Osteochondroma and multiple cartilagenous exostosis involving the distal radius and ulna on adjacent cortices – an unusual manifestation of a common condition

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SUMMARY

The findings of two canine patients presenting with distal antebrachial multiple cartilaginous exostoses (MCE) highlights the difference in presentation and clinical significance of MCE impinging on the adjacent radius and ulna. We suspect that a lesion in this region, affecting the adjacent bones, may be missed as a cause of lameness. This may be due to a lack of knowledge of the normal radiological anatomy of this region on a mediolateral view, and because of the close anatomical association between the distal ulna and radius and thus superimposition on radiographs. Additionally, skeletal immaturity may mask the exostosis as the thicker cartilage cap is not visible radiologically. Computed tomography (CT) allows a much better understanding of the pathology involved due to its cross sectional imaging.

BACKGROUND

MCE are well documented in the dog, but no reports exist documenting their presence as a cause of lameness in the distal antebrachium. The radiological lesions may be missed if the veterinarian is not aware of and specifically looking for this condition on radiographs, and because of a lack of knowledge of the normal radiographic anatomy of this region.

CASE PRESENTATION

Case 1: A 20-month-old, 24.5 kg castrated male Pitbull presented with a month's history of right thoracic limb lameness, especially after rising.

Case 2: An 18-month-old, 63 kg intact male Great Dane presented with mild right radiocarpal swelling after sustaining bite wounds a few days earlier. Initially the patient responded to antibiotics and non-steroidal drugs, but had relapsed and was now mildly lame after the drugs were discontinued.

INVESTIGATIONS

Case 1: Mediolateral, dorsopalmar and rotated mediolateral radiographs (Fig 1a-c) of the right carpus and a mediolateral radiograph of the left carpus (Fig 1d) were taken under general anaesthesia with 0.1 mg/kg acepromazine (Neurotranq®, Virbac, South Africa) and 20 mg/kg thiopentone sodium (Bomathal™, Merial, South Africa). Initially no radiological abnormalities were detected, thus the radiographs were submitted to a specialist radiologist. A single, irregular, sharply pointed triangular 4 x 5 mm craniodistomedial ulnar metaphyseal exostoses was seen causing a corresponding smooth semicircular lucent defect in the caudodistolateral radial metaphysis.

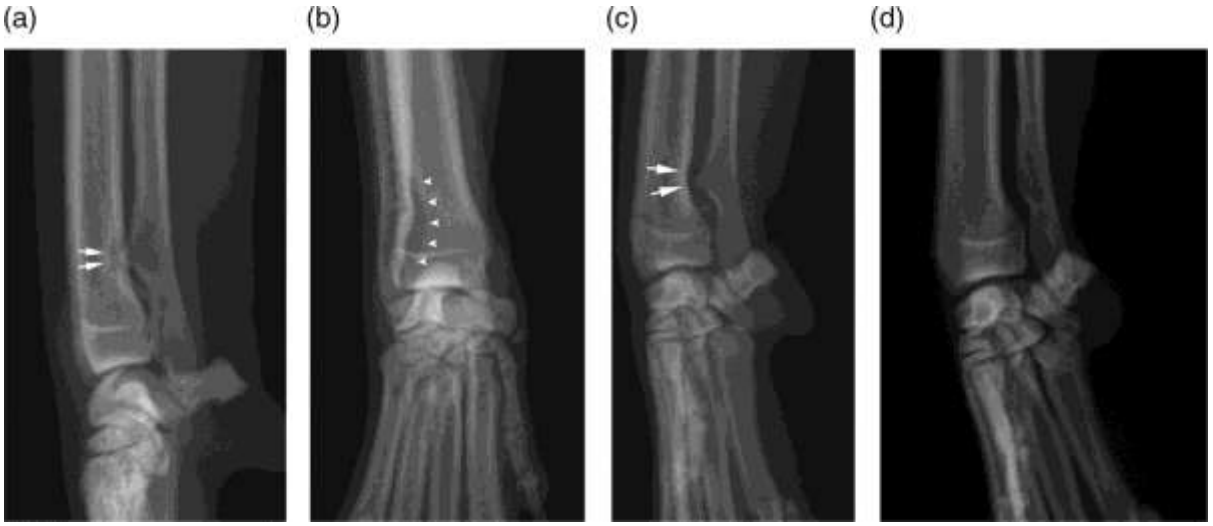

FIG 1: (a) Mediolateral, (b) dorsopalmar, (c) and rotated mediolateral radiographs of the right distal antebrachium and (d) mediolateral view of the unaffected left antebrachium, of case one. There is a distocranial ulnar exostoses causing a subtle distocaudal radial cortical defect and lucency (white arrows), appreciable only on the mediolateral views. The prominent radiolucency on the dorsopalmar view (white arrowheads) is caused by anatomically normal decreased soft tissue thickness laterally on the caudal aspect of the distal antebrachium. This should not be misinterpreted as bone lysis.

