

Severe Carpometacarpal Osteoarthritis in Older Arabian Horses

ERIN D. MALONE, DVM, PhD, Diplomate ACVS, CLIFF M. LES, DVM, PhD, and
TRACY A. TURNER, DVM, MS, Diplomate ACVS

Objective—To report a severe form of carpometacarpal osteoarthritis (CMC-OA) affecting primarily older Arabian horses.

Study Design—Retrospective study.

Animals—Thirty-one horses with CMC-OA.

Methods—Carpal radiographs (405 sets) from 3 hospitals were evaluated to identify horses with marked osteoproliferative reaction across the medial aspect of the CMC joint. Owners were contacted to obtain detailed histories and status updates. Necropsy specimens of the CMC joint were evaluated at 2 hospitals to determine the prevalence of 2 variations in the articulation between the proximal second and third metacarpal bones.

Results—Thirty-one horses were identified as having marked osteoproliferative reaction at the CMC joint. Twenty-three (74%) were Arabian horses. Of the Arabian horses, the average age at admission was 14.4 years. Eight (34.8%) Arabian horses had a known history of trauma. Most were no longer rideable at presentation. Ten of the horses were subsequently euthanatized because of lameness. The dorsal and palmar articulations between the second and third metacarpal bones were examined in 177 horses. The palmar articulation was absent in 48% of Arabian horses and 12.5% of non-Arabian horses at 1 center, including 4 horses with CMC-OA. At the second center, the palmar articulation was present in 8 of 8 Arabian horses but was absent in 22 of 92 (24%) non-Arabian horses.

Conclusions—An increased frequency of this crippling form of OA was observed in Arabian horses. It may reflect an increased prevalence, in some geographical regions, of an absent palmar articulation between the second and third metacarpal bones.

Clinical Relevance—Carpal trauma in some Arabian horses may result in unexpectedly severe carpometacarpal osteoarthritis.

© Copyright 2003 by The American College of Veterinary Surgeons

CARPAL DISEASE has been reported in racing Standardbred, Thoroughbred, and Quarter Horse horses but rarely in other breeds or horses used for other activities.¹⁻³ The carpometacarpal (CMC) joint is uncommonly affected because it has a negligible range of motion.² Fractures of the third carpal bone or of the proximal aspect of the third metacarpal bone can lead to CMC osteoarthritis (OA) and is most commonly observed in horses used for racing.¹ We have identified a severe form of CMC-OA in aged Arabian horses that has consistent radiographic and clinical findings. This syn-

drome is associated with a gradual onset of lameness, followed by rapid progression to a debilitating disorder. The objective of our study was to describe this CMC-OA syndrome and to identify potential contributing factors, including anatomic differences that might contribute to its development.

MATERIAL AND METHODS

The medical records and radiographs of horses with a diagnosis of carpal disease (n = 107) between 1983 and

From the Department of Clinical and Population Sciences, College of Veterinary Medicine, University of Minnesota, St. Paul, MN; and Bone and Joint Center, Henry Ford Hospital, Detroit, MI.

Presented in part at the Veterinary Orthopedic Society meeting, 1995.

Work was performed at the University of Minnesota College of Veterinary Medicine and at the JD Wheat Veterinary Orthopedic Research Laboratory, School of Veterinary Medicine, University of California-Davis.

Address reprint requests to Erin D. Malone DVM, PhD, 225K VTH, 1365 Gortner Ave St Paul, MN 55108.

© Copyright 2003 by The American College of Veterinary Surgeons

0161-3499/03/3203-0001\$30.00/0

doi:10.1053/jvet.2003.50026

2000 at the University of Minnesota (UMN) were reviewed. Carpal radiographs were also evaluated at Littleton Large Animal Clinic, Colorado (n = 267; 1987-1995, all horses with carpal radiographs) and at the University of Florida (n = 31; 1989-1993, horses with a diagnosis of OA only). Cases of CMC-OA seen by local practitioners were also solicited by UMN newsletter. Owners of horses with CMC-OA were contacted to obtain a detailed clinical history and to ascertain the status of the horse in terms of lameness, therapeutic responses, and usage. Follow-up examinations (n = 8) including ultrasound of the carpus (n = 6) were performed at UMN. Necropsy examination of affected joints was performed on 4 horses at UMN.

