across the parking lot. This move allows the client service and technical teams to focus completely on our Specialty and ER clientele, improving flow and client service. This will also improve the experience of our General Practice clients by providing a more home-like environment and customer service tailored to general practice needs. Be on the lookout for upcoming information on our Open House, anticipated to occur sometime in November.

Lastly, with so many great changes occurring at once here at Alameda, our Annual Symposium has been postponed until after the first of the year. Once we have a date set for that, we will be sending Save the Date postcards.

As always, we appreciate each and every one of you and your consistent support and referral. If you need anything at all or have suggestions for upcoming CE topics, please let me know. I always enjoy speaking with and getting to know better our referring clientele. Have a great Fall and see you soon!

Sincerely,

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Medical Director

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Canine Shoulder Injury: A Difficult Diagnosis to Make

John Stephan, DVM, MS, DACVS

Part 2 of a 2 part series (Continued from Part 1 in the April 2019 edition of The Benchmark)

"Supraspinatus tendinopathy can present as an isolated condition or be diagnosed with concurrent biceps tendon injury or medial shoulder pathology."

Supraspinatus Tendinopathy

Supraspinatus tendinopathy (ST) is a common condition in humans, particularly those active in sports involving repetitive overhead motion such as swimming and tennis. It is also a common cause of forelimb lameness in performance, working and active companion dogs. Supraspinatus tendinopathy can be challenging to diagnose and treat, with recurrence of pain and lameness being common.

In dogs, the most common cause of ST is tendon overuse from long-term repetitive activity. Activities that involve landing with outstretched forelimbs, quick, cutting-like turns or jump-turn combinations place shoulder joint components under extreme stress. These maneuvers are often routine for companion animals (chasing a squirrel or fetching a ball), canine athletes (agility or flyball) and working dogs (canine officers, hunting or herding).

Repetitive activity puts increased biomechanical load on the ST tendon, eventually leading to a strain injury. If the condition progresses, scar tissue buildup within the tendon, referred to as a supraspinatus bulge, often develops causing compression to the adjacent biceps tendon and increased patient discomfort.

Clinical Signs

Patients with ST may be presented early or late in duration of their condition; usually when lameness is observed. Unilateral lameness is most common, although bilateral lameness is present in about one-third of patients. Some rather astute owners will recognize a subtle abnormality, such as shortening of a forelimb's swing phase observed during gait analysis or an unexplainable decline in athletic performance prior to noticeable lameness. Often, these owners are engaged in canine sports or working duties with their dogs on a regular basis. Patients asymptomatic at presentation can exhibit lameness following exercise. Often, lameness does not improve with rest and NSAIDs.

Diagnosis

Supraspinatus tendinopathy can present as an isolated condition or be diagnosed with concurrent biceps tendon injury or medial shoulder pathology. Its diagnosis relies predominantly on orthopedic evaluation and diagnostic imaging. During orthopedic exam, direct pressure of the supraspinatus tendon can elicit pain. Discomfort, with or without tendon spasm, may be noted with shoulder flexion or extension. In more severe or chronic situations, the belly of the Supraspinatus m. may be atrophied; revealing the scapular spine.

In chronic conditions where a supraspinatus bulge impinges the biceps tendon, pain is often caused with direct pressure applied to the biceps tendon or the biceps muscle is stretched (shoulder flexion with elbow extension). Ultrasound exam can differentiate whether biceps discomfort is due to tendinitis from impingement or more severe biceps tendinopathy.

Forelimb abduction causes lateral (outward) rotation of the humeral head. If discomfort and tendon spasm occur during this motion, examination for the presence of concurrent medial shoulder instability should be performed.

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Diagnostic imaging includes radiographic imaging of the shoulder. Radiographs are useful to rule out or reveal presence of other bone and joint pathology. Mineralization of the supraspinatus tendon and/or bony remodeling at its insertion can be radiographically evident. In some cases, mineralization is noted in both shoulders, even when a patient presents with unilateral lameness.

To be complete, elbow radiographs can be performed to identify elbow joint incongruency and/or other pathology that may contribute to a patient's clinical condition. Elbow pathology is not uncommon in patients diagnosed with ST.