Case 2: Mediolateral and dorsopalmar radiographs (Fig 2a-b) of the right carpus were taken under sedation with 0.2 mk/kg morphine (Fresenius Kabi, South Africa) and 0.01 mk/kg medetomidine (Domitor®, Pfizer A.H., South Africa). Mild radiocarpal swelling was seen. On the distolateral ulna there was a 3 x 6 mm smooth triangular exostoses, with a similar but larger 16 x 6 mm mildly sclerotic exostoses on the craniomediodistal ulna impinging of the adjacent radius, which showed a focal smooth seimicircular radiolucent defect. A broad irregular sclerotic band was associated with and located cranial to this defect. Located just proximally on the cranial ulnar metaphysis, was a 10 x 5 mm semicircular lucent defect with a fine sclerotic rim. A radiological diagnosis of septic arthritis with incidental MCE was made.

Radiocarpal arthrocentesis confirmed the diagnosis of septic arthritis.

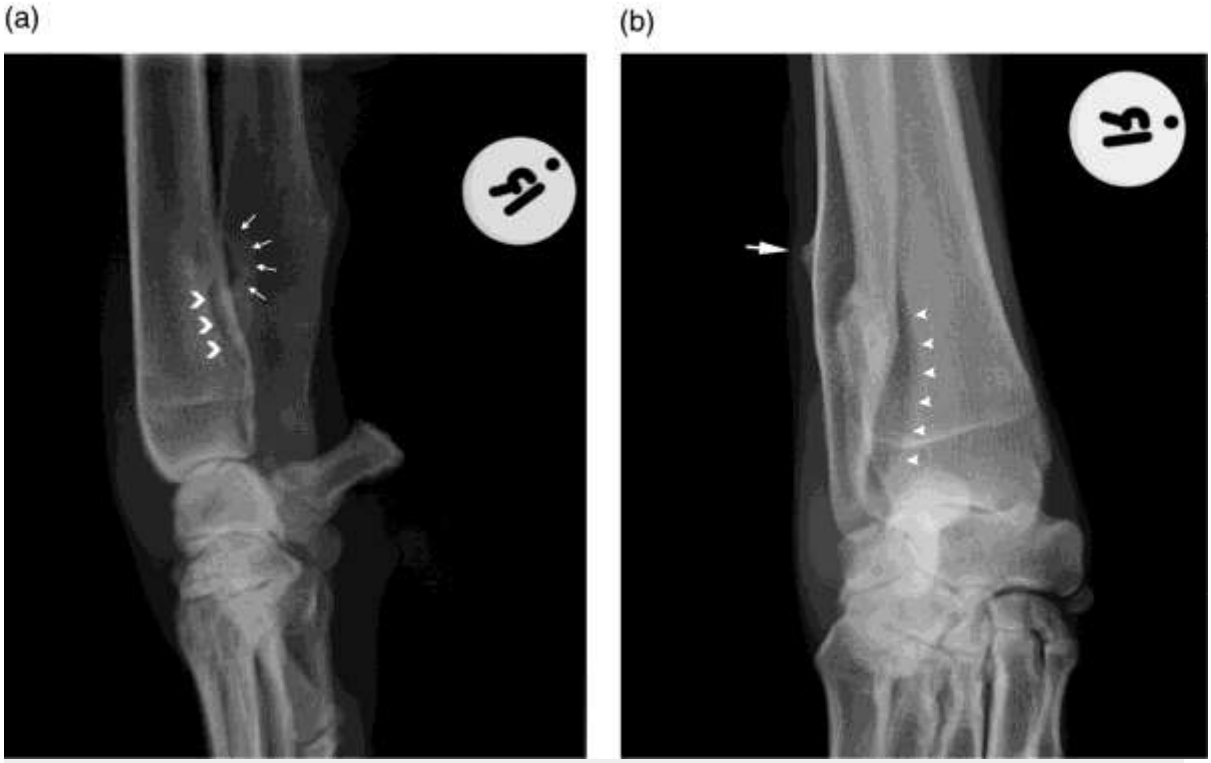

FIG 2: (a) Mediolateral, (b) dorsopalmar radiographs of the right distal antebrachium of case 2. A craniodistal ulnar lucency (small white arrows) and caudodistal radial lucency (white chevrons) with an opposing ulnar exostoses is visible. The above described exostoses are not visible on the dorsopalmar view, but a smaller clinically insignificant caudolateral ulnar exostoses (white arrow), which was a bilateral finding on CT, is seen on this view. The normal distal lateral ulnar antebrachial lucency is also seen (white arrowheads)

TREATMENT

Case 1: Surgery was performed via a lateral approach at the distal ulna, with the same anaesthetic protocol as described (Fig 3a-b). Pre-operative pain control included 0.4mg/kg morphine (Fresenius Kabi, South Africa) and 0.2mg/kg meloxicam (Mobic®, Boehringer-Ingelheim, South Africa). The exostosis was excised using an oscillating saw with a fine blade, and the remaining bone smoothed with a rasp. Wound closure was routine. The patient was discharged on 20 mg/kg cephalexin bid (Ranceph®, Ranbaxy, South Africa) and 0.1mg/kg meloxicam oid (Petcam®, Ciplavet Pty. Ltd. South Africa). The excised bone was submitted for histopathology which confirmed the diagnosis of MCE.