To determine the prevalence of anatomic variation in the MC2-MC3 articulation, necropsy studies were performed at the University of California (n = 100)⁴ and at UMN (n = 77). Ultrasonographic examination of the carpus was also performed in 18 horses that did not have evidence of carpal disease and were located near UMN. Breed distribution and age comparisons were made using a Wilcoxon signed rank test. Comparisons between anatomic variations were made using a χ^2 test. Significance was set at $P < .05$.

RESULTS

University of Minnesota

Of 107 horses with lameness localized to the carpus, 36 had radiographic lesions of CMC-OA. Whereas 15 horses had mild osteophyte production along the margins of the carpal bones with minimal periosteal response, 21 (58%) had marked osteoproliferative reaction centered over the second carpal bone (C2), second metacarpal bone articulation (C2-MC2). Of these, there were 17 (81%) Arabian (hospital population, 23.4% Arabian horses; $P = .0001$), 2 Thoroughbred, and 2 Quarter horses. The Arabian horses with C2-MC2 OA were older (mean, 14.4 years; $P = .0001$) than horses with other forms of degenerative carpal disease (mean, 8 years). Horses with C2-MC2 OA were admitted 1-48 months (mean, 8.7 months; median, 6 months) after owners noted either lameness or carpal enlargement. Local practitioners identified 4 other horses with C2-MC2 OA, all Arabians.

Littleton Large Animal Clinic

Six horses (2 Arabians, 2 Quarter horses, 2 Thoroughbreds) with C2-MC2 OA were identified.

University of Florida

No horses with C2-MC2 OA were identified; 52% of these horses were Thoroughbreds, and there were no Arabians.

Arabian Horses with C2-MC2 OA

Therefore, 31 horses with C2-MC2 OA were identified, 23 (74%) of which were Arabian (Table 1). Of the Arabian horses, 13 (56.5%) were mares, and 10 were geldings. At least 16 Arabian horses had previously been shown (various classes, including halter, jumping, eastern and western pleasure, dressage, and endurance); 2 horses were still being ridden at the time of admission, whereas the others were too lame for riding. None of the horses had previous limb surgery, 1 had a preexisting conformational defect (mild fetlock varus), and 8 had known episodes of carpal trauma (Table 1) before clinical signs developed.

Two Arabian horses with C2-MC2 OA syndrome were grade II/V lame⁵ at admission, whereas the other 21 Arabians were grade III lame, and all had a strong positive response to carpal flexion. Decreased range of motion of the carpus was evident in all affected horses. At least 21 of 23 Arabian horses had abnormal angulation (carpus varus) of the limb with bony proliferation along the distomedial aspect of the carpus when evaluated for lameness. The left leg was affected in 9 horses, the right in 6, and bilateral lesions occurred in 7 (lame leg not recorded in 1 horse). Middle carpal joint anesthesia alleviated the lameness in 4 horses in which it was performed. C2-MC2 OA was the primary lameness problem identified in most horses; however, 2 Thoroughbreds also had severe OA of the fetlock joint in another limb.

Radiographic lesions (Fig 1) were best observed on dorsopalmar and dorsomedial-palmarolateral oblique views. In the Arabian horses with C2-MC2 OA, a moderate to severe osteoproliferative reaction was noted, originating at the proximal portion of MC2 in the area of the attachment of the joint capsule and medial collateral ligament. The reaction extended proximally, following the approximate path of the collateral ligament and partially bridging the medial aspect of the carpometacarpal joint. Loss of joint space occurred at the MC2-C2 and MC2-MC3 articulations. Lysis of the proximal aspect of MC2 was common. Most horses also had osteophytes within the middle carpal joint.

Follow-up information was obtained for 20 Arabian horses. Two horses improved several months after diagnosis of C2-MC2 OA coincident with radiographic

