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Carpometacarpal Osteoarthritis in Thirty-Three Horses

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Objective—To describe signalment, clinical, and radiographic changes associated with carpometacarpal osteoarthritis (CMC-OA) and to report long-term outcome.
Study Design—Case series.
Animals—Horses (n = 33) with CMC-OA.
Methods—Medical records (1992–2007) of horses diagnosed with CMC-OA were reviewed and signalment, clinical, and radiographic findings retrieved. Owners were contacted for information on the impact of lameness on intended use, response to treatment, progression of lameness, outcome, and owner satisfaction with response to treatment.
Results—CMC-OA identified in 39 limbs, occurred predominantly in middle-aged and older Quarter Horses and Arabians, and caused severe lameness that prevented normal use. Characteristic swelling was centered over the 2nd metacarpal bone/2nd carpal bone articulation. Radiographic changes consisted of proliferative new bone, narrowed joint space, and subchondral lysis. Of 20 horses with follow-up, 7 of 14 treated horses were euthanatized within 4 years and 4 of 5 nontreated horses within 3 years. Response to treatment was short lived and considered very poor by most owners.
Conclusion—CMC-OA seemingly occurs primarily in Quarter Horses and Arabians in our region. Response to conservative treatment is very poor and short-lived with most horses being euthanatized.
Clinical Relevance—Conventional treatment methods are unsuccessful for treating CMC-OA.

INTRODUCTION

CARPOMETACARPAL (CMC) syndrome was originally reported as arthritis of the CMC joint unique to Arabian horses.[1] It was described as a progressive debilitating lameness, with arthritis of the CMC joint evident radiographically by periosteal reaction centered over the 2nd metacarpal bone/2nd carpal bone (MC2/C2) articulation and lysis of the proximal aspect of MC2. Subsequently the same authors reported on 31 horses with this condition calling it CMC-osteoarthritis (OA), and described it as primarily affecting older Arabian horses in their region.[2] We have recognized the same degenerative condition of the CMC joint for many years, primarily in Quarter Horses. This condition has also been termed carpal spavin.[3]

Our objective is to describe the clinical and radiographic changes of 33 horses (39 limbs) with CMC-OA, report long-term outcome in 20 horses and compare our findings with previous reports.

MATERIALS AND METHODS

Medical records (January 1, 1992 to July 1, 2007) were reviewed to identify horses with clinical and radiographic signs of CMC-OA, a specific form of degenerative joint disease of the CMC joint centered over the MC2/C2 articulation. Retrieved data were signalment, occupation, clinical signs, limbs involved, lameness duration and severity, presence or absence of carpal swelling, and effusion of the middle carpal joint, range of carpal flexion, response to a carpal flexion test, response to intra-articular carpal anesthesia, treatment, and presence of other lameness problems or history of trauma.

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Table 1. Radiographic Grading Criteria, Limbs Involved in 32 Horses (38 limbs)* with Carpometacarpal Osteoarthritis

Criteria	Grade 0 (Normal)	Grade 1 (Mild)	Grade 2 (Moderate)	Grade 3 (Marked)
MC2/C2 joint space width	Normal	<25% narrowing, whole joint, visible on multiple views	≥ 25% ≤ 75% narrowing, whole joint, visible on multiple views	>75% narrowing, barely visible, only visible in part of joint, or not visible
Limbs (%)	0	1 (2.6%)	14 (36.8%)	23 (60.5%)
Subchondral bone lysis MC2 or C2	None	<25% of joint surface and <0.25 cm deep	≥ 25% ≤ 50% of joint surface and 0.25–0.5 cm deep	>50% of joint surface and/or >0.5 cm deep, or on both surfaces
Limbs (%)	10 (26.3%)	3 (7.9%)	8 (21.1%)	17 (44.7%)
Proliferative new bone on MC2	None	Thickness <5 mm widest part, length <2 cm proximal to distal on dorsopalmar view	Thickness ≥ 5 mm ≤ 8 mm widest part, length ≥ 2 cm ≤ 3 cm proximal to distal, and located well below joint level on dorsopalmar view	Thickness >8 mm in widest part, length >3 cm proximal to distal, may extend to joint level on dorsopalmar view
Limbs (%)	0	8 (21.1%)	11 (28.9%)	19 (50.0%)

*Radiographs of 1 horse could not be located for examination.
MC2, 2nd metacarpal bone; C2, 2nd carpal bone.