Case 2: The joint was flushed and treatment instituted with amoxicillin-clavulonic acid at 20mg/kg (Ranclav 625®, Ranbaxy (SA) Pty. Ltd, South Africa) and 4.4 mk/kg carprofen (Rimadyl®, Pfizer A.H, South Africa). A repeat joint flush was performed one week later, with sedation as previously described, at which stage the dog was free of pain and lameness.

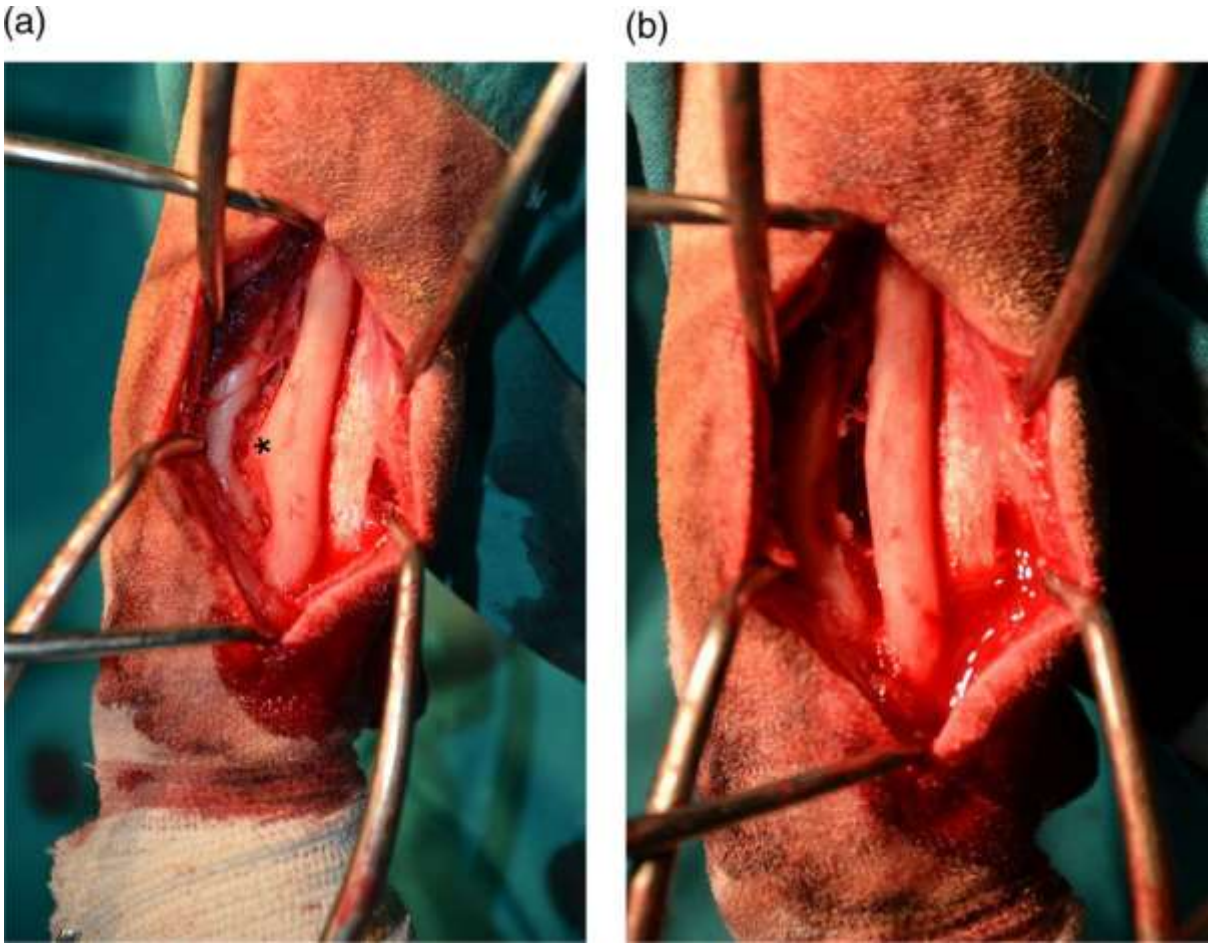

FIG 3: Intraoperative images of the ulnar exostoses in case 1, immediately prior to removal (a) and after removal (b) of the ulnar exostosis. Cranial is to the left of the image, and the antebrachiocarpal joint is located ventrally on the image. The degree of impingement of the adjacent radius by the ulnar exostoses (black asterix) is clearly demonstrated in (a)

OUTCOME AND FOLLOW-UP

Case 1: Follow up radiographs taken 8 months later showed subtle filling of the radial defect with mature bone (Fig 4). There was no evidence of regrowth of the exostoses and the ulnar surgical site had a 1 mm high, mildly undulating bony margin. The dog was not lame.