Table 1. Descriptive Information for Horses With Carpometacarpal Osteoarthritis

Case	Breed	Sex	Age (years)	Known Trauma	Palmar Articulation	Duration of Lameness at Follow up (years)	Follow up
UMN1	Arab	MC	20	Fell—trail ride	Unknown	0.9	E—lameness
UMN2	ArabX	F	13	No	Absent (US)	7.8	Alive
UMN3	Arab	MC	18	Fell through ice	Unknown	0.5	E—lameness
UMN4	Arab	F	15	No	Absent (necropsy)	9.3	E—lameness
UMN5	Arab	MC	15	Fell—trail ride	Absent (US)	10.6	Alive
UMN6	Arab	F	10	Cart accident	Unknown	2	E—multiple reasons
UMN7	Arab	F	20	No	Absent (US)	6.8	Alive
UMN8	Arab	MC	12	Kick to carpus	Absent (US, Necropsy)	10	E—lameness
UMN9	Arab	MC	11	Ran into pole	Unknown	7	Lost
UMN10	Arab	MC	11	No	Absent (US)	1.5	Lost
UMN11	ArabX	F	19	No	Absent (US)	6.3	Alive
UMN12	Arab	F	9	No	Unknown	1.2	Alive
UMN13	Arab	F	12	No	Unknown	2.9	Alive
UMN14	ArabX	F	9	No	Unknown	1	Sold; Lost
UMN15	ArabX	F	16	Injured during lesson	Unknown	0.8	Alive
UMN16	Arab	MC	20	No	Absent (necropsy)	3	E—lameness
UMN17	ArabX	F	13	No	Unknown	2.5	E—lameness
LLAC1	Arab	MC	15	No	Unknown	3	E—lameness
LLAC2	Arab	MC	13	No	Unknown	0.8	Lost
OTH1	Arab	F	14	No	Unknown	2.5	E—lameness
OTH2	Arab	MC	14	No	Unknown	9	Lost
OTH3	Arab	F	18	Ran into object	Unknown	6	E—multiple reasons
OTH4	Arab	F	?	No	Unknown	?	Lost
UMN18	QH	F	27	Kick to carpus	Unknown	6	E—multiple reasons
UMN19	QH	F	12	No	Unknown	0.5	Lost
UMN20	TB	MC	28	Fell—racing	Unknown	21	Alive
UMN21	TB	F	24	Injured—racing	Absent (necropsy)	?	E—multiple reasons
LLAC3	QH	F	7	No	Unknown	?	Lost
LLAC4	QH	F	16	No	Unknown	?	Lost
LLAC5	TB	MC	24	No	Unknown	?	Lost
LLAC6	TB	MC	10	No	Unknown	?	Lost

Abbreviations: UMN, University of Minnesota; LLAC, Littleton Large Animal Clinic; OTH, other sites of origin; QH, Quarter horse; TB, thoroughbred; F, female; MC, gelding; US, diagnosed by ultrasound.

evidence of fusion of the C2-MC2 articulation; however, neither improved sufficiently to be ridden. On arthroscopic examination in 1 Arabian horse, a torn medial palmar intercarpal ligament and moderate cartilage fibrillation throughout the intercarpal joint were identified. Ultrasonographic evaluations of the collateral ligaments and palmar ligaments of 2 horses did not reveal any lesions within these structures; however, bony reaction made some areas difficult to evaluate. Ten Arabian horses were euthanatized because of severe lameness, 7 had been retired, and 6 were lost to follow-up. Five horses lived at least 5 years (range, 0.5-10.6 years) after the lameness became severe enough to preclude use for riding.

Four horses (3 Arabians and 1 Thoroughbred) with C2-MC2 OA syndrome were necropsied. Both limbs were affected to some degree in 3 horses, with 1 limb being more severely affected. Disarticulation and evaluation of the joint were difficult because of osseous proliferation and bony remodeling. Collateral ligaments were displaced by the bony proliferation but appeared intact albeit adhered to the bone in some areas. In the primarily affected limbs, minimal carti-

lage could be detected grossly or on histopathology. Histopathologic evaluation revealed extensive remodeling of bone and marrow with fibrous tissue infiltration, consistent with severe osteoarthritis. All 4 horses were missing the palmar articulation on MC2 on the more normal leg; however, this could not be evaluated on the more severely affected leg because of osseous proliferation. In 1 horse, both the medial and lateral interosseous ligaments of the carpometacarpal joint were grossly enlarged. On histopathology, portions of the normal stroma of these ligaments were found to be infiltrated with fibrocartilage, and some areas appeared mildly disrupted. No inflammatory lesions were detected.