Lameness was characterized[4] and a 4-projection radiographic examination was performed on all affected limbs, and of the contralateral limb in 4 horses with unilateral involvement. The 4 projections were: dorsopalmar (DP), lateromedial, dorsolateral palmaromedial oblique (medial oblique; DLPMO) and dorsomedial–palmarolateral oblique (lateral oblique; DMPLO). Radiographs were evaluated by a single author (S.M.B.) to identify the types, magnitude and frequency of radiographic changes of each affected CMC joint (Table 1), to identify any other changes in the middle carpal and antebrachiocarpal joints, and to record the incidence of the 1st carpal bone (C1).

Outcome was determined by telephone questionnaire of owners who were asked subjective information about: amount of interference of CMC-OA with normal activity, treatments administered and response to treatment, ability to return to normal activity, assessment of the treatment success (very poor, poor, fair, moderate, and very good), and final outcome (change of occupation, sold, pasture sound, and euthanatized).

Statistical Analysis

Descriptive statistics were used to examine the distribution and variance of the data (Statistics 8 for Windows, Analytical Software Inc., Tallahassee, FL). χ^2 analysis was used to compare age, sex, and breed of affected horses with normal hospital population and to compare the incidence of disease between Quarter Horses and Arabian horses. A 1-sample proportion test was used to determine whether carpal spavin was more likely to occur in the left or the right forelimb. A P-value $<.05$ was considered significant. A Spearman's rank correlation was used to examine the effect of duration of lameness on lameness grades. Interpretation was as follows: $\rho<0.1$, trivial correlation; $0.1 \geq \rho>0.3$, slight correlation; $0.3 \geq \rho>0.5$, moderate correlation; $0.5 \geq \rho>0.7$, substantial or large correlation; and $\rho>0.7$ very large correlation.[5,6]

RESULTS

Thirty-three horses (39 limbs) with CMC-OA were identified. Affected horses were significantly older (mean $\pm$ SD $= 13.6 \pm 4.7$ years; range, 5–23 years) than the general hospital population (7.8 ± 6.2 years; range, 1 day–43 years; $P<.0001$). The data set was divided into 3 age groups: young (<8 years), middle-aged (8–15 years inclusive) and older horses (>15 years) for further analysis. Horses with CMC-OA were significantly more likely to be in the middle-aged group (8–15 years) than in the young group (<8 years, $P<.0001$), but no significant difference was found between the middle-aged group and older group ($P=.2$).

Affected horses included 21 geldings (63.6%), 11 mares (33.3%), and 1 stallion (3%). For this period, the hospital population had 39.7% geldings, 45.3% mares, and 15% stallions. Males (22 horses) were not statistically more likely to be admitted for CMC-OA than females (11; $P=.16$; hospital population: 7285 males, 6040 females).

There were 23 (69.6%) Quarter Horses, 6 (18.2%) Arabians, 2 Paints (6%), 1 Thoroughbred, and 1 Pinto. The hospital population included 39.8% Quarter Horses, 22.5% Thoroughbreds, 8.9% Arabians, and lower frequencies of other breeds. Quarter Horses were over-represented compared with the general hospital population ($P=.02$); however, this was not so for Arabian horses ($P=.23$). There was no significant difference ($P=.74$) in incidence of CMC-OA between Quarter Horses and Arabians.

Twelve horses (36.3%) were used for western riding or ranch work; 8 (24.2%) for show, jumping, or 4-H; 7 (21.2%) for pleasure riding; 5 (15.1%) for breeding, and 1 horse for endurance.

Clinical Findings

Horses were admitted for lameness, and CMC-OA involved the left carpus in 19 horses (57.6%), the right carpus in 8 horses (24.2%) and was bilateral in 6 with no significant difference between left and right sides ($P = .1$). One horse with bilateral disease had unilateral lameness, and for another horse information regarding lameness of the less affected limb was not recorded. Five horses (6 limbs) had other concurrent lameness including osteochondral chip fracture in the middle carpal joint, OA of the middle carpal joint, OA of the proximal interphalangeal joint, bilateral navicular syndrome, OA of the antebrachiocarpal joint, and bilateral tarsometatarsal OA.

