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The role of osteonecrosis in canine coronoid dysplasia: Arthroscopic and histopathological findings

I.C. Mariee[a], A. Gröne [b], L.F.H. Theyse[a,*]

[a] Department of Clinical Sciences of Companion Animals, Faculty of Veterinary Medicine, Utrecht University, Yalelaan 108, 3584 CM, Utrecht, The Netherlands

[b] Department of Pathobiology, Faculty of Veterinary Medicine, Utrecht University, Yalelaan 1, 3584 CL, Utrecht, The Netherlands

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ABSTRACT

Coronoid dysplasia (CD) or medial coronoid disease is part of canine elbow dysplasia and eventually results in osteoarthrosis. Although CD was originally attributed to disturbed endochondral ossification, more recent data point to the subchondral bone. The objective of this study was to assess dysplastic bone and cartilage of dogs that underwent unilateral or bilateral arthroscopic subtotal coronoidectomy for the treatment of CD. Arthroscopic findings and histopathology of bone and cartilage removed from elbow joints with CD were compared.

The most common arthroscopic finding was fragmentation with softening of the subchondral bone of the central part of the medial coronoid process. In dogs without obvious fragmentation, CD was characterised by bone softening and chondromalacia. During arthroscopic intervention dysplastic bone and cartilage were collected for histopathological assessment. Forty-five slices of formalin-fixed, paraffin-embedded bone and cartilage samples were stained using haematoxylin and eosin and evaluated. Histopathological findings primarily consisted of osteonecrosis of subchondral bone with necrosis within the marrow spaces. Histopathological changes in the articular cartilage were characterised by fibrillation, chondrocyte clone formation, and focal cartilage necrosis. The pathology was found primarily in the subchondral bone and not in the articular cartilage. Vascular compromise may play a role in the pathogenesis of osteonecrosis in CD.

Introduction

Canine elbow dysplasia (CED) is the most common developmental disorder of the elbow joint and results in forelimb lameness in juvenile and adult medium and large breed dogs (LaFond et al., 2002). Elbow dysplasia (ED) is a syndrome which consists of several conditions, including (1) fragmentation of the medial coronoid process of the ulna also known as medial coronoid disease (MCD), (2) osteochondritis dissecans of the medial part of the humeral condyle, (3) ununited anconeal process, and (4) elbow joint incongruity. These conditions can occur as single traits or in combination. Each can cause irreversible elbow osteoarthrosis (OA) due to cartilage damage, medial joint instability, and chronic synovitis of the affected elbow joint. Symptoms of MCD may be subclinical initially but will eventually result in marked lameness (Kirberger and Fourie, 1998; Samoy et al., 2012b). OA is progressive in the long term (Theyse et al., 2000).

CED is a polygenic trait and both environmental and hereditary influences play a role in its development (Kirberger and Fourie, 1998; LaFond et al., 2002; Temwichitr et al., 2010). The aetiology of MCD is currently unknown. Several hypotheses regarding the pathogenesis of MCD have been formulated, most of which involve abnormal endochondral ossification or abnormal mechanical forces arising from incongruity of the radius, ulna and humeral condyle (Trostel, 2003; Temwichitr et al., 2010). Recent studies have shown abnormalities in the bone structure and density of the coronoid (Fitzpatrick et al., 2009; Burton et al., 2010).

Coronoid dysplasia (CD) usually presents as fragmentation of a central part of the medial coronoid process of the ulna with bone softening and chondromalacia as the most common arthroscopic findings. Fragmentation of the coronoid can result in a fragment in situ or complete fragmentation with a displaced fragment (Moores et al., 2008; Fitzpatrick et al., 2009). In view of this we will use the term 'coronoid dysplasia' to address the abnormalities in the coronoid process.

CD is presumed to start in immature animals and the first clinical signs of lameness due to CD may appear at 4–6 months of age (Lau et al., 2013a, b, and c). The dogs usually have a history of forelimb lameness. However, CD is also seen in middle-aged and older dogs without any symptoms early in life (Vermote et al., 2010). The diagnosis CD is based on clinical signs including joint distension with pain during flexion, pronation, and supination of the effected joint.

