Supraspinatus tendinosis associated with biceps brachii tendon displacement in a dog

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- In humans, tendinosis is a common problem that is characterized by persistent, localized, activity-related pain and swelling associated with the common calcaneal (Achilles), patellar, and supraspinatus tendons and generally caused by overuse.
- Diagnosis of supraspinatus tendinosis in dogs may be made on the basis of similarities in clinical, magnetic resonance imaging, and arthroscopic findings to those reported in affected humans.
- Histologically, affected tendons of humans are characterized by disorganized and disrupted collagen fibers (typified by loss of reflectivity under polarized light), an increase in mucoid matrix, fibrocartilaginous metaplasia of tenocytes, and hypercellularity (fibroblastic-appearing cells).

A 4-year-old 17.7-kg (38.9-lb) spayed female Australian Cattle Dog (Blue Heeler) was evaluated at the Washington State University Veterinary Teaching Hospital because of right forelimb lameness of 5 months' duration. Prior to development of lameness, the daily activity level of the dog was high; activities included daily 2-hour runs (off leash), fetching thrown items, and chasing turkey or deer on the owners' large property. No known trauma or incident was associated with the onset of lameness. Treatment provided prior to evaluation at the hospital included rest, chiropractic manipulation, and administration of carprofen (2.2 mg/kg [1.0 mg/lb], PO, q 12 h for 14 days) followed by treatment with naproxen (dose unknown, PO, for 3 days) and later prednisone (0.28 mg/kg [0.13 mg/lb], PO, q 12 h for 2 days). None of the treatments improved the dog's lameness. Because administration of naproxen was associated with vomiting and inappetence and administration of prednisone was associated with diarrhea, those treatments were discontinued. The dog intermittently had mild pruritus, which the owners presumed to be a result of atopy and had not pursued further. Otherwise, the dog was in good health; vaccination status was current.

Other than the orthopedic abnormalities, the physical examination findings were unremarkable. Orthopedic evaluation revealed a consistent, noticeable, weight-bearing lameness and mild atrophy of supraspinatus and infraspinatus muscles of the right forelimb. Signs of pain were elicited during flexion of each shoulder joint and via digital pressure in the intertubercular grooves. Signs of pain were also associated with maximal extension of the right shoulder joint. Results of drawer tests[1] and abduction of each forelimb (with the shoulder and elbow joints at standing and extended angles) suggested that both shoulder joints were stable. Maximal shoulder joint flexion during elbow joint extension in combination with digital pressure over the biceps brachii tendon was resisted. Results of the remainder of the orthopedic evaluation were within normal limits, and findings of a neurologic examination were unremarkable.

Initial diagnostic testing included a CBC and serum biochemical analyses; no clinicopathologic abnormalities were detected except eosinophilia (648 eosinophils/μL; reference range, 0 to 100 eosinophils/μL). Radiography of the shoulder regions (mediolateral and craniproximal-craniodistal [skyline projection] views) revealed no abnormalities.

A force plate[a] analysis was performed. Of the 66 force plate readings obtained, 9 readings for each body side were considered acceptable; readings were excluded if the dog's velocity over the force plate was < 1.6 or > 2.0 m/s; acceleration or deceleration exceeded 0.5 m/s; or 3 feet, rather than the ipsilateral 2, contacted the plate. The mean ± SD peak force, expressed as percentage body weight, for the left forelimb, right forelimb (the affected limb), left hind limb, and right hind limb was 252 ± 22, 219 ± 33, 163 ± 17, and 163 ± 12 (100•N/N), respectively; the corresponding impulse area was 31 ± 2.7, 28 ± 5.3, 17 ± 1.7, and 18 ± 1.8 (100•N-s/N), respectively.