Nine limbs were excluded from calculation of the duration and severity of lameness because of either presence of concurrent lameness (6 limbs) or imprecise history (3 limbs). Duration of lameness ranged from 3 weeks to 72 months (mean, 11.6 months). Thirteen limbs (43.3%) had grade 4/5 lameness, 11 limbs (36.7%) were grade 3, 5 limbs (16.7%) grade 2, and 1 (3.3%) was without evident lameness. There was moderate correlation ($P = .3$) between lameness grade and duration (mean, 12.8 months; median, 6 months; interquartile range, 3–12 months). For bilateral involvement, the severity of the lameness was often different between limbs. In 2 horses, lameness and a positive flexion test was only present in 1 limb on initial examination despite clinically noticeable swelling and radiographic changes typical of CMC-OA in both limbs. Both horses were readmitted 7 months later with lameness in the previously nonlame limb and were positive to carpal flexion. In 1 horse with bilateral involvement that had unilateral involvement initially, lameness, carpal swelling, and radiographic changes developed in the opposite leg after only 7 months.

Swelling considered typical of CMC-OA was located on the medial aspect of the carpus in all 39 limbs, and was firm, nonfluctuant, and not painful on palpation. It was best identified by facing the dorsal aspect of the carpus or by observing from a slightly dorsolateral angle where the swelling gave the carpus a "squared off" or "shouldered" shape to its distal and medial aspect (Fig 1). Swelling varied dramatically in size, but was consistently centered over the MC2/C2 articulation. In horses with large masses, the swelling extended dorsally onto MC3 and proximally toward the middle carpal joint. In 2 limbs, there was slight varus deformity of the carpus.

Middle carpal joint effusion was present in 3 limbs (7.7%), not present in 18 limbs (46.1%), was not recorded in 18 limbs. Of the 3 limbs with middle carpal joint effusion, 1 had history of carpal trauma, osteophytes, and a small osteochondral fragment in the middle carpal joint, and 1 had osteophyte production. Range of carpal motion was reduced in 16 limbs (41%), normal in 2 (5%), and not recorded for 21 limbs (53.8%). Reduction in carpal flexion was not measured, but was estimated at ~ 15–20°. The decreased range of motion prevented contact of the palmar aspect of the metacarpal region with the caudal aspect of the antebrachium during flexion of the carpus. After flexion of the carpus for 2 minutes, the severity of the lameness was increased in 28 limbs (71.8%), unchanged in 2 (5.1%), and was not recorded for 9 limbs (23%).

Fig 1. A horse with the typical swelling (double arrows) associated with carpometacarpal osteoarthritis. There is a "squared-off" or "shouldered" appearance to the distal medial aspect of the carpal joint at the 2nd carpal bone/2nd metacarpal bone articulation.

Intra-articular anesthesia of the middle carpal joint resulted in marked reduction or elimination of lameness in 6 of 7 horses. In the horse that did not improve, lameness was eliminated by injection of local anesthetic into the CMC joint through the palmar pouches.

Radiographic Findings (Table 1, Figs 2–4)

Radiographic changes were centered over the MC2/C2 articulation, and occasionally involved MC3 and adjacent area of the CMC joint. Extra-articular changes consisted of soft tissue swelling and proliferative new bone growth at the proximal aspect of MC2. In some limbs, swelling was almost exclusively soft tissue (Fig 2A and B)