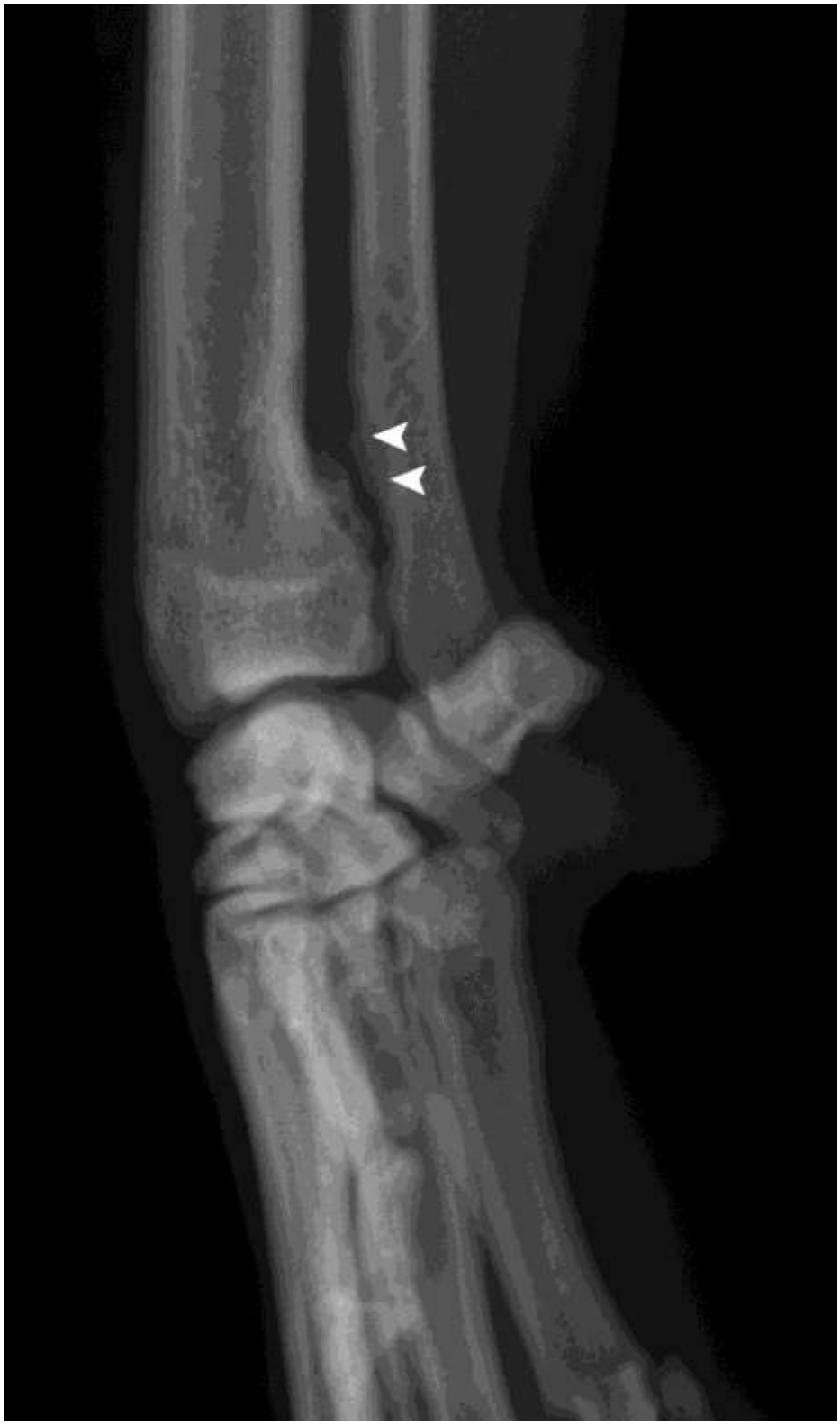

FIG 4: (a) Mediolateral, (b) Dorsopalmar radiographs of the right distal antebrachium of case two. A craniodistal ulnar lucency (small white arrows) and caudodistal radial lucency (white chevrons) with an opposing ulnar exostoses is visible. The above described exostoses are not visible on the dorsopalmar view but a smaller clinically insignificant caudolateral ulnar exostoses (white arrow), which was a bilateral finding on CT, is seen on this view. The normal distal lateral ulnar antebrachial lucency is also seen (white arrowheads)