Magnetic resonance (MR) images of each shoulder joint were obtained by use of an MR scanner (1.0 T)[b] with a quadrature spine coil designed for humans. Sagittal and transverse images were obtained from the midscapula through midhumerus regions. The sagittal images were oriented parallel to the craniocaudal axis of the scapula and humerus, and the transverse images were oriented perpendicular to the axis of the biceps brachii tendon, proximal to the intertubercular groove. Bilateral sagittal sequences included **T2-weighted gradient echo (T2*-W)** images; **proton density spectral presaturation inversion recovery (PD-SPIR)** images; T1-weighted fast-field echo, 3-dimensional acquisition gradient echo images; and **short tau inversion recovery (STIR)** images. In addition, 3-dimensional SPIR images of the right shoulder joint were obtained. In the axial plane, T2*-W images were obtained bilaterally. Evaluation of the various image sequences of the left and right shoulder areas revealed similar findings. At its insertion on the greater tubercle, the supraspinatus tendon was large with a masslike appearance (20 × 7 mm in the right forelimb and 13 × 5 mm in the left forelimb) and extended into the intertubercular groove. The supraspinatus tendon mass had an increased signal on T2*-W, PD-SPIR, and STIR images,

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compared with the biceps brachii tendon, and typical tendon appearance that was generally consistent with increased fluid and proton content (**Figure 1**). Imaging of the **supraspinatus musculotendinous junction (SMTJ)** revealed streaks wherein the signal was decreased in intensity, which is consistent with fat. The mass compressed and displaced the biceps brachii tendon medially. The tendon displacement was mild in the left forelimb but more pronounced in the right forelimb; in the latter, the tendon was displaced partially out of the groove onto the lesser tubercle (**Figure 2**). Thickening of the transverse humeral ligament was detected bilaterally, and a small amount of effusion was evident in the biceps brachii tendon sheath in both forelimbs. On the basis of these findings, degenerative or inflammatory processes were suspected as the cause of a mass in the supraspinatus tendon bilaterally, which resulted in the compression and displacement of the biceps brachii tendons (right forelimb being affected more severely than the left forelimb).

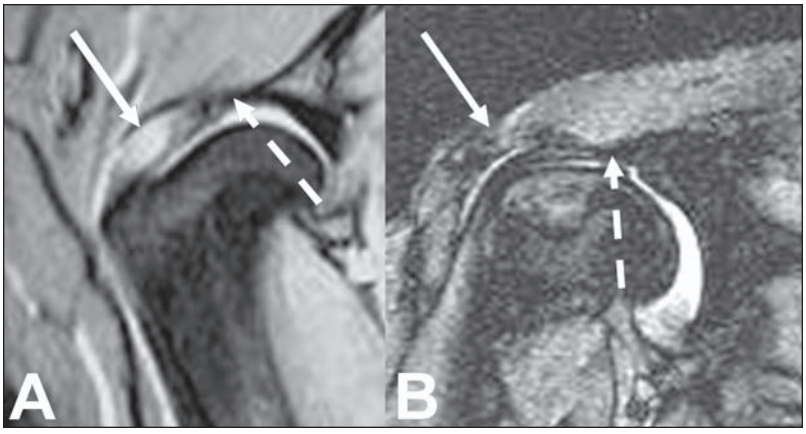

Figure 1—Sagittal T2-weighted magnetic resonance images of the right shoulder region of a dog that was evaluated because of right forelimb lameness of 5 months' duration (A) and that of a cadaver of a clinically normal dog (B). In the image from the lame dog, notice that the tendon of insertion of the supraspinatus muscle has increased mass and signal intensity (solid arrow), compared with the biceps brachii tendon, which is consistent with an abnormally high fluid content. The tendon of origin of the biceps brachii muscle is visible (dashed arrow). In the image from the clinically normal dog, notice the low signal intensity representative of normal mass in the biceps brachii tendon (dashed arrow) and the tendon of insertion of the supraspinatus muscle (solid arrow).