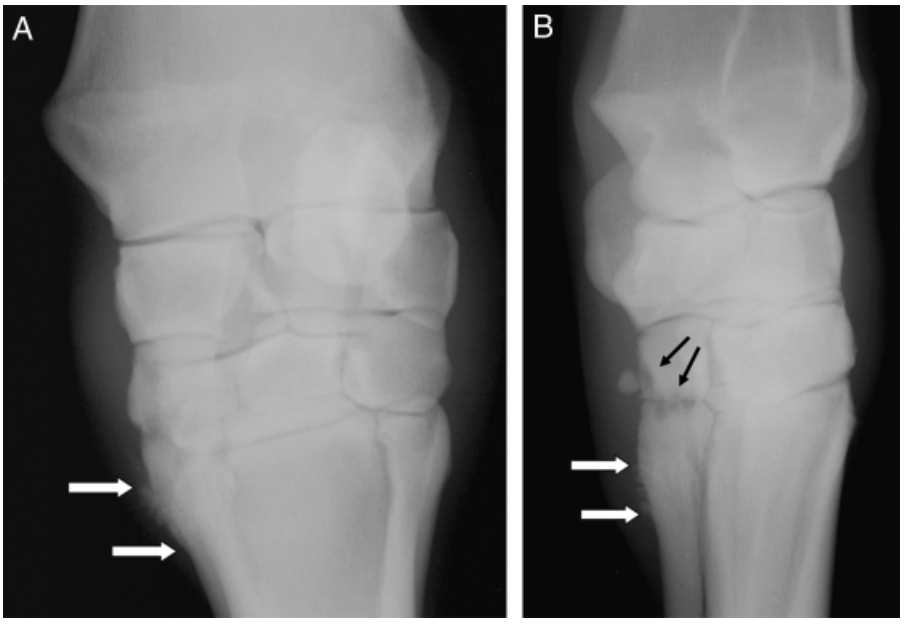

Fig 2. Dorsopalmar (A) and dorsomedial–palmarolateral oblique (lateral oblique; B) radiographic projections. Radiographic grades are reported sequentially as 2nd metacarpal bone (MC2)/2nd carpal bone (C2) joint space width, subchondral bone lysis on MC2 or C2; and proliferative new bone on MC2, respectively. There is very slight proliferative new bone on proximal MC2 starting ~ 1.5–2 cm below the articular surface and extending distally for 1.5 cm (between white arrows). There is extensive subchondral lysis, especially of MC2 (black arrows). This limb was graded as 2, 2, and 1.

whereas in others, moderate (Fig 3A and B) or severe (Fig 4A and B) bony reaction was present. Proliferative new bone was best observed on either the DP or DMPLO (lateral oblique) projections and in some limbs, it extended more proximally in the DP than in the lateral oblique projection (Fig 5A and B). In limbs with very slight proliferative new bone, the bony reaction started ~ 1.5 cm below the MC2/C2 articulation (Fig 2A and B)

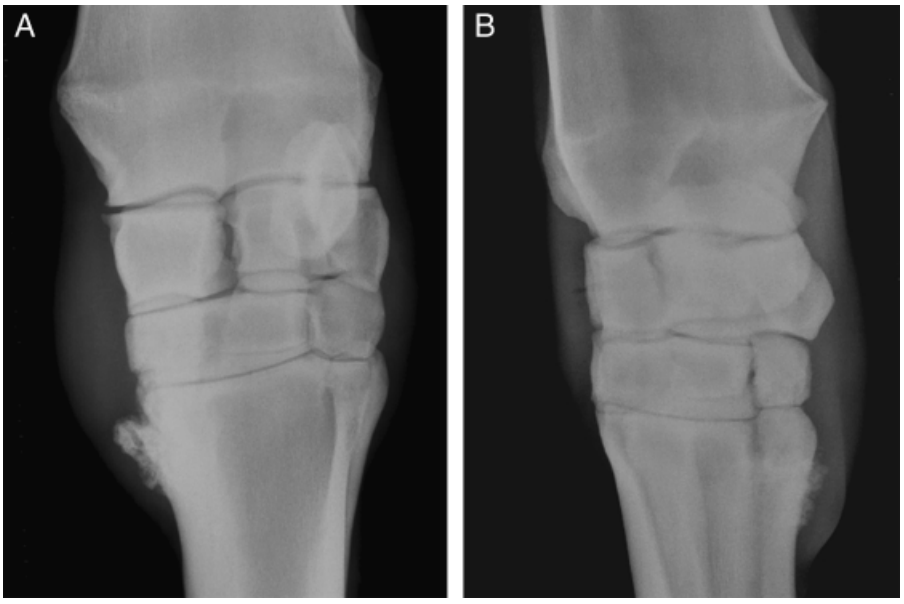

Fig 3. (A, B) Proliferative new bone is similar in distribution to Fig 2. but more readily identified. There is an absence of subchondral lysis of the 2nd metacarpal bone /2nd carpal bone articulation, but the joint space is narrowed. This limb was graded as 1, 0, and 2.