Case 2: Helical CT (Siemens Emotion Duo, Siemens Medical Systems, Forchheim, Germany) was performed 3 weeks later under sedation with 0.01 mg/kg medetomidine (Domitor®, Pfizer A.H., South Africa). Three millimetre slices of both distal antebrachia were acquired in a bone window (window width 1400 and window level 300), and using multiplanar reconstruction, 2 mm slices were reconstructed over the lesions (Fig 5 a-e). A caudolaterally projecting 9 x 5 x 9 mm pedunculated exostosis was present on the right distolateral radius, causing a deep semicircular 10 x 7 mm cortical defect in the adjacent ulna, with an irregular thin sclerotic border (Fig 5a, c and e). There was an additional 4 mm diameter defect within the distal radial exostoses with a 5 x 6 mm opposing irregular distocraniomedial ulnar exostosis impinging on it (Fig 5a, c and e). Unrelated bilateral caudolateral mid-ulnar exostoses of 5 x 18 mm were also present (Fig 5a).

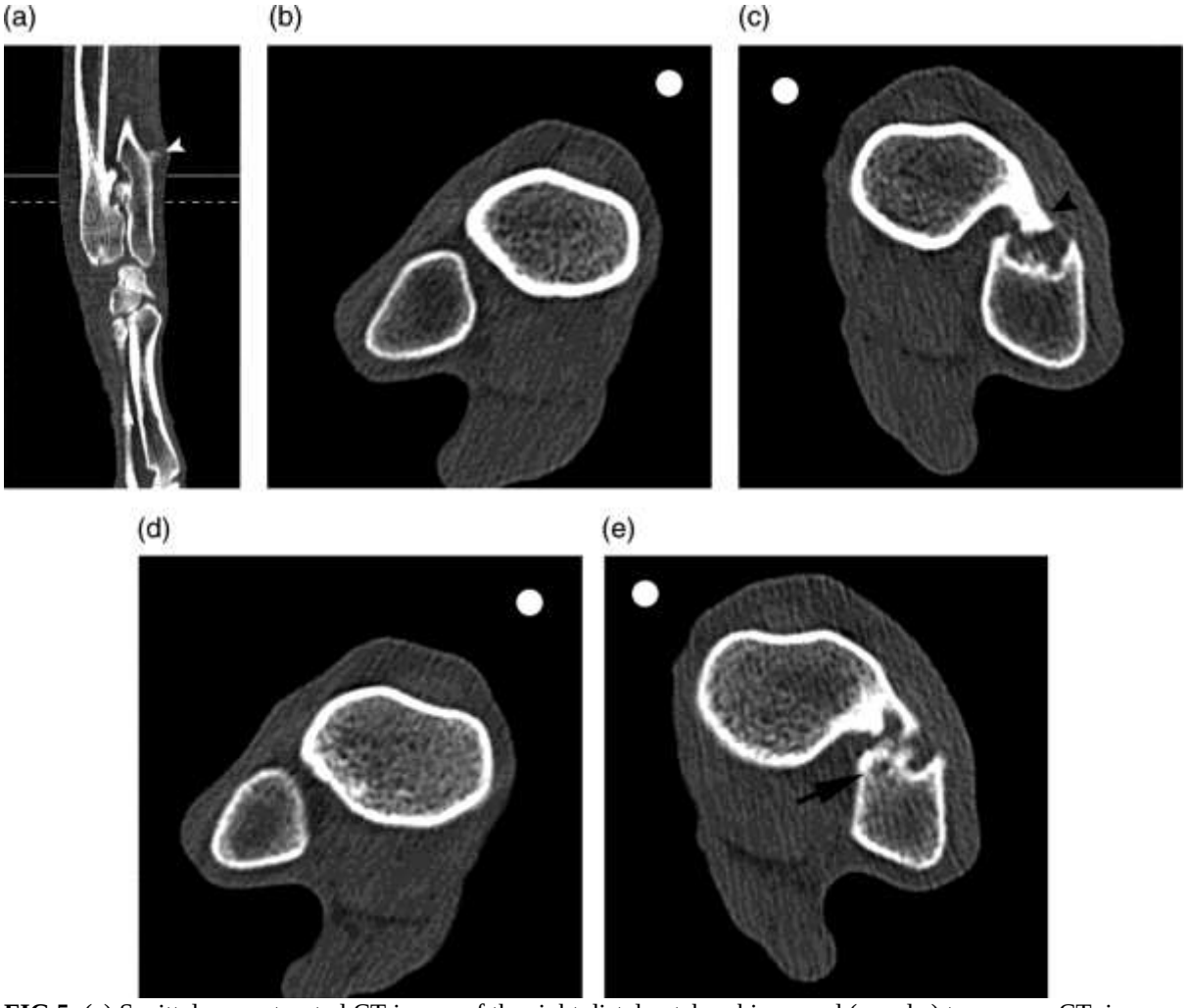

FIG 5: (a) Sagittal reconstructed CT image of the right distal antebrachium, and (c and e) transverse CT images of the affected right and (b and d) normal left radius and ulna for comparison. Images (b) and (c) correspond to the level of the radius and ulna demonstrated by the solid line on the sagittal image, and (d) and (e) correspond to more distal dotted line. The radius is located towards the top of the image; medial is indicated by the white circle. A prominent radial exostoses (black arrowhead) with associated ulnar defect is visible on the proximal slice, with an ulnar exostoses (black arrow) and radial defect located immediately distally. The edge of the incidental caudolateral ulnar exostoses is seen caudally in (a) (white arrowhead). Note the decreased bone and soft tissue dorsopalmar thickness laterally compared to medially which corresponds to the radiolucency seen on the distolateral antebrachium on the radiographs.

DISCUSSION

Introduction and literature review:

Multiple cartilaginous exostoses (MCE), or osteochondromatosis, is a benign proliferative disease of bone and cartilage that occurs in dogs, humans, cats and horses (Jacobson and Kirberger 1996; Pollard and Wisner 2013). It is a monogenetic, autosomal dominant disorder in man with two specific genes affected in 90% of patients (Ham 2013). It is also suspected to have a hereditary component in dogs (Pollard and Wisner 2013) but has not been proven. Historically, MCE in man has been referred to under many titles, which include diaphyseal aclasis, chondrodysplasia, hereditary deforming chondrodysplasia, osteogenic disease and hereditary multiple exostoses (Dingwall and others 1960). It is now generally accepted that a solitary lesion is referred to as an osteochondroma whilst if more than one lesion is present, it is referred to as MCE (Green and others 1999).