Anatomic Variation in MC2-MC3 Articulation

In necropsy studies, 2 forms of MC2-MC3 articulation were identified. In most horses, MC2 and MC3 were in contact at 2 sites, both dorsal and palmar to a CMC interosseous ligament (Fig 2A). In 100 horses examined at the University of California-Davis, the palmar articulation was absent in 22 of 92 (24%) non-Arabian horses

and was present in all 8 Arabian horses; the breed differences were not significant ($P = .12$). At UMN, the palmar articulation was absent in 14 of 29 (48%) Arabian horses and 6 of 48 (12.5%) non-Arabian horses (Fig 2B); these breed differences were significant ($P = .001$). When the data from both institutions were combined, significantly more Arabian horses did not have a palmar articulation than would be expected ($P = .044$). Both the depth (dorsal to palmar) of the palmar articulation (if present) and the width (medial to lateral) of any gap between the bones varied widely between horses but were similar between limbs of the same horse in 172 of 177 (97%) horses. No noticeable variation occurred in the dorsal articulation.



Fig 1. Radiograph: dorsopalmar projection. Arabian horse with carpometacarpal osteoarthritis. There is marked osteoproliferative reaction, which originates at the proximal portion of the second metacarpal bone in the area of the attachment of the joint capsule and medial collateral ligament and extends proximally, following the approximate path of the collateral ligament. Lysis of the proximal aspect of the second metacarpal bone is evident as is loss of joint space at the articulation between the second metacarpal bone and the second carpal bone and between the second metacarpal bone and the third metacarpal bone.

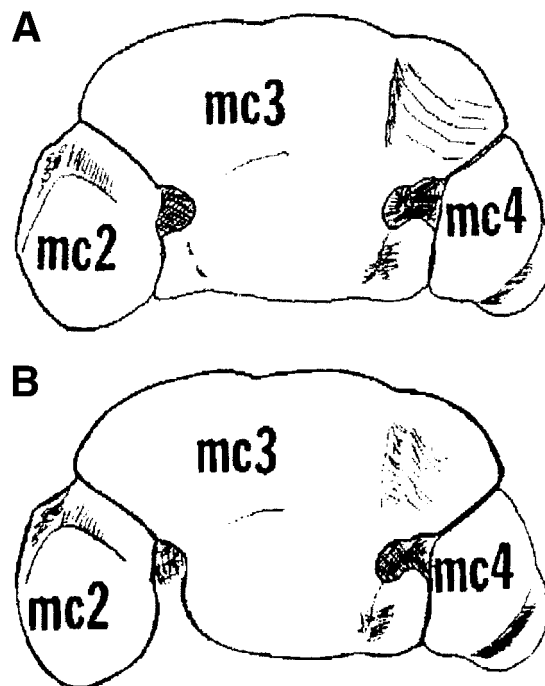


Fig 2. Dorsal view of the proximal aspect of the metacarpal bones at the carpometacarpal joint. (A) Most horses have contact between MC2 and MC3 at 2 sites, both medial and palmar to a carpometacarpal interosseous ligament. (B) The palmar articulation was absent in 13 of 28 (46%) Arabian horses and 6 of 48 (12.5%) non-Arabian horses at the University of Minnesota. MC2, second metacarpal bone; MC3, third metacarpal bone; MC4, fourth metacarpal bone.

The interosseous ligament located between the 2 bones could be observed ultrasonographically in horses without the palmar articulation. Six Arabian horses with C2-MC2 OA that had ultrasound examinations did not have the palmar articulation. When horses with no evidence of carpal disease were examined by ultrasound, the palmar articulation was absent in 13 of 18 (72%) Arabian horses and 1 of 10 (10%) non-Arabian horses; this difference was significant ($P = .002$).

DISCUSSION

C2-MC2 OA is, to our knowledge, a previously undescribed condition that appears to be strongly associated with the Arabian breed, at least in Minnesota. The osseous reaction identified in these horses suggests a significant loss of joint stability because the carpometacarpal joint is a low motion joint and ordinarily would not be expected to undergo callus-type formation. Instability may occur because of trauma, conformational defects, or inflammatory-mediated degeneration of the ligamentous structures of the carpus.

Conformational defects are often breed associated but are certainly not restricted to Arabian horses. Many of these horses were shown successfully in halter and performance classes, and only one had a preexisting limb conformation abnormality.

In dogs, cruciate ligament damage has been associated with collagen antibodies and immune complex formation.⁶ Although only a few horses were examined for ligamentous injury by ultrasound and/or histopathology, we found no evidence for primary degeneration or inflammation of these structures. However, we were not able to evaluate intraarticular structures in most joints, and degeneration of these structures has been speculated to lead to gradual development of arthritis in the horse.⁷ Such deterioration could help explain the later age of onset of C2-MC2 OA.