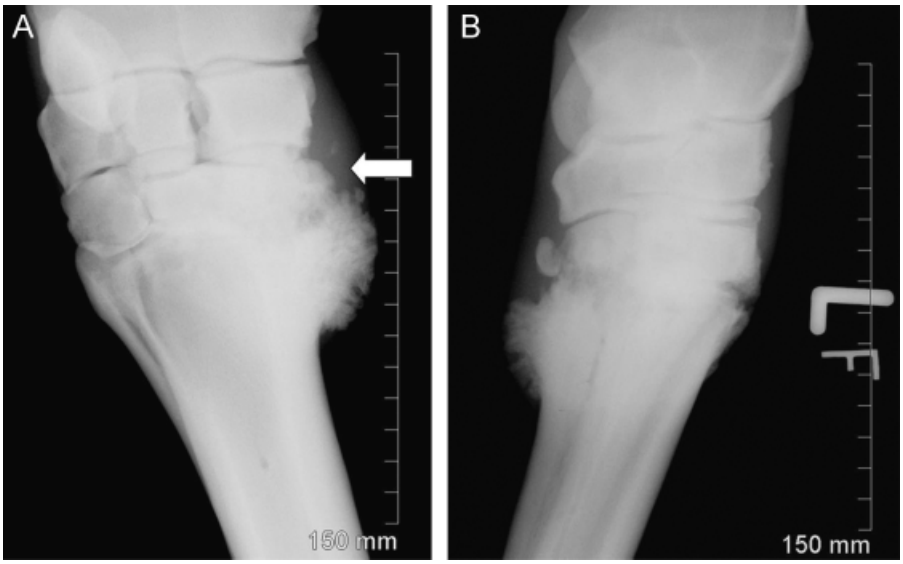

Fig 4. (A, B) Marked proliferative new bone extends proximally onto the 2nd carpal bone (C2), above the 2nd metacarpal bone (MC2)/C2 articulation (arrow) and extends more proximally on the dorsopalmar than on the lateral oblique view. Also note the loss of joint space, the subchondral remodeling, and the apparent fusion of the MC2/C2 articulation. This limb was graded as 3, 3, and 3.

and with more new bone proliferation, extended both proximally toward the CMC joint, and distally on MC2 for a distance of 4.5–5 cm from its articular surface (Fig 4A and B). With increased new bone production, the mass became larger, had a mottled and lobulated surface, and eventually developed into a dense mass of bone with a smooth surface. In some limbs, the reaction was so extensive it appeared to be attempting to ankylose the CMC joint at the MC2/C2 articulation, often seen dorsal to MC2 on the adjacent MC3, and occasionally on C2 (Fig 4A and B). Two types of intra-articular changes were observed: narrowing of the joint space at the MC2/C2 articulation, and lysis of the subchondral bone of MC2 or C2. Both changes were best evaluated on the lateral oblique (DMPLO) projection (Figs 2B and 5B). Radiographs of 32 horses (38 limbs) were available for review.

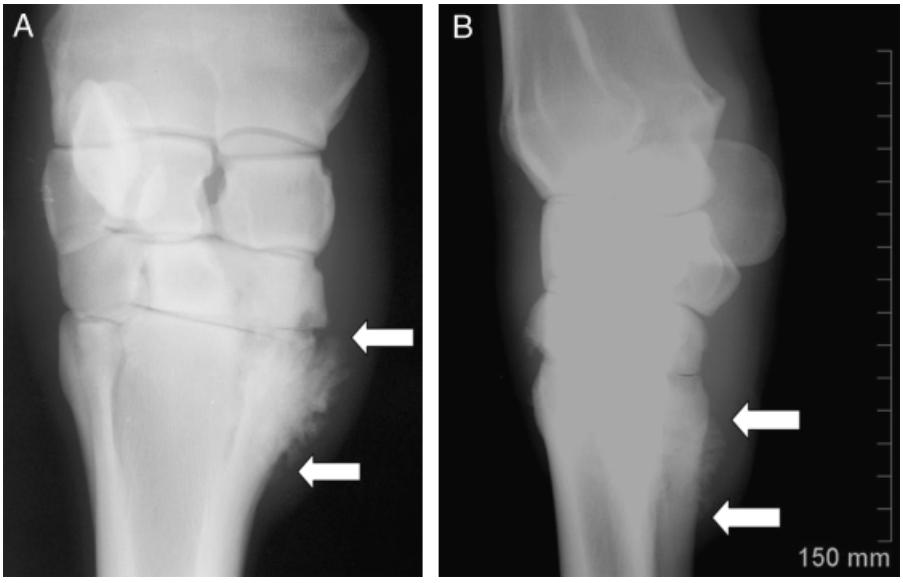

Fig 5. Dorsopalmar (DP) (A) and lateral oblique radiographic (B) projections. The arrows mark the proximal and distal edges of the proliferative new bone, which extends higher on the DP projection than on the lateral oblique projection. This limb was graded as 2, 2, and 3.