The condition develops in young growing dogs but is usually only diagnosed in older dogs, and is characterized by single or multiple hyaline cartilage-capped bony protuberances which arise from the surfaces of any bone that develops from endochondral ossification (Dingwall and others 1960; Jacobson and Kirberger 1996). Bones that form from intramembranous ossification, such as the skull, are not affected (Pollard and Wisner 2013; Dingwall and others 1960). Bones affected by MCE, in decreasing order of frequency, have been reported as the vertebrae, ribs, long bones, digits and pelvis (Caporn and Read 1993).

MCE generally occur on the metaphyses of bone (Silver and others 2001). Lesions may less commonly be found on the diaphyses, especially the tibia and femur (Finnie and Sinclair 1981) but the epiphyses are never involved (Jacobson and Kirberger 1996). In dogs, these exostoses usually stop growing at skeletal maturity, along with physeal closure. The resultant non-painful bony protruberances have no clinical significance in most cases, unless they occur in an anatomical region where they may compromise function (Pollard and Wisner 2013), such as within the vertebral canal causing spinal cord compression (Dingwall and others 1960; Caporn and Read 1993; Bhatti and others 2001; Green and others 1999). A solitary tracheal osteochondroma has been described that caused dyspnoea (Gourley and others 1970), with another case described occurring concurrently with vertebral MCE (Beck and others 2006). In man, clinical signs such as pain are often attributed to compression of tendons, muscles and nerves, bursitis and fracture of a pedunculated osteochondroma. Impingement by an osteochondroma may cause restricted joint motion, joint subluxation and neurovascular compression (Ham 2013). Cosmetic disfiguration caused by osteochondromas and MCE is especially important in man (Ham 2013).

The pathogenesis of MCE is uncertain in canines, and several theories have been proposed. It has been suggested that pieces of cartilage separate from the lateral margins of the physes (Dingwall and others 1960) and thus form a physis-like structure perpendicular to the bone shaft (Jacobson and Kirberger 1996). Additional theories are that there may be a defect in the periosteum, allowing its osteogenic layer to regain its perichondral, or chondrocyte and chondroblast-forming, potential and thus form excessive cartilage due to an unknown initiating factor (Pool 1993). Alternatively, the physis may direct its growth away from the normal direction, due to a defect or absence of the connective tissue "perichondrial ring" which may allow physeal herniation and aberrant lateral growth of cartilage perpendicular to the bone (Pool 1993). This last theory is supported in recent human literature (Vanhoenacker and others 2001). Despite sharing the same terminology and structural features (Pool 1993), tracheal osteochondromas may not share the same pathogenesis as the trachea does not undergo endochondral ossification (Pool 1993). The presence of tracheal osteochondromas may thus challenge the widely held endochondral ossification theory (Beck and others 2006). In man, solitary osteochondromas have been reported to occur post trauma or irradiation. One

canine case report exists that supports the findings of a post-traumatic osteochondroma (Koehler and others 2010).