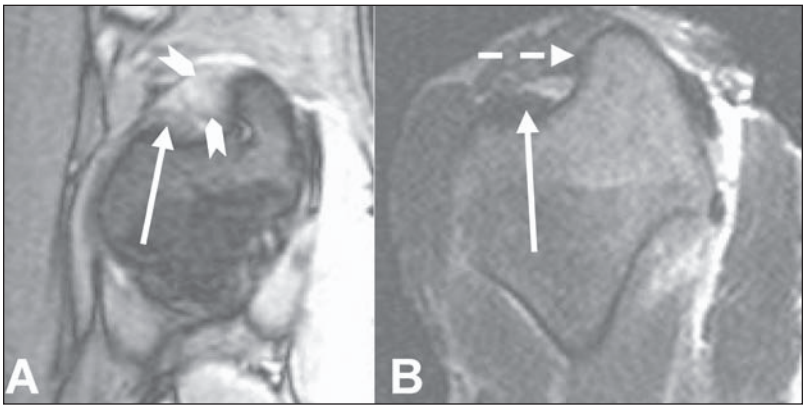

Figure 2—Axial T2-weighted magnetic resonance images of the proximal portion of the right humerus in the lame dog in Figure 1 (A) and that of a cadaver of a clinically normal dog (B). In the image from the lame dog, notice that the tendon of insertion of the supraspinatus muscle has increased mass and signal intensity, compared with the biceps brachii tendon, which is consistent with increased fluid content (chevrons). The biceps brachii tendon (solid arrow) is displaced onto the lesser tubercle and partially out of the intertubercular groove. In the image from the clinically normal dog, notice that the signals in biceps brachii tendon (solid arrow) and supraspinatus tendon (dashed arrow) are of low intensity.

The following day, the dog was anesthetized and placed in dorsolateral recumbency; an arthroscopic examination of each shoulder joint was performed. The surgical procedure has been described previously.[2] A 2.3-mm, 30° fore oblique arthroscope[c] was introduced through a 2.8-mm sleeve into the joint 1 cm caudal and 1.5 cm ventral to the acromion. A xenon light source[d] was used for illumination, and images were captured by use of a medical digital camera[e] with a 24-mm objective lens. Joint distension was maintained via infusion of lactated Ringer's solution (1.8 L/min) with a lavage pump.[f] The medial glenohumeral ligament, subscapularis tendon, articular cartilage of the glenoid cavity, humeral head and intertubercular groove, synovium of the glenohumeral joint and biceps brachii tendon sheath, and the biceps brachii tendon and its insertion on the supraglenoid tubercle were viewed and considered normal in appearance. After the arthroscopic examination of each joint, the ipsilateral supraspinatus tendon was explored by use of an approach similar to that previously described for evaluation of supraspinatus mineralization.[3] Briefly, the insertion of the supraspinatus tendon was exposed through a 5- to 7-cm longitudinal incision through the skin and underlying brachiocephalicus muscle, cranial to the greater tubercle. The supraspinatus tendon, the SMTJ, and the distal muscle belly of the supraspinatus muscle were thoroughly palpated and inspected from the superficial and deep aspects. The insertion of the supraspinatus tendon had lost the typical glistening white appearance; it was pink-yellow and appeared more voluminous than normal. Guided by the information obtained via MR imaging, excess tissue was excised with the goal of removing the abnormal part of the supraspinatus tendon, thereby allowing the biceps brachii tendon to resume its normal position in the intertubercular groove. The cut surfaces of the supraspinatus tendon were pale yellow; the tendon fibers had lost their typical longitudinal formation and were instead arranged in an erratic manner that resembled unorganized fibrous tissue. The longitudinal defect in the supraspinatus tendon was closed by use of size 0 polydioxanone suture in an interrupted cruciate suture pattern, and the remainder of the closure was performed in a routine fashion. The resected tissue was placed in neutral-buffered 10% formalin for subsequent histologic examination.[g]

Histologic examination of H&E-stained sections of the tissue specimen revealed discontinuous and disorganized

collagen fibers that lacked reflectivity under polarized light. The most prominent finding was severe myxomatous degeneration, with the remaining collagen separated by a palely basophilic myxoid matrix and edema. The mucoid nature of the matrix was verified by use of Alcian blue stain. Individual small spindloid (fibroblastic) cells were scattered throughout the matrix. Subjectively, the number of cells was increased in the tendon, but because of the separation of the cells by the myxomatous matrix accumulation, an abnormally high cell density could not be diagnosed.

The following day, the dog was discharged from the hospital and the owners were instructed to provide strictly restricted activity (limited to leash walks of 5 to 10 minutes' duration 4 times daily) and passive range of motion exercises of both shoulder joints (at least 10 minutes 3 times daily) for 3 weeks. During the subsequent 3-week period, the owners were instructed to gradually increase the duration of the leash walks to 25 minutes each. Swimming was discouraged for several months as that activity has been suggested[3] to constitute excessive load on a healing SMT unit.