All limbs had MC2/C2 joint space narrowing, 37 had either moderate or marked MC2/C2 narrowing, 25 had moderate or marked subchondral lysis, and 10 (26%) did not have any visible subchondral lysis. Thirty limbs (79%) had moderate or marked proliferative new bone growth. C1 was only identified in 9 (24%) limbs. Mild signs of degenerative joint disease were present in the middle carpal joint of 2 limbs (5%), with a chronic osteochondral fragment present in 1 joint. Signs of degenerative joint disease were also observed in the antebrachiocarpal joint of 2 other horses.

Follow-up Questionnaire

Thirteen horses (17 limbs) were treated by CMC arthrodesis.[7] Of the other 20 horses, all but 1 had follow-up with a mean time of 5.2 years (median, 6.2 years; range, 0.5–11.6 years). Seven horses (7; 37%) were used for western riding; 6 (32%) for show, jumping, or 4-H; 3 for pleasure riding (16%); and 3 as broodmares. Before treatment, the severity of CMC-OA was sufficient to interfere with normal activity in 18 of the 19 horses and was considered marked in 13 horses, moderate in 4, and slight in 1. In 1 broodmare, the owner did not think CMC-OA interfered with use.

Fourteen horses were treated; 7 were administered phenylbutazone intermittently, 2 had intra-articular (middle carpal joint) corticosteroids (1 and 3 times, respectively), 1 horse had intra-articular (middle carpal joint) hyaluronic acid administration once, 1 horse had 4 injections of intramuscular polysulphated glycosaminoglycans, 2 horses had a single intra-articular (middle carpal joint) injection of corticosteroids combined with intermittent phenylbutazone administration, and 1 horse had surgical removal of MC2. Slight to moderate improvement for a mean of 4.6 months (range, 1–12 months) occurred in 9 horses: 4 administered phenylbutazone, 2 administered intra-articular corticosteroid, 1 with removal of MC2, 1 administered intramuscular polysulfated glycosaminoglycans, and 1 administered intra-articular hyaluronic acid. No improvement occurred in 3 horses and for 2 horses, owners were uncertain of outcome. Information about return to original activity was available for 12 treated horses. Six horses returned to their original activity for a mean of 18 months (median, 7.5 months; range, 3–60 months) including 2 broodmares, 1 of which was serviceable for 5 years, and 1 saddle horse that was used despite lack of improvement with treatment. Six of these 14 treated horses were euthanized because of disease severity by 4 years after diagnosis (mean, 15.1 months; range 2–48 months). Treatment outcome was rated by the owners of 12 horses; for 11 of them, outcome was rated as very poor, whereas the owner of 1 horse that responded well to intermittent administration of phenylbutazone, rated outcome as very good.

Five horses (26%) were not treated and none returned to their original use; 4 were euthanized because of lameness severity: 1 at diagnosis, 1 at 3 months, and 2 at 2–3 years after diagnosis. The 1 horse alive at follow-up (6 months) could not be used by the owners. Combining outcome for untreated (5) and treated (14) horses, 10 (53%) were euthanized by 4 years, 2 (11%) died of unknown reasons, 1 (5%) was euthanized because of equine protozoal myelitis at 6 years, and 6 (32%) were still alive. Of those alive, 3 (16%) were unusable, 1 was sold because it was too lame for intended use, 1 was considered comfortable at pasture, and 1 was used as a broodmare.

DISCUSSION

CMC-OA was initially described as unique to Arabian horses[1] and subsequently as primarily affecting mostly female Arabian horses, even though it was also observed in Quarter Horses and Thoroughbreds.[2] We identified CMC-OA in 5 breeds and were unable to show a significant difference in disease incidence between Arabian horses and Quarter Horses, but found a higher incidence in horses >8 years. Like others[2] we found a higher incidence of left carpal involvement but, a lower incidence of bilateral involvement (18.2% versus 30.4%[2]).

