Diagnostic findings in a lame-free dog with complete rupture of the biceps brachii tendon

A case report in a unilaterally affected working Labrador Retriever

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Summary

A case of a complete rupture of the biceps brachii tendon in a lame-free three-year-old Labrador Retriever is described. The dog had a history of chronic lameness following a trauma, but had become lame-free shortly before presentation. Clinical examination, biceps tendon test and shoulder instability tests under general anaesthesia were inconspicuous for shoulder instability. Routine radiographs, arthrography and ultrasound were suspicious for bicipital tendopathy. The definite diagnosis of a complete rupture of the biceps brachii tendon was made upon arthroscopic inspection of the gleno-humeral joint.

Keywords

Biceps brachii, tendon, rupture, diagnostic findings, dog

Vet Comp Orthop Traumatol 2007; 20: 73–77

Introduction

Bicipital tendon rupture is an acknowledged cause for shoulder lameness in the dog (1, 2). The pathogenesis is complex and the causes can be various. Specific disorders of the biceps tendon and its tendon sheath have been described in veterinary literature and include calcifying biceps tendinopathy (3), chronic bicipital tenosynovitis (4-7), medial luxation of the biceps tendon (8), partial or complete rupture of the biceps tendon (2, 6), avulsion fracture at the tuberculum supra-glenoidale (9), scar tissue formation (2), trauma (10) and joint mice entrapment (6) in the biceps tendon sheath. In a study evaluating bicipital tendon disease in 23 dogs and one cat, the most common bicipital tendon disorders were found to be partial or complete tears in the area of its origin at the supraglenoid tubercle (11).

The clinical signs of biceps tendon rupture may vary with its aetiology, but normally a moderate to severe forelimb lameness is present (2, 12). The traditional clinical approach for shoulder lameness is an orthopaedic examination involving a proper anamnesis, a general physical evaluation, static and dynamic palpation of the shoulder joint, evaluation of range of motion, biceps tendon test (1, 12), as well as the performance of crano-caudal (13) and medial shoulder instability (14) tests. Furthermore, a full neurological examination should always accompany the orthopaedic examination to rule out other disease processes that might mimic shoulder lameness, such as an asymmetrical herniated disc causing root signature or nerve-root tumours (15).

An important clinical finding for bicipital disease is the provocation of pain during the biceps tendon test (1). This test is carried out by applying digital pressure on the biceps tendon in the intertubercular groove while flexing the shoulder and extending the elbow. The provocation of pain during this test accounts as positive in the Anglo-American literature. In German literature, however, the term positive biceps tendon test describes the complete extension of the elbow joint in the completely flexed shoulder due to the loss of tension function in the shoulder joint and flexion function in the elbow joint in a completely ruptured biceps brachii tendon. It has been accredited to be pathognomonic for non-avulsion, complete biceps tendon rupture (16-18).

Diagnostic imaging techniques that have been modified for the detection of tendopathies include radiography (19), arthrography (20-22), ultrasonography (23, 24) and arthroscopy (25-27). These imaging techniques can detect osteoarthritis, fluid and joint mice in the tendon sheath, mineralization and scar tissue formation within the tendon, tendinitis, and partial or complete tendon ruptures and are used to substantiate clinical findings.

Treatment of bicipital tendon rupture consisted traditionally in tenodesis as described by Vasseur (28) followed by a resting period. Tendory is can be tried in freshly ruptured biceps tendons (2). Recently, new treatment approaches have been advocated. Wall and Taylor (15) described an arthroscopically-guided tendon transection without tenodesis in cases of tenosynovitis and partial tears with good to excellent outcome. Cook has successfully performed several arthroscopically-guided tenodeses with good results (29). However, the outcome of both methods is based on subjective preliminary results and awaits further evaluation.
Case history

A three-year-old, male castrated active Labrador Retriever was presented at the Department of Orthopaedics and Medical Imaging of the Faculty of Veterinary Medicine at Ghent University for a second opinion regarding the clinical evaluation and treatment options for a unilateral left shoulder problem. The dog had a medical history of an assumptive complete shoulder luxation of unknown origin six months before. After this event the dog was acutely lame and lameness persisted for several months. The dog recovered slowly and gradually with rest and anti-inflammatory drugs. At the time of presentation, the dog had been free of lameness for four weeks without restriction of activity.