Malignant transformation to chondrosarcoma or osteosarcoma is infrequently reported in mature dogs (Pollard and Wisner 2013; Finnie and Sinclair 1981). The incidence of malignant transformation in man has traditionally been estimated at 10-20% (Doige 1987), with recent literature estimating it to be only 3 – 6 % (Ham 2013) . The incidence of malignant transformation is suspected to be higher in dogs, based on a report in which three out of eight dogs with MCE developed malignancies (Doige 1987). This suspicion may be biased due to the largely asymptomatic nature of MCE, which thus leads to a large portion of undiagnosed cases. (Caporn and Read 1993). Solitary osteochondromas have rarely been reported to undergo malignant transformation (Green and others 1999; Pool 1993).

Dogs diagnosed with MCE generally fall into two age groups. Dogs diagnosed prior to skeletal maturity may present to a veterinarian because of disfiguring exostoses noted by the owner, or due to functional disturbances if the growths encroach upon vital anatomical structures. Older dogs may present due to malignant transformation of MCE, or MCE are incidentally found during radiography (Caporn and Read 1993).

Many atypical cases of MCE have been reported, including cases still growing after skeletal maturity. A two-year-old Great Dane developed MCE after reaching skeletal maturity, which continued to grow and cause some bridging of physeal regions (Jacobson and Kirberger 1996). A three-and-a-half-year-old Bernese Mountain dog had a lesion on the dorsal aspects of C1-C2 which was only diagnosed after skeletal maturity and was suspected to have progressed after maturity (Bhatti and others 2001). MCE may display atypical radiological features. A case has been described involving a four-month-old border collie that had unusual stippled tumours that were not contiguous with the adjacent bones, and had an appearance more consistent with tumoral calcinosis (Jacobson and Kirberger 1996). A recently published case report describes the concurrent finding of MCE and calcinosis circumscripta associated with the vertebrae of an immature St. Bernard (Engel and others 2013). All cases were confirmed by histopathology. Three mixed-breed dogs from the same litter developed symmetrical semi-annular and annular osteochondromas that were accompanied by limb shortening, limb deformation and angular deformity (Mozos and others 2002).

In cats, MCE has a slightly different aetiology and pathophysiology. A viral aetiology has been suggested, and feline leukaemia virus has been implicated (Pool and Carrig 1972). In contrast to dogs, MCE in the cat is a progressive condition with bony proliferation continuing after skeletal maturity. Contrary to dogs, bones of both intramembranous and endochondral ossification can be affected, including the skull (Mozos and others 2002). Clinical signs are associated with pain and loss of function, and malignant transformation to parosteal osteosarcoma may occur (Pollard and Wisner 2013; Mozos and others 2002). Recently, cavernous sinus syndrome due to intracranial osteochondroma was described in a cat (Perazzi and others 2013).

Radiologically, MCE are seen as variably sized outgrowths of dense cancellous bone that is continuous with the cortex of the parent bone (Jacobson and Kirberger 1996). A trabecular pattern is generally visualised within the exostoses on the extremities (Finnie and Sinclair 1981) and thus a distinct cortex and medulla may be visualised. There is no evidence of bony lysis or periosteal reaction, and thus they have a radiologically benign appearance (Green and others 1999).

Rib exostoses tend to be a more irregularly marginated and heterogeneous whilst long-bone and vertebral exostoses tend to display a more organized appearance (Pollard and Wisner 2013; Finnie and Sinclair 1981), with vertebral masses tending to a circular appearance with a sessile base, but long bones having a more varied sessile shape (Jacobson and Kirberger 1996). The affected bone may be deformed (Pollard and Wisner 2013) or adjacent closely

associated bones may be deformed secondary to compression by the bony mass (Finnie and Sinclair 1981). Lesions may appear mottled during active growth due to incompletely mineralised hyaline cartilage, and apparent enlargement of the lesion on subsequent radiographs may be due to ossification of the cartilage cap. Radiology may be useful to detect additional lesions and to monitor changes in size of the protruberences (Jacobson and Kirberger 1996; Ham 2013).

Macroscopically the exostoses appear as a white to bluish hyaline cartilage cap overlying a bony outgrowth during active growth, but if the lesion is mature the cartilage cap may be incomplete or absent (Jacobson and Kirberger 1996). In adult man, the cap is usually only 2-3 mm thick but may be thicker in adolescents, prior to complete ossification (Malghem and others 1992). Histologically, the cartilage cells resemble a physis with cartilage columns arranged perpendicularly to the underlying cortical bone (Dingwall and others 1960). The exostoses are covered by a membranous lining that is continuous with the normal periosteum (Jacobson and Kirberger 1996).