Musculoskeletal ultrasonography is highly effective for ST diagnosis as well as treatment response monitoring. Its benefit of enabling contralateral limb evaluation is particularly valuable in dogs with bilateral forelimb lameness, reported in greater than 37% of dogs with ST.

Treatment

Supraspinatus tendinopathy is notoriously difficult to treat. Acute ST treatment consists of conservative medical management (controlled activity and NSAIDs) and rehabilitation therapy (laser therapy, acupuncture, passive ROM with progression to stretching and strengthening exercises) as described for acute BT. However, ST conditions are often refractory to medical management and rehabilitation therapy.

Surgery is recommended for refractory and chronic cases of ST. Surgical treatment includes resection of mineralized and degenerative tissue foci and transverse humeral ligament release. Excellent functional outcomes have been reported in up to 50% of non-mineralized ST and 64% of mineralized ST. Arthroscopic evaluation should also be considered in surgical planning to identify and treat possible concurrent BT and/or shoulder instability.

More recent research evaluating response to intralesional injection of stem cells coupled with platelet-rich plasma (PRP) has been promising. Platelet-rich plasma provides a fibrin scaffold required for stem cell survival as well as growth factors that assist with stem cell activation and differentiation. Ultrasound guidance is used to identify tendon pathology and direct an intratendinous injection of combined stem cells and PRP.

Medial Shoulder Instability

When shoulder pathology is severe enough to cause joint laxity that initiates clinical signs, the condition is termed shoulder joint instability. Shoulder joint instability in the dog can be medial, lateral or multidirectional with medial shoulder instability (MSI) being the most common type. Instability can result from a single traumatic event. The more common situation is chronic repetitive use resulting in microtears, degeneration and eventual breakdown of tissue leading to instability. The most severe form of shoulder joint instability is luxation, which most often results from trauma.

Medial shoulder instability results from isolated MGL tearing, laxity or avulsion or MGL damage combined with tendinopathies and/or joint capsule tearing. A grading system for MSI has been described. Grade 1 or mild MSI is defined as laxity without gross tearing of the MGL or supraspinatus tendon (SST), Grade 2 or moderate MSI is defined as partial tear of the MGL, SST or both, Grade 3 or severe MSI is defined as complete tear of the MGL, SST, or both and Grade 4 or luxation is complete displacement of the humeral head in relation to the glenoid cavity.

Clinical Signs

Shoulder instability in general may present as a subtle change in performing a particular movement or more severe as weight-bearing lameness. The most common clinical sign with MSI is unilateral lameness localized to the shoulder joint. Majority of patients' conditions are commonly worse after exercise or heavy activity.

Diagnosis

A presumptive diagnosis of MSI is made on orthopedic evaluation and diagnostic imaging. Orthopedic evaluation including gait analysis is necessary for patients with suspected shoulder instability. Gait abnormalities may range from a slight shortening of stride in the affected forelimb to significant weight-bearing lameness. Shoulder muscle atrophy and decreased forelimb circumference may be noted in more chronic cases. Dogs with MSI typically have a restriction and decreased ROM during shoulder extension.

The normal shoulder abduction angle of the dog is approximately 30-33°. In dogs with MSI, abduction angles average around 50°and can exceed 65°; short of luxation. Pain and spasm are almost always noted during shoulder

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abduction. In more severe cases of instability, abduction may reveal a slip or "thud" from humeral head subluxation. If concurrent ST is present, discomfort may be elicited during shoulder flexion, stretching the supraspinatus m., and direct pressure applied to the supraspinatus insertion.

Suspect MSI may be supported by advanced imaging with detection of MGL, supraspinatus or other structural damage. Radiographic findings with MSI are often unremarkable.

Definitive diagnosis of MSI is made by systematic arthroscopic assessment of the shoulder joint while performing dynamic testing. Arthroscopic verification of damage to the MGL, SST, joint capsule or a combination of these structures can be followed with treatment.

Treatment

Medial shoulder joint instability can be managed with or without surgery. Nonsurgical MSI management consists of the use of administration of NSAIDs, hobbles to prevent limb abduction and extensive physical rehabilitation. Nonsurgical management may be offered to patients with mild instability (abduction angle of 35° to 45°) and arthroscopic findings consisting of mild pathology (inflammation without fraying or disruption of the MGL and joint capsule).