Four weeks after surgery, a golf ball–sized fluctuant mass was detected under the healed incision. The mass, which had developed after the dog had escaped from confinement to chase a squirrel, was presumed to be a seroma and resolved without treatment. The owners reported that the dog's forelimb gait was initially short strided after surgery, but that no lameness was detectable within 2 weeks. Four months after surgery, the dog had returned to its normal activity level. Seventeen months after surgery, a gait evaluation was performed by one of the authors (BAF) via review of videotape footage, which revealed a normal gait during walking, trotting, and loping. Twenty-two months after surgery, the owners reported that the dog's high activity level was maintained.

To compare the MR imaging and histologic findings in the dog of this report with potential age-related changes in dogs without forelimb lameness, 6 shoulder joints from 3 canine cadavers were examined; these dogs were euthanatized by IV injection of pentobarbital solution for reasons unrelated to joint disease. Dog 1 was a 9-year-old 45-kg (99-lb) castrated male Golden Retriever, dog 2 was a 7-year-old 32-kg (70-lb) mixed-breed spayed female, and dog 3 was a 10-year-old 50-kg (110-lb) castrated male Golden Retriever. The shoulder regions of these dogs were collected within 3 hours of euthanasia, cooled, and stored at 4°C (40°F) for 3 days until MR imaging was performed to obtain T2-weighted, PD, and STIR image sequences. Immediately after MR images were obtained, biopsy specimens of the supraspinatus tendons were collected. Examination of the sagittal T2-weighted and STIR images of these 6 shoulders revealed homogenous hypointense signal, typical for normal tendons, in both the supraspinatus tendon and biceps brachii tendon (Figure 1). In PD and T2-weighted axial images, a well-defined hypointense bone signal at the supraspinatus enthesis on the medial and cranial greater tubercles was identified (Figure 2). The biceps brachii tendons in all shoulders were well defined and positioned in the intertubercular groove. Histologic examination of these tendons revealed longitudinally organized collagen fibers with good reflectivity under polarized light. Stainable chondroid or mucin ground substance was minimal or absent, and a low density of primarily mature tenocytes was observed.

In humans, several degenerative disorders in the insertion of the supraspinatus tendon have been identified, including rotator cuff tears, calcifying tendinitis or tendinosis, and tendinosis as a result of overuse. Degeneration of the supraspinatus tendon has been suggested[4,5] to be a factor in the development of rotator cuff tears in humans. An avascular zone (the so-called critical zone) of this tendon is located close to the enthesis in humans and dogs,[6-8] and this localized area of relative ischemia has been suggested[7,8] to be a cause of tendon degeneration with subsequent tearing in this region.

Calcifying tendinitis or tendinosis of the supraspinatus tendon is a common disorder of unknown etiology in humans, which appears to share many features of supraspinatus mineralization in dogs.[9-11] In both species, cartilaginous metaplasia in the critical zone appears to precede or be associated with calcium deposition in the tendon[11,12]; the calcium deposits are not always associated with clinical signs and may constitute incidental findings.[10,12-14]

Tendinosis is a chronic degenerative tendinopathy of unknown etiology that commonly affects the common calcaneal (Achilles), patellar, and supraspinatus tendons in humans.[15-17] Overuse injury has been suggested[15,17] as the cause of this disorder, and the role of overuse in the pathogenesis has been supported by findings of experimental studies.[18,19] Clinically, tendinosis is associated with long-standing, activity-related pain and swelling, which are often refractory to treatment.[15] Histologically, affected tendons contain discontinuous and disorganized collagen fibers that lack reflectivity under polarized light and an abnormally large amount of myxomatous ground substance; fibrocartilaginous metaplasia of tenocytes is detectable. Compared with unaffected tendon tissue, affected tendons have an increased number of cells with a fibroblastic or myofibroblastic appearance, which are often accompanied by prominent capillary proliferation. Typically, no inflammatory cells are detected.[15]

The clinical and histologic findings in the dog of this report are similar to those in humans with tendinosis. Clinically, the dog had a long-standing, activity-related lameness that was refractory to medication and short periods of rest. Histologically, the most prominent findings included a considerable increase in myxomatous ground substance, compared with the amount typically detected in unaffected tendon tissue, and discontinuous and disorganized collagen fibers that lacked reflectivity under polarized light; subjectively, the number of cells of fibroblastic appearance was increased. However, capillary proliferation was not detected. On the basis of clinical and histologic features and the lack of inflammatory changes or other abnormalities in the shoulder joints in the dog of this report, a diagnosis of supraspinatus tendinosis was made.