On clinical examination there was not any visible lameness, neither walking nor trotting. Palpation of the musculature was unobtrusive and no atrophy could be detected. The range of motion of the shoulder was within normal limits. Pain and hyperextension of the elbow in complete flexion of the shoulder joint could not be elicited. Only a slight discomfort at flexion and extension of both elbows could be noted.

Neurological examination, including palpation of the cervical spine and the plexus brachialis, was inconspicuous.

Prior to further diagnostic work-up the dog was sedated with acepromazine/methadone (0.01 mg/kg iv. Acepromazine; 0.1 mg/kg methadone iv.) and anaesthesia was induced with pentothal (10 mg/kg iv.) An endotracheal tube number 10 was placed and the dog was anaesthetized using an isoflurane/O₂ mixture.

The shoulder was then tested for instability according to Cook (Fig. 1) and Bardin (Fig. 2). A positive drawer sign could not be elicited and the angle of abduction was around 30° and therefore within reference range (32.6 ± 2.0: Cook) in both limbs.

Radiographs of both shoulders were taken in mediolateral and caudocranial projection. The radiograph of the right shoulder revealed signs of degenerative joint disease on the cranial aspect of the joint and a moderate osteophytosis (osteophyte formation in the range of 2 – 4 mm) at the caudal distal part of the glenoid. An irregular outline of the supraglenoid tubercle and new bone formation in the proximal part of the bicipital groove was also visible (Fig. 3).

Ultrasonographic examination of the right shoulder joint was performed in left lateral recumbency with the right shoulder joint in abduction and in flexion. The area of the shoulder joint was clipped and coupling gel was applied. A multifrequency linear array probe of 7–12 MHz was used* The examination showed an amorphous, inhomogeneous and hyperechoic structure 0.5 cm distal to the proximal insertion of the biceps brachii tendon. There was also presence of a mild degree of joint effusion with an anechoic appearance (Fig. 4). The proximal attachment of the tendon could not be visualized and moderate os-
Diagnostic findings in complete biceps tendon rupture

Osteophyte formation was present in the area of insertion. In the intertubercular groove the biceps brachii tendon was homogeneous and normal sized without presence of peritendinous fluid. Alterations in this area could not be found except for a small osteophyte at the medial aspect of the tuberculum majus (Fig. 5). The contralateral joint was scanned for comparison and did not show any abnormalities. The ultrasonographic findings were compatible with a right bicepital tendon rupture at the level of its proximal attachment.

Synovial fluid was aspirated from the right shoulder joint prior to the injection of 4 ml positive contrast agent\(^a\) diluted with equal parts of sterile Hartmann’s solution to establish a total iodine concentration of 150 mg/ml. The volume of the withdrawn synovial fluid was 2 ml. The synovial fluid appeared to be of normal colour and viscosity. The cell count was within normal range (<3000 cells/µl) (30).

A positive contrast arthrogram (Fig. 6) showed insufficient filling of the biceps brachii tendon sheath. Contrast pooling was prominent at the insertion of the biceps and a light distension of the joint capsule as well as an irregular outline of the caudal joint pouch was visualized.

Arthroscopy was performed via lateral approach. The tendon of the biceps brachii was found to be completely ruptured at the level of its insertion with the calcified and thickened remnants still visible (Fig. 7). There was a moderate synovitis and a slight general fibrillation of the cartilage. The osteophyte at the caudal rim of the glenoid cavity could not be visualized as an intra-articular structure and there were no erosions on the opposite site of the humeral head.

The dog was discharged on the same day. Due to the absence of lameness, exercise restrictions were not imposed. Feeding of supplementary glucosamines was advised. Ancillary anti-inflammatory treatment (Carprofen 2 mg/kg) for three weeks was prescribed to reduce the aggravation of synovitis.

Discussion

According to Bardet (11), rupture of the biceps brachii tendon in dogs may result

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\(a\) Omnipaque®, Amersham Health, Princeton, NJ, USA.
from transection within the tendon (direct injury), avulsion from bone at the origin (indirect injury) or from intrasubstance damage from intrinsic or extrinsic factors, which subsequently lead to failure. Avulsion fractures and muscle-tendon junction ruptures are the most common sites of tendon injury, whereas mid-tendon ruptures are rare due to their greater tensile strength and require the presence of a pre-existing pathological condition. Traumatic shoulder luxations have been described as a frequent cause for rupture of the biceps tendon (28, 31). In traumatic shoulder luxation, a sudden onset of non-weight bearing lameness is accompanied by intense pain, followed by chronic lameness (2). Treatment for traumatic shoulder luxation can be either surgical or medical, involving rest and immobilization with a Valpeau sling (12). In our case, the onset of the reported lameness had been sudden and persistent, which therefore makes an acute trauma to the shoulder a likely cause.