* Corresponding author. Tel.: +31 302539411.
E-mail address: l.f.h.theyse@uu.nl (L.F.H. Theyse).

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In severe cases, secondary signs of OA can be present. The radiographic evaluation of CD typically follows the guidelines of the International Elbow Working Group (IEWG)[1] (Voorhout and Hazewinkel, 1987; Berry, 1992). Computed tomographic (CT) evaluation enables for a more sensitive visualisation of the coronoid process including transverse slices and multi-planar reconstructed images (Moores et al., 2008; Samoy et al., 2012a).

Bony abnormalities associated with CD include (1) structural changes, (2) sclerosis, (3) osteophytosis, (4) fissure formation, and (5) fragmentation (Reichle et al., 2000). Arthroscopy is considered to be the 'gold standard' as it is the most valuable diagnostic and therapeutic tool for ED, due to direct visualisation and assessment of the articular cartilage and subchondral bone (Van Ryssen and Van Bree, 1997; Fitzpatrick et al., 2009; Samoy et al., 2012b). In general, CD is treated by removing fragments, dysplastic bone and cartilage using curettage and partial ostectomy (Sams, 2000; Theyse et al., 2000). A dual approach of CT assessment and arthroscopy of the elbow joint provides the most accurate evaluation of pathological changes in the coronoid process (Burton et al., 2010). The aim of the present prospective study was to evaluate the arthroscopic changes in elbows affected by CD and to assess the dysplastic bone and cartilage removed during arthroscopy using histopathology. We hypothesised that CD is primarily located in the bone and not in the cartilage.

Materials and methods

Dogs

Thirty-eight dogs with CD, referred to the Department of Clinical Sciences of Companion Animals, University of Utrecht, between October 2010 and October 2012, were included in the study. All dogs were presented with front limb lameness. The diagnosis of CD was confirmed based on clinical evaluation in combination with CT imaging in 27 dogs and radiography in 11 dogs.

Diagnostic imaging

In 11 dogs, the diagnosis CD was based on plain radiography in accordance with the guidelines of the IEWG. In 27 dogs, an additional CT of both elbows was undertaken to confirm CD and to confirm or rule out bilateral involvement.

CT scans (Philips Secura single slice-helical CT scanner) were performed under general anaesthesia with the dogs positioned in dorsal recumbency on the CT table with the antebrachia parallel to each other and the forelimbs extended cranially. Perpendicular to the antebrachia, transverse views were made in 1 mm thick slices with 120 kV, 120 mA using a bone algorithm with a 1 s scanning time. Transverse slices were used to create sagittal and dorsal reconstructions. Every elbow was examined on transverse slices and on sagittal and dorsal reconstructed images. The evaluation criteria included the following signs: (1) fragmentation without displacement, (2) fragmentation with displacement, and (3) structural changes of the coronoid without obvious fragmentation.

Arthroscopic assessment

The medial coronoid process (MCP) was evaluated by arthroscopy using a 1.9 mm 30° angle forward arthroscope (Storz) in 45 elbows of 38 dogs. Thirty-one dogs underwent unilateral and seven dogs bilateral subtotal coronoidectomy, giving a total of 45 elbows examined arthroscopically (performed by LT). The dogs were placed in lateral recumbency with the affected limb positioned on the edge of the operating table and standard medial portals were used (Van Ryssen et al., 1993; Sams, 2000). During arthroscopy the articular cartilage of the medial coronoid process and the medial side of the humeral condyle were examined and probed thoroughly. In addition, the synovial membrane was evaluated for signs of acute and chronic inflammation and lesions were recorded. The evaluation criteria included the following signs: (1) displaced osteochondral fragment, (2) non-displaced osteochondral fragment, and (3) osteomalacia and chondromalacia without obvious fragmentation with softening of the subchondral bone and cartilage on palpation with a probe. Dysplastic bone and cartilage were removed by curettage and mini-ostectomy, and joint lavage was performed before closure (Sams, 2000; Theyse et al., 2000).