However, nonsurgical management is not reported to be as successful as surgical treatment because of continued joint laxity and stress on associated soft tissues. In one study, dogs treated by surgical reconstruction were 3 times more likely to have a successful outcome as dogs treated without surgery.

Surgical stabilization of the shoulder joint is indicated in moderate to severe MSI and cases where previous nonsurgical treatments have failed. Surgical MSI treatments include arthroscopically assisted radio frequency-induced thermal capsulorrhaphy, medial joint imbrication through an open approach and stabilization with use of a prosthetic ligament via an open approach.

Radio frequency-induced thermal capsulorrhaphy is a safe, effective and minimally invasive method for treating MSI and can be considered in mild and moderate cases. There is limited data available for MSI treated with thermal capsulorrhaphy in dogs. Two studies have reported improved limb function following treatment. However, patients typically did not reach improved function until 12 to 16 weeks after treatment and did not reach optimal function until 5 to 6 months after treatment.

Moderate to severe case of MSI or shoulder joint luxation do not benefit from thermal capsulorrhaphy treatment, as the pathological changes most commonly consist of complete tears of the MGL and severe disruptions or tears of the SST.

Moderate to severe MSI requires reconstruction of the medial joint compartment by primary stabilization. Arthroscopic findings usually include complete tears of the MGL and severe disruption of the subscapularis tendon and joint capsule. Following prosthetic ligament stabilization, patients are placed in a customized hobbles for greater than four weeks followed by a shoulder support system for two to three months. They are enrolled in a rehabilitation program during this time. Patients return to full activity approximately 16 to 20 weeks after the procedure and return to competition by approximately six months.

Conclusion

Dog owners know their pets' behavior, activities and personalities better than any other individual. However, limited understanding of joint disease and the nonspecific and subtle manner that shoulder pathology often reveals itself clinically, make it quite challenging for owners to recognize problems in a timely manner. Nevertheless, a dog presented with a nonspecific alteration to its biomechanics, with or without noticeable forelimb lameness, is an opportunity to evaluate shoulder health and provide appropriate treatment and rehabilitative therapy to any diagnosed tendinopathy and/or joint instability.

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Update on Treatment of IMHA in Dogs

Katherine Scott, DVM, DACVIM (SAIM)

A new consensus paper has just been published by ACVIM on treatment of immune-mediated hemolytic anemia (IMHA) in dogs. Consensus papers are written by a group of experts in the topic, to help provide evidence-based recommendations when possible, and add personal comments when there is a lack of evidence to support a firm conclusion. The entire paper is available to read online, but the key conclusions are listed below. Enjoy!

1. Administration of transfusions should be performed as needed. There is no evidence that transfusion(s) increase risk of mortality.
2. After diagnosis of IMHA is confirmed, prednisone or prednisolone should be used at an initial dose of 2-3mg/kg/day. If the dog is >25kg, a dose of 50-60mg/m2/day should be used. This can be once or divided twice daily. Dexamethasone 0.2-04.mg/kg/day is an IV alternative.
3. A second immunosuppressive may be given at the start of therapy to decrease steroid dose required. This should be strongly considered when the dog is severely ill from IMHA, PCV continued to decline quickly, or if serious prednisone side effects are anticipated (as may occur in >25kg dogs).
4. Suggested secondary immunosuppressive options include azathioprine (2mg/kg/day then qod after 2-3 weeks), cyclosporine (5mg/kg q12h), and mycophenolate (8-12mg/kg q12h).
5. Human immunoglobulin (IVIG) can be a salvage measure in non-responders already on 2 immunosuppressives, but is not recommended for routine use.
6. Avoid use of 3 or more immunosuppressive drugs at the same time.
7. When PCV is stable at >30% for 2 weeks and evidence of active IMHA (spherocytosis, agglutination, increased bilirubin) has improved, prednisone dose should be decreased by 25%.
8. If PCV continues to remain stable and >30%, then continue to decrease the dose of prednisone by 25% every 3 weeks. A greater % reduction can be considered if a second drug is being used.
9. Recheck PCV before any dose reduction to confirm continued response to treatment.