Bardet, who investigated and classified bicipital tendon lesions (11) found in his study a positive biceps test in every affected shoulder joint. He also stated that a total of 76% of these shoulders showed signs of instability. Kramer reported various degrees of lameness, swelling and pain in the shoulder joint as prominent clinical features for complete biceps tendon rupture (2). An important clinical characteristic is the loss of functionality of the biceps tendon as flexor of the elbow and extensor of the shoulder. A situation that will allow the clinical examiner to hyperextend the elbow in a fully flexed shoulder and which is accredited as a ‘pathognomonic’ sign for complete biceps tendon rupture (16-18). In our case, however, clinical examination of the shoulder joint of the presented dog was uneventful. Lameness was not visible upon standing and trotting. Static and dynamic palpation of the shoulder and its surrounding soft tissue structures as well as shoulder and abduction tests were inconspicuous at the time of the presentation. Flexion and extension of the joint were within normal range and did not elicit any pain reactions. The biceps tendon test was negative and the elbow could not be fully extended in full flexion of the shoulder joint. To our knowledge, absence of a positive biceps tendon test in the presence of a ruptured biceps tendon as well as the impossibility to hyperextend the elbow in full flexion of the shoulder has not been previously described.

Diagnostic imaging findings of the glenohumeral joint, on the other hand, were highly indicative for bicipital tendopathy. Radiography showed an irregular outline of supraglenoid tunnel, calcification of the proximal intertubercular groove in combination with arthrosis and moderate osteophyte formation at the caudal margin of the glenoid cavity. According to Lechleitner, the most prominent radiographic feature, indicative for bicipital tendon disease, is sclerosis formation in the intertubercular groove (19). This radiographic feature is in alignment with our findings. However, it must be stressed that radiography can only display the disintegration of soft tissue structures in form of secondary degenerative changes such as arthrosis, mineralization and irregularities of the bony insertion points (12). Such changes can also be caused by extrinsic/intrinsic conditions and are not specific for the sole pathology of bicipital disorders (32).

Ultrasonography of the glenohumeral joint showed an inhomogenous, hyperechoic structure at the insertion of the bicipital tendon, which correlated with features that Kramer found in ruptured, bicipital tendons (24). Arthrography revealed positive contrast pooling at the insertion of the biceps tendon and insufficient filling of its tendon sheath, which is highly indicative for biceps tendon pathology (12). The sensitivity for detecting discrete bicipital tendon lesions, however, remains low (21).

Arthroscopy instantly detected rupture of the biceps tendon at the level of its insertion at the supraglenoid tunnel and the presumed shoulder pathology in this case could be confirmed. Besides a slight fibrillation of the cartilage and a moderate synovitis, further pathological features could not be seen.

Although our diagnostic imaging findings of the examined shoulder were highly indicative of bicipital tendon disease and corresponded with various features described in the veterinary literature, none of our clinical findings matched the findings that Bardet (11) and Kramer (2) described for bicipital tendon lesions. Furthermore, the criterion for complete biceps tendon rupture, being the ‘pathognomonic’ sign of full elbow extension in a completely flexed shoulder, was not fulfilled. The absence of a positive biceps tendon test in the presence of complete rupture of the biceps tendon, as well as the inability to hyperextend the elbow in full flexion of the shoulder, might indicate the reattachment of the tendon within its sheath. This is a theory that has been favoured by Wall and Taylor (15).

This might lead one to speculate that not every pathological shoulder can be clinically detected unless there is accompanying lameness or an indicative medical history. This theory would be supported by the work of Mayrhofer and Lechleitner (19, 33) who radiographically evaluated the shoulders of 100 euthanized dogs and found pathological features in the intertubercular groove in 40 dogs that had not been clinically suspicious for shoulder disease previously. This also might explain the frustration in the work-up of some cases of obscure shoulder lameness/pathology and stresses the importance of arthroscopy for the diagnostic evaluation of shoulder lameness.

References

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