Magnetic resonance imaging (MRI) and computed tomography (CT) are occasionally utilised in animals to assess MCE particularly when spinal cord compression is suspected (Caporn and Read 1993; Engel and others 2013; Silver and others 2001). In man, MRI and CT have been used to assess the thickness or the cartilage cap, which correlates well with malignant transformation and development of a chondrosarcoma (Bernard and others 2010). On MRI, the cartilage cap, because of its high fluid content, is readily seen as a T2-weighted hyperintensity (Lee and others 2000). MRI is stated to be the gold standard to detect neoplastic transformation by using contrast studies (Kitsoulis and others 2008) and is superior to CT for demonstrating the cartilage cap, especially in cases where the cap is poorly mineralised or very small (Engel and others 2013). CT is only accurate for detection of cartilage caps, and to measure their size, when the caps are large. This is thought to be due to the similar attenuating characteristics of muscle and cartilage, partial volume averaging of the cartilage with adjacent bone with thicker slice thickness, and the orientation of the CT scan plane relative to the cartilage cap (Hudson and others 1984). Ultrasound is a good modality to evaluate cartilage cap thickness in accessible lesions and compares favourably to MRI findings. Ultrasonographically, the cartilage cap appears as a hypoechoic region overlying hyperechoic cortical bone (Malghem and others 1992).

Discussion:

The authors commonly see distal radius and ulna MCE in dogs as incidental findings but the exostoses occur laterally on the ulna and medially on the radius. Painless pressure deformation of the ulna due to an atypical mid-radius exostosis has been described (Finnie and Sinclair 1981). A painless case of distal radial MCE with ulnar lysis and deformation and another painful case involving phalanx one of digit four have been described in Sweden (Andersson 2009) but it was not specified whether the diagnoses were confirmed histopathologically.

In children, osteochondromas on the forearm can cause significant decreases in rotational motion, growth discrepancies between the radius and ulna and radial head subluxation. The distal forearm is affected in 80% of cases. (Ham 2013). In the paired bones, like the radius and ulna, MCE may cause pressure on the adjacent bone and even form a pseudoarticulation with it (Vanhoenacker and others 2001).

This communication highlights the difference in clinical presentation of MCE impinging on the adjacent radius and ulna in the distal antebrachium. In case one, confirmed surgically, the ulnar lesion impinged mainly on the adjacent radius, causing radial lysis. This lesion resulted in lameness. In case two, where lameness was not due to the MCE but due to carpal septic arthritis, the radiological appearance was that of a large radial exostoses with smaller radial

and ulnar lytic lesions. The close anatomical association between the distal ulna and radius and thus superimposition on radiographs may be confusing, with mediolateral views being most diagnostic. In contrast to the radiographs, CT allowed a much better understanding of the pathology involved due to its cross sectional imaging. CT has been shown to be an excellent modality for evaluating orthopaedic conditions due to its cross sectional images and ability to post process images into multiple planes (Ohlerth and Scharf 2007). As described in man (Hudson and others 1984), we were not able to detect any cartilage caps. Factors contributing to this include possible small size of the cartilage cap in mature osteochondromas, similar attenuation of cartilage and the adjacent soft tissue structures within the interosseus space, and partial volume averaging artefact.

Surgical treatment was successful in case one, with no regrowth of the exostoses, in line with expectations in a skeletally mature dog. In the second case, the exostoses were merely an incidental finding, and lameness was due to septic arthritis. It may be speculated that lameness is more likely to occur with ulnar exostoses causing pressure related lesions in the radius, which is the greater weight-bearing bone, but no specific literature exists to support this.

The authors are unaware of previous reports of limb lameness due distal antebrachial MCE. We suspect that a lesion in this region, affecting the adjacent bones may be missed as a cause of lameness. This may be due to a lack of normal radiological anatomy knowledge of this region on a mediolateral view. Additionally, skeletally immaturity may mask the exostosis as the thicker cartilage cap is not visible radiologically and the only indication of an osteochondroma may be a subtle radiolucent defect in the opposing bone. The latter must not be confused with the normal relative radiolucency seen distolaterally on the radius and ulna on craniocaudal views.

LEARNING POINTS/TAKE HOME MESSAGES

- Mediolateral radiographs are the most diagnostic for identifying radial or ulnar exostoses as a cause of lameness.
- Skeletal immaturity may mask the true extent of the exostoses due to the radiolucent appearance of the cartilage radiologically.
- CT findings may differ substantially from radiographic findings but may not be helpful to identify a cartilage cap.
- We suspect that an ulnar exostoses impinging on the radius is more likely to cause lameness that a radial exostoses impinging on the ulna, although there is no literature supporting this.
- Surgical treatment appears to be successful in cases of lameness caused by a single exostoses.

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