[1] See: http://www.vet-iewg.org/ (Accessed 6 July 2013).

Sample collection, processing and histopathology

Forty-five osteochondral samples were obtained from 38 dogs that underwent unilateral or bilateral subtotal coronoidectomy by arthroscopy as a treatment for CD. During arthroscopic assessment osteochondral samples (at least one sample per affected joint) were obtained during subtotal coronoidectomy from each elbow joint. Samples were fixed and conserved in 10% neutral buffered formalin until processing.

Samples containing both articular cartilage and subchondral bone were selected for evaluation. As these samples were collected during arthroscopy in a patient population standardisation of the specimens was limited. Subsequently, the samples were placed into a 10% ethylenediaminetetraacetic acid (EDTA) decalcifying solution at pH 7. The MCP samples were decalcified for at least 19 h and kept at room temperature on a shaker table. To prevent over-decalcification, a quick decalcification endpoint test was used including six samples of dogs of different ages at the time the osteochondral samples were collected (Verdenius and Alma, 1958; Mawhinney et al., 1984). This test was used to determine the endpoint by carefully weighing the specimen each day with a milligram balance until decalcification was completed.

All samples were embedded in paraffin wax and sectioned at 4 μm thicknesses before being stained with haematoxylin and eosin (HE) (Heidenhain, 1896; Bancroft and Stevens, 1982). The direction of sectioning was perpendicular to the articular joint cartilage. The sectioned osteochondral samples were evaluated microscopically (Olympus BX 40), and digital images (Olympus CAMEDIA) were obtained. The microscopic slides were subsequently evaluated independently by two of the authors (IM and AG) at different magnifications (100× and 400×).

Results

Dogs

The study population included 12 Labrador retrievers, 8 cross breeds, 4 Bernese mountain dogs, 2 Rhodesian ridgebacks, 2 Staffordshire bullterriers, and 1 each of the following breeds: German shepherd, Rottweiler, Picardian shepherd, Wetterhoun, Flat-coated retriever, Golden retriever, Hungarian Viszla, Old English bulldog, Boxer, and Australian cattle dog. The breed and sex distribution are presented in Table 1. Overall, the male to female ratio was 2:1. Eight of the male dogs and 13 of the female dogs were neutered. The mean age of the affected dogs was 25 months (range, 5–89 months; median, 1 year 1 month and 20 days) with a mean bodyweight of 34.5 kg (range, 10.6–49.8 kg; median, 34.9 kg).

Diagnostic imaging

CT scans of 27 dogs including both elbow joints were available. In 44 joints, the medial coronoid process showed obvious pathology The most common CT finding was fragmentation of the MCP which was present in 25 (56.8 .%) joints. Displaced fragments were diagnosed in 18 (72.0%) and non-displaced fragments in 7 (28.0%) elbow joints. On the CT images, fragmentation of the MCP was diagnosed when a separation of the craniomedial part of the coronoid was present, while displacement was diagnosed if the fragment or fragments were clearly dislocated in a cranial direction. In non-displaced cases the fragment remained wedged in place between the radius and intact part of the coronoid. Structural changes of the coronoid without clear fragmentation on CT imaging were present in 19 (43.2%) elbow joints. Osteochondrosis-like lesions were found

Table 1
Breed and sex distribution of the dogs with coronoid dysplasia.

Breed	Male	Female	Gender ratio (Male : Female)	Number of dogs (%)
Labrador retriever	9	3	3:1	12 (31.6)
Cross-breed	5	3	2:1	8 (21.0)
Bernese mountain dog	3	1	3:1	4 (10.5)
Rhodesian ridgeback	1	1	1:1	2 (5.3)
Staffordshire bull terrier	2	0	2:0	2 (5.3)
Other breeds	5	5	1:1	10 (26.3)
Total	25	13	2:1	38 (100)