Osteochondritis dissecans of the humeral head in two small-breed dogs
Osteochondritis dissecans of the humeral head in two small-breed dogs

M. Bruggeman, D. Van Vynckt, B. Van Ryssen, G. Bolln, K. Chiers, I. Gielen, H. de Rooster

Shoulder pathology consistent with osteochondritis dissecans of the humeral head was diagnosed in two small-breed dogs. In both cases, the diagnosis was made tentatively on the basis of radiography and confirmed by arthroscopy of the affected shoulder joint. Both dogs were successfully treated by surgical removal of a flap of cartilage from the affected area. Clinical and radiographic follow-up was available in both cases. The excised cartilage flaps were examined histopathologically and showed the same characteristics as cartilage flaps from dogs of large breeds prone to developing osteochondritis dissecans of the shoulder.

Osteochondrosis is a common joint disorder that has been recorded in various species, most commonly human beings, pigs, horses and dogs. It is a disorder of epiphyseal cartilage that manifests in both the articular-epiphyseal cartilage complex and the growth plates (Hill and others 1964). In osteochondrosis, there is a disturbance of endochondral ossification in which the cartilage of the epiphysis is retained rather than converted to subchondral bone (Craig and Riser 1965). This results in thickened, abnormal cartilage that is prone to injury from mechanical stress. Fissures develop and progress, in the more chronic stages of disease, to separation of a segment of cartilage extending from the articular surface to the subchondral bone; this condition is termed osteochondritis dissecans (OCD) (Olsson 1981). Partial separation of the cartilage leads to a hinged cartilage flap, while complete separation progresses into a joint mouse.

OCD of the humeral head is frequently identified as a cause of forelimb lameness in dogs. It is observed at the age of four to 10 months in rapidly growing dogs of large and giant breeds (generally, the breeds with individuals weighting more than 20 kg). However, certain large breeds, including the doberman, rough collie and Siberian husky, are at low risk of developing OCD of the humeral head (Rudd and others 1990).

This paper describes the presence of unilateral lesions of OCD in the shoulder joint in two small-breed dogs. In both cases the clinical signs and radiographic, arthroscopic and histological features were similar to those of classical cases of shoulder OCD in large- and giant-breed dogs.

Case report

Case 1

An 8.5-month-old male English cocker spaniel was presented because of lameness in the left thoracic limb. The dog had a history of acute-onset lameness after a jump four months previously. At that time, the dog had been treated with 0.2 mg/kg meloxicam (Metacam, Boehringer Ingelheim), administered subcutaneously, followed by 0.1 mg/kg meloxicam, administered orally once a day for three weeks. The dog’s initial response was favourable. However, it was presented for re-evaluation because of persistent left thoracic limb lameness.

On physical examination, a pain response was elicited during extension of the left shoulder. There was marked atrophy of the shoulder muscles on the affected side. Bilateral shoulder and elbow radiographs were taken. Both elbows and the right shoulder joint were radiographically normal. On a lateral radiograph of the left shoulder joint, there appeared to be a thin radioluent area on the caudal aspect of the humeral head (Fig 1). In the caudal aspect of the humeral head, the radiographic signs included a slightly irregular contour of the humeral articular surface as well as subchondral lucency surrounded by sclerosis. No osteoarthritic changes were visible.

Anaesthesia was induced with propofol (Narcofol, CP Pharma) at 4 to 6 mg/kg intravenously and was maintained with 4 per cent sevoflurane (Sevorane, Abbott Pharma) and oxygen. Arthroscopic examination of the left shoulder joint was performed to assist in establishing a definitive diagnosis. The dog was positioned in right lateral recumbency, with the limb to be operated on placed uppermost. The joint was punctured with a 19 G needle placed cranially between the acromion and the greater tubercle, and irrigation fluid was injected to distend the joint. A 2.4 mm arthroscope was inserted via a standard lateral approach to the shoulder (Van Ryssen and others 1993). Hardly any inflammatory change was visible within the affected shoulder joint, and the bicipital tendon also appeared normal. On the caudal aspect of the humeral head, a fissure line was visible in the cartilage. Although still in its anatomical position, a flap of cartilage appeared to be completely detached and could easily be lifted up from the subchondral bone (Fig 2). No other fragments were found. The flap was removed in small pieces by using graspers. The superficial bone within the defect had a straw-coloured appearance, with a pink curved line at the contour of the lesion. Superficial abrasion of the underlying bone was performed with a hand burr until the bone bled freely. The joint was copiously flushed under pressure before the arthroscope portals and the needle were removed.