Direct injury was temporally associated with development of bony enlargement in 35% of the horses and may have occurred but been unobserved in the others. Injury is also consistent with the delayed onset of disease and the presence of other lesions such as fetlock arthritis and medial palmar intercarpal ligament tears.⁷ The osseous reaction appeared to follow the lines of the collateral ligament and could indicate significant trauma to this structure or to the joint capsule. Trauma and subsequent alterations in stability could also account for the hypertrophy observed in the interosseous ligaments of the CMC joint in 1 horse.⁸ However, a purely traumatic origin would not explain the apparent predisposition for Arabian horses to develop C2-MC2 OA.

The anatomic variation in the articulations between MC3 and MC2 found in some horses could predispose them to trauma in that region or could increase the risk of instability after trauma. There is evidence that the dorsal and medial aspects of the carpus are already more prone to damage because of the anatomic alignment.^{2,9} The medial facet of the distal radius is the largest of the 3 articulating regions.¹⁰ In both proximal joints, some of the load is distributed to the intercarpal ligaments by medial-to-lateral displacement of the carpal bones. However, the radial facet of the third carpal bone receives the entire load from the radial carpal bone because of its concave surface.² MC2 normally carries more weight than the lateral fourth metacarpal bone (MC4) because of its flat articulation when compared with the oblique structure of MC4.² It may be that horses without a palmar articulation between MC2 and MC3 are predisposed to tearing of the soft tissue structures because of decreased bony support or have abnormal mobility of the CMC

joint. This would also account for the tendency of lesions to occur bilaterally.

A regional variation in the prevalence of one or another form of metacarpal articulation is conceivable. It may be that certain lines of horses are more prone to not having a palmar articulation and that more problems associated with this anatomic variant could be found in regions of the country where those bloodlines are more popular.

Regardless of the inciting cause, C2-MC2 OA is apparently a progressive degenerative joint disease that leads to loss of use of the horse. At the end stage of the disease, conservative medical management is largely ineffective. Early intervention to minimize the effects of instability may be necessary to restore a functional degree of soundness. On the other hand, even with delayed diagnosis, these horses do remain comfortable at pasture as broodmares or pets for a prolonged period. If the anatomic variant described is found to predispose horses to C2-MC2 OA, ultrasound evaluation may be useful to identify horses at risk.

REFERENCES

1. Ross MW: Unusual lamenesses associated with the carpus and proximal aspect of the third metacarpal bone in Standard-bred racehorses. *Proc Am Assoc Equine Practnr* 37:565-577, 1991
2. Bramlage LR, Schneider RK, Gabel AA: A clinical perspective on lameness originating in the carpus. *Equine Vet J Suppl* 6:12-18, 1988
3. Pool RR: Joint disease in the athletic horse: A review of pathologic findings and pathogenesis. *Proc Am Assoc Equine Practnr* 41:20-34, 1995
4. Les CM, Stover SM, Willits NH: Necropsy survey of metacarpal fusion in the horse. *Am J Vet Res* 56:1421-1432, 1995
5. American Association of Equine Practitioners Newsletter, Lexington, KY, March 1983, pp 12
6. Niebauer GW, Wolf B, Bashey RI, et al: Antibodies to canine collagen types I and II in dogs with spontaneous cruciate ligament rupture and osteoarthritis. *Arthritis Rheum* 30:319-327, 1987
7. Whitton RC, Rose RJ: The intercarpal ligaments of the equine midcarpal joint. Part 2: The role of the palmar intercarpal ligaments in the restraint of dorsal displacement of the proximal role of carpal bones. *Vet Surg* 26:367-373, 1997
8. Whitton RC, Kannegieter NJ, Rose RJ: The intercarpal ligaments of the equine midcarpal joint. Part 3: clinical observations in 32 racing horses with midcarpal joint disease. *Vet Surg* 26:374-381, 1997
9. Colahan P, Turner TA, Poulos P, et al: Mechanical functions and sources of injury in the fetlock and carpus. *Proc Am Assoc Equine Practnr* 33:689-699, 1987
10. Getty R: Equine osteology, in Getty R (ed): *Sisson and Grossman's The Anatomy of the Domestic Animals, Volume 1* (5th ed). Philadelphia, PA, Saunders, 1975, pp 279-282