A radiographic survey of the glenohumeral joint in racing Greyhounds

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Introduction

Lameness associated with shoulder pain in the racing Greyhound has a reported incidence of around 20% (Prole 1976, Agnew 1992) with almost all cases associated with tears of the triceps muscle group. Diagnosis of muscle tears is by palpation. Injury to the biceps brachii tendon of origin is reported and rare (Boemo and Eaton-Wells 1995). Fractures of the caudal border of the scapula are occasionally seen. Idiopathic shoulder lameness also occurs in Greyhounds and further diagnostic investigations are rarely made due to financial constraints.

The aims of this study are to identify radiographic pathological changes in the area of the glenohumeral joint, record their incidence and to determine their significance.

Materials and methods

Two cohorts of Greyhounds were selected for the study, dogs with shoulder lameness and dogs that were anaesthetised for reasons other than shoulder lameness. The latter group consisted of racing Greyhounds and retired Greyhounds. Shoulder lameness was diagnosed by a repeatable pain response to forced shoulder flexion in dogs that had thoracic limb lameness or were presented for poor racing performance.

All the dogs had mediolateral radiographs taken of both shoulders in the extended position. Observed pathological changes were recorded together with the limb and the severity of change.

Results

One hundred Greyhounds were in the study of which 16 were classified as having shoulder pain and 84 not presented with shoulder pain. Of these 43 were racing and 41 retired racers.

Pathological changes were identified in nine of the lame dogs (56%) and 45 of the non-lame dogs (54%). These changes were fragmented caudal glenoid (FCG), mineralisation of the tendon of insertion of the supraspinatus muscle (SS), osteoarthritis (OA) and calcification of the origin of the acromion head of the deltoid muscle (CD). In addition single cases were seen of mineralisation of a bursa over the acromion and a small avulsion of the origin of the biceps brachii muscle. The distribution and incidence of the lesions are tabulated.

Discussion

FCG, SS and OA have all been reported as causes of shoulder lameness and as coincidental findings in pet dogs (Olivieri and others 2004, Lafuente and others 2009). One reference to CD was found (Blythe and others 1994).

Ages of the dogs were not recorded but the racers would be on average a younger group than the retired dogs. There was no statistical difference in the incidence of any of the types of lesion between the two groups even though the retired dogs would have competed in more races than the racing group.

There was no statistical difference in the representation of each limb containing a lesion. Over-representation of one limb implies that the injury occurs on the bend due to cyclical asymmetric loading.

FCG is considered to be due to a non-fusion of a secondary centre of ossification and lameness is reported to occur when the fragment, probed arthroscopically, is loose (Olivieri and others 2002, Olivieri and others 2004). The radiographic appearance varied from mild sclerotic changes to the glenoid rim with vague mineralisation of the adjacent labrum to an obvious separate fragment. Although occurring in 30% of dogs in varying degrees of severity, there was concomitant mild OA in only two of the cases. However some dogs had localised secondary osteophytic changes to the glenoid rim at the site of fragmentation.

Osteoarthritis was identified as a sclerotic line at the margin of the joint capsule on the humeral head and was only identified in six non-lame dogs. Five dogs had very mild changes. The assumption would be that due to its infrequency, chronic glenohumeral joint lameness is uncommon in racing Greyhounds.

The calcified masses in the origin of the deltoid muscle just distal to the acromion were up to one square centimetre in size. The fragment was removed from the one case in the lame cohort and the dog's performance did not improve. The trainer of a retired dog with CD reported no previous shoulder lameness. The aetiology may be similar to that of mineralisation of the flexor tendons of the elbow. An avulsion fracture is an unlikely cause as no deficit was seen in the outline of the acromion.

The mineralisation observed superimposed on the greater tubercle is assumed to be within the insertion of the supraspinatus muscle. There were three cases of what had the appearance of a very small avulsion fracture. Pathological changes to the tendon in pet dogs have been reported with and without mineralistion and have been associated with lameness (Lafuente and others 2009). Ultrasonography and MRI detected enlargement of the tendon, increased fluid content and displacement of the biceps tendon.

Other diagnostic modalities would be necessary to further determine the pathology of these lesions. In this survey no connection could be made with the cause of lameness and the observed pathological changes.

Conclusions

Pathological radiographic changes around the glenohumeral joint are common and appear to be unassociated with lameness. Chronic lameness associated with the glenohumeral joint is uncommon born out by the low incidence of OA in older dogs. Radiography is a poor modality in identifying the cause of shoulder lameness in racing Greyhounds.

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