The cartilage flap was submitted for histological examination, which revealed random proliferation of chondrocytes with the formation of small clusters (chondrones), fissures, and bleeding.

The dog was discharged from the hospital on the same day, and was treated with 0.2 mg/kg meloxicam, given orally once a day for
A six-month-old male pug was admitted with a two-month history of lameness of the right thoracic limb, without a history of known trauma. The lameness was severe and had worsened progressively; administration of meloxicam had no effect. The dog's gait was evaluated subjectively and the lameness was graded 4/5. On clinical examination, there was mild atrophy of the right shoulder muscles and a discrete pain reaction when the shoulder was hyperextended. The dog was sedated with acepromazine (Placivet; Kela) at 0.001 mg/kg intravenously and methadone (Mephenon; Denolin) at 0.1 mg/kg intravenously and was maintained with end-tidal isoflurane (100 mg iodine/ml) (Omnique; GE Healthcare) was injected into the caudomedial part of the humeral head (Fig 3). The caudal aspect of the glenoid fossa appeared rounded. No obvious signs of osteoarthritis were seen.

Case 2

A six-month-old male pug was admitted with a two-month history of lameness of the right thoracic limb, without a history of known trauma. The lameness was severe and had worsened progressively; administration of meloxicam had no effect. The dog's gait was evaluated subjectively and the lameness was graded 4/5. On clinical examination, there was mild atrophy of the right shoulder muscles and a discrete pain reaction when the shoulder was hyperextended. The dog was sedated with acepromazine (Placivet; Kela) at 0.001 mg/kg intravenously and methadone (Mephenon; Denolin) at 0.1 mg/kg intravenously. Standard orthogonal radiographs were taken of both shoulder joints. In the caudal area of the right humeral head, a radiolucent line surrounded by sclerosis was visible, suggesting fragmentation of the humeral head (Fig 3). The caudal aspect of the glenoid fossa appeared sclerotic. The proximal humeral growth plate appeared to be partially closed. Following the plain radiographs, a contrast study of the right shoulder joint was performed, 2 ml of diluted iohexol contrast medium (100 mg iodine/ml) (Omnique, GE Healthcare) was injected into the cranial aspect of the shoulder joint. The contrast radiograph showed a large amount of contrast medium under the cartilage. Anaesthesia was induced with propofol (Propovet; Abbott) at 4 to 6 mg/kg intravenously to effect, and was maintained with end-tidal isoflurane (Isoflo; Abbott) on 1.5 times MAC and oxygen. Arthroscopy was performed with a 1.9 mm arthroscope via a standard lateral approach; 1 ml of yellow synovial fluid was retrieved from the joint. On arthroscopic inspection, the synovial membrane appeared to be severely inflamed. The biceps tendon was intact. A large, partially detached flap was visible at the caudomedial part of the humeral head. The flap extended far medially and was difficult to reach with the surgical instruments (Fig 4). The flap consisted of cartilage and a thin layer of soft, yellow bone. The flap was removed completely using 2 mm and 2.7 mm grasps via a 2.5 mm instrument cannula. The underlying subchondral bed was curetted. At the end of the procedure, 1 ml of 2 per cent bupivacaine (Marcaine, AstraZeneca) was injected into the joint. The osseocartilaginous flap was submitted for histopathology. Histologically, the flap consisted of mineralised and non-mineralised cartilaginous tissue containing a tidemark, with no reactive changes.