Clinical Findings

Affected horses commonly have chronic severe lameness, typical carpal swelling, positive response to carpal flexion, and remission of lameness with intra-articular injection of local anesthetic into the middle carpal joint. We identified 2 horses where carpal swelling and radiographic changes were identified before recognizable lameness and a positive flexion test, and another horse where lameness, swelling, and radiographic changes developed over 7 months. These observations support the typical chronicity of CMC-OA, but suggest that mild or even subclinical CMC-OA occurs and that changes can progress rapidly. Contrary to another report where carpal varus occurred in 21 of 23 horses with CMC-OA,[2] we identified varus in 2 of 39 limbs (5%). The reason for this difference is unknown; however, it may be a consequence of CMC-OA rather than a predisposing factor.

Similarly, we found that intra-articular injection of local anesthetic into the middle carpal joint was diagnostically useful but also had 1 limb, where lameness was only relieved by direct administration into the palmar aspect of the CMC joint. This observation suggests that the normal communication between the CMC and middle carpal joint, between C3 and C4 on the lateral side of

the joint,[8,9] are not present in all limbs, or may obstructed by CMC-OA changes. Presence or absence of middle carpal joint effusion was not reported for CMC-OA,[2] even though 35% of horses in that study had carpal trauma and most had osteophytes within the middle carpal joint. We found middle carpal joint effusion in only 3 horses, 2 of which had osteophyte production but only 1 had a history of carpal trauma. Although it seems logical that CMC-OA could lead to degenerative changes and osteophyte production of the middle carpal joint, because of their normal communication,[8,9] we do not believe that this necessarily occurs.

Radiographic Findings

The radiographic changes associated with CMC-OA are quite characteristic and usually allow easy differentiation from other carpal problems. The 3 radiographic changes we identified were (1) proliferative new bone growth, primarily on proximal MC2 but also on adjacent MC3, C2, and C3; (2) narrowing of the MC2/C2 articulation; (3) subchondral bone lysis of MC2 and/or C2. Subjectively, joint narrowing was seemingly an early radiographic change; however, in some joints there was substantial proliferative bone growth without marked joint space narrowing. Lysis of subchondral bone often seemed to occur earlier, and subjectively seemed more common and marked in MC2 than C2 (Fig 2B); however, generalized lysis of subchondral bone of MC2 and C2 was observed in severe cases.

The grading system we used was helpful characterizing the incidence and severity of radiographic changes. Proliferative new bone and decreased MC2/C2 joint width were identified in all affected limbs; 79% having moderate and 97% having severe changes. Subchondral bone lysis was not as reliable, occurring in 74% of affected joints. New bone production on proximal MC2 may develop from injury to the distal attachment of the medial collateral ligament, which is located more medial than palmar,[8] leading to secondary arthritic changes. C1, located in the distal portion of the medial collateral ligament,[10] is present in 33% of horses.[11] Its presence theoretically could weaken the distal attachment of the medial collateral ligament, but given that we only identified C1 in 9 of 38 affected limbs (24%) it seems unlikely that C1 plays a role in the pathogenesis of CMC-OA.

Outcome

In an earlier report, 20 Arabian horses with CMC-OA had a very poor prognosis[2]; 2 horses improved, but none were able to return to work and 10 were euthanatized. None of our untreated horses returned to work, and 4 of 5 were euthanatized within 3 years, confirming that horses with CMC-OA that are untreated have a grave prognosis and short life expectancy. Treated horses faired only slightly better with 64% (9 of 14) having slight or moderate improvement, although this only lasted a mean of 4.6 months. Whereas 6 of 12 horses returned to work, this included 2 broodmares, and 1 saddle horse that was used despite apparent lack of improvement with treatment. Most owners considered the response to treatment very poor and 6 of 14 treated horses were euthanized within 4 years.

We believe that CMC-OA[1,2] and carpal spavin[3] are the same condition. Whereas the clinical and radiographic changes associated with CMC-OA are quite characteristic, its low incidence may explain the earlier lack of detailed reports. Further, we suggest that CMC-OA is not a new condition, but likely reflects what has previously been called a knee splint, an enlargement of the proximal portion of the splint bone that may lead to OA within the CMC joint.[12] A more specific description of the signalment, and clinical and radiographic signs for knee splints could not be found.

In conclusion, we found no difference in incidence of CMC-OA between Quarter Horses and Arabian horses in our geographic region. Swelling and radiographic signs typical of CMC-OA can occasionally be identified before onset of lameness and progression can occur rapidly. Conventional treatments for CMC-OA are ineffective and alternative treatment methods are needed.

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