In humans, the degenerative changes in tendinosis are associated with localized pain,[15] and it has been suggested[16] that 3 to 6 months of continuous rest is necessary to combat collagen breakdown and promote

recovery from chronic tendinosis. After rest periods of such long duration, approximately 80% of affected humans return to athletic function.[16] Surgical treatment of this disorder consists of resection of abnormal tissue, and as many as 85% of treated patients return to full function.[16] Because of the small difference in treatment success between surgical intervention and conservative management, surgery has been considered the treatment of last resort for humans with tendinosis.[16] The lack of inflammatory changes associated with tendinosis minimizes the beneficial effect of steroidal or nonsteroidal anti-inflammatory treatments, and injections of corticosteroids in or around the tendon inhibit collagen repair.[16]

In the dog of this report, the reason for the lameness is unclear. It is possible that the degenerated tendon itself or pressure exerted by the increased tendon mass on structures in the intertubercular groove was a cause of pain. The biceps brachii tendon in the lame right forelimb was partially displaced out of the groove, whereas the biceps brachii tendon in the left forelimb was displaced to a lesser degree. It seems reasonable that if tendon degeneration was the sole reason for lameness, the dog would have had an altered forelimb gait bilaterally. However, it appears generally accepted that bilateral bone or joint disease (eg, osteochondritis dissecans, elbow dysplasia, and panosteitis) can be associated with unilateral clinical signs. Mild disuse atrophy was detected in the dog's right forelimb only, and the results of the force plate testing and gait evaluation suggested that the right forelimb was clinically affected and the left forelimb was subclinically affected by tendinosis.

In the dog of this report, the reason for recovery of full function and return to high-level activity after surgery is also unknown. During surgery, the degenerated part of the tendon was resected, but in humans, this procedure appears to be associated with an extremely limited increase in treatment success rate, compared with conservative management of the disorder.[16] Furthermore, the dog was not strictly rested for 4 to 6 months after surgery, as suggested[16] for humans following surgical treatment of tendinosis. However, impingement on and displacement of other structures in the shoulder joint have not been described in humans with supraspinatus tendinosis. It is possible that the main cause of lameness in the dog of this report was the impingement of the supraspinatus tendon mass on the biceps brachii tendon. The biceps brachii tendon is restricted in its medial translation by the transverse humeral ligament; during its range of motion, it may be affected by increased pressure and friction associated with the supraspinatus tendon mass. Consequently, the main reason for improvement after surgery may be the decompression of the biceps brachii tendon. Another possibility is that the surgical incision stimulated an inflammatory response accompanied by neovascularization that improved healing of the degenerated tendon. However, results of recent studies[20,21] of surgical treatment of patellar tendinosis in humans do not support this theory, and to our knowledge, there is no information in the literature regarding surgical treatment of tendinosis in dogs.

Prior to surgery, the dog of this report had been treated with rest without visible improvement but had not been consistently or strictly rested for a prolonged time. It is possible that the dog could have recovered with a strict rest regimen implemented over several months. Other than rest, several physical therapy procedures may be of benefit in humans with tendinosis, including laser treatment, high-voltage galvanic stimulation, application of load-decreasing devices, and strengthening exercises.[16] None of these procedures had been implemented prior to surgery in the dog of this report.

As the findings in the dog of this report illustrate, tendinosis in dogs and humans appears to have many similar clinical and histologic features. In this particular dog, supraspinatus tendinosis was apparently the cause of biceps brachii tendon displacement; the resolution of clinical signs of lameness after resection of the degenerated tendon suggests that subsequent decompression of the intertubercular groove and the biceps brachii tendon contributed to treatment success.

a. AMTI biomechanics force platform, Advanced Mechanical Technology Inc, Watertown, Mass.
b. Philips Gyroscan NT, Philips Medical Systems, Andover, Mass.
c. Stryker Endoscopy, San Jose, Calif.
d. X-6000 xenon light source, Stryker Endoscopy, San Jose, Calif.
e. 888 medical video digital camera, Stryker Endoscopy, San Jose, Calif.
f. Continuous Wave II arthroscopy pump, Arthrex Inc, Naples, Fla.
g. Washington Animal Disease Diagnostic Laboratory, Pullman, Wash.

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