The dog was discharged on the same day after surgery and given 2 mg/kg carprofen (Rimadyl; Pfizer) orally, twice a day, for three weeks; for the owners were advised to restrict its exercise to walks on a lead for six weeks. During the first few days after surgery, the dog showed signs of pain, although it was weight-bearing on the affected limb. The limping gradually resolved, and the dog became more active and playful. At re-examination six weeks after surgery, moderate lameness and mild atrophy of the right shoulder muscles were still present. There was a normal range of motion and no pain reaction was elicited on passive motion. At three months postoperatively, the dog had improved further, although mild lameness and discrete muscle atrophy were still present. At the authors' request, the dog was presented for another examination five months postoperatively. The dog was completely sound. Plain radiographs of the right shoulder joint were obtained and revealed moderate radiographic signs of osteoarthritis and that the caudal area of the humeral head had a deformed, flattened and sclerotic appearance (Fig 5).

Discussion

Cases of shoulder OCD have occasionally been reported in small-breed dogs (Dingwall and others 1972, Johnson and Dennis 1973, Rudd and others 1990). The smallest dog reported previously was a miniature poodle (Dingwall and others 1972). There is also one published report of a case of OCD in a domestic cat (Peterson 1984). The only breed of dog with a bodyweight less than 20 kg frequently reported to be clinically affected with OCD is the Border collie (Knecht and others 1977, Olivieri and others 2007). It was postulated that the conformational and behavioural characteristics of this breed, such as its crouched-down herding position, fast turns and speed changes, may stress the area of the joint at risk of developing OCD (Knecht and others 1977). Among dogs presented to the authors' institution, Border collies have the second highest incidence of shoulder OCD (27 of 224 dogs); this high incidence cannot be explained by, for example, a high popularity of the breed among the dogs that make up the hospital population.

The joint most likely to be affected by OCD varies between species. After the elbow, the shoulder joint is most commonly affected in dogs (Olsson 1987). In human beings, OCD in the shoulder is rare; it is more common in joints such as the knee, elbow and talar dome (Mahirogullari and others 2008). In both of the cases described in this paper, the affected site was identical to the predilection area of OCD in the shoulder joint of commonly affected dog breeds.

Subchondral fracture is a differential diagnosis for OCD (Ytrehus and others 2007). In cases of subchondral fracture, a history of significant trauma would be expected, and the animal would be affected unilaterally. Trauma that would be insufficient to cause disease in a normal joint might predispose to a vertical fracture of the articular cartilage in cases of OCD (Johnston 1998, Ytrehus and others 2007). In approximately 80 to 85 per cent of the cases the joints on both sides are affected (as determined radiographically), although clinical signs of pain and lameness may be apparent on only one side (Johnston 1998, Tacke and others 1999, Vandevelde and others 2006). In the first case reported here, the cartilage flap might have resulted from trauma (potentially minor), but a traumatic aetiology was considered highly unlikely in case 2. In the second case, the humeral head growth plate appeared more closed in its caudal aspect, a finding commonly observed in OCD cases.

Visualisation of the lesions might have been improved by taking additional radiographic views, for example, a mediolateral view with pronation of the distal thoracic limb. CT and/or MRI could also have been considered, as both dogs were of breeds in which OCD would not be expected, but it is unlikely that MRI would aid in differenti-
Moderate (arrowheads). The unusual appearance of the caudal humeral head of case 2 was probably the result of a combination of the healing process and the asymmetric closure of the proximal humeral growth plate.

The cases described here emphasise that OCD should be considered in the differential diagnosis of shoulder lameness in any young dog, regardless of its size or breed.

References


FIG 3: Standard lateral radiograph view of the right shoulder joint of a six-month-old pug (case 2) at presentation. In the caudal area of the humeral head, a radiolucent line (black arrowheads) surrounded by sclerosis (black arrow), suggesting fragmentation of the humeral head, is visible. The caudal aspect of the glenoid appears sclerotic (white arrow). The growth plate (white arrowhead) appears partially closed.