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Osteoarthrosis of the Antebrachiocarpal Joint of 7 Riding Horses

By *L-E. Magnusson*[1] and *S. Ekman*[2]

[1]Noläng, Axvall, and [2]Department of Pathology, Faculty of Veterinary Medicine, Swedish University of Agricultural Sciences, Uppsala, Sweden.

Magnusson L-E, Ekman S: Osteoarthrosis of the antebrachiocarpal joint of 7 riding horses. Acta vet. scand. 2001, 42, 429-434. – Osteoarthrosis (OA) of the antebrachiocarpal joint from 7 riding horses is described. The horses were old mares and developed severe OA, with ankylosis in some of the joints. The lesions were bilateral, and the owners noticed the lameness in a late event. The cause of severe OA in these mares is not clear. The fact that OA was bilateral indicates that a single traumatic injury is unlikely as an etiologic factor. Considering the severe joint lesions it took long time before the horse-owners noticed the lameness. It is discussed if the threshold of pain is higher in the antebrachiocarpal joint compared with the middle carpal joint.

Introduction

Lameness originating from the carpal joints is common in the racehorse, where excessive, rapid and repetitive or inappropriate loading and movement are thought to induce joint damage (*Bramlage et al.* 1988). During the protraction phase the carpus is freely movable in an anterioposterior direction. The different joints within the carpal joint do not fit perfectly but slide into position when loaded, with an increased fitting of the articular cartilages at high loads (*Firth & Hartman* 1983). *Johnston et al.* (1997) showed that the carpus of the racehorse is overextended for most of the stance phase. The ability of joint surfaces to dissipate axial force by transfer to the interosseous ligaments is the principal means by which carpal injury is avoided (*Bramlage et al.* 1988). In racehorses, most interest is focused on the middle carpal joint because of a higher rate of injuries compared with the antebrachiocarpal joint (*McIlwraith et al.* 1987, *Palmer* 1986).

The aim of this case report was to point out the severe chronic osteoarthrosis (OA) that can develop in the antebrachiocarpal joint compared to the middle carpal joint, in order to make clinicians aware of this. We wanted to describe the clinical features of 7 riding horses with severe chronic OA of the antebrachiocarpal joints and in some of these also describe the radiological and pathological changes.

Materials and methods

Seven riding horses with bilateral lameness originating from the antebrachiocarpal joints were examined repeatedly (Table 1) during 1993-98 by the first author, in his private practice. A lameness examination with flexion tests was performed together with inspection and palpation of the front legs and documentation of the range of movements. The localisation of the lameness was confirmed by intraarticular anaesthesia. Only 1 horse (no 1) was radiographed. This is a retrospective study where most of the material had been collected and stored prior to designing the study. All horses were mares and the mean age when examined

the first time was 19.5 (13-26) years and the mean age at euthanasia was 22.3 (17-29) years (Table 1).

The following breeds were represented: 3 Swedish warmblood horses, 2 mixed Swedish warmblood horses – Standardbred trotters, 1 Arabian horse and 1 pony of unknown breed.

The carpal joints from 4 of these horses (nos. 1,2,3,5) were collected after euthanasia and stored in a freezer until examination. All 8 joints were opened for a macroscopical examination of the articular cartilage and synovium. Four joints were macerated allowing examination of the osteophyte formations. The other joints from 3 horses were not available for post-mortem examination.

Three of the joints were radiographed and 2 (left carpal joint from horses nos. 1 and 3) were sampled for microscopic examination. The joint for microscopic examination was thawed and then fixed in buffered formalin and 1 cm thick slabs were cut with a bandsaw. These slabs were radiographed and samples from the articular cartilage and subchondral bone of the distal radius and the radial carpal bone were decalcified in formic acid, embedded in paraffin, cut into 6 μm sections and stained with Hematoxylin & Eosin (H&E).

Results

History

Three (nos. 1,2,3) of the horses had, according to the owners, shown a short stride with decreased shoulder action for a long time (years) prior to the first clinical examination. The other 4 were diagnosed at a routine health control at a riding school (nos. 4,5,6,7). All the horses except 1 (no. 3), a brood mare, were used for all-round riding prior to the first noted lameness. The horses had not been treated for carpal lameness prior to the first examination.

Clinical examination

All horses showed bilateral frontleg lameness with pain in flexion of the carpal joints. The lameness was located to the antebrachiocarpal joints by intra-articular anaesthesia. Horses 1, 2 and 3 revealed moderate synovial distension on palpation of the antebrachiocarpal joints at the first clinical examination. Some years later, marginal osteophytes and severe periosteal proliferations were palpated. The lameness increased and joint stiffness changed from mild, moderate to severe. The other horses (nos. 4, 5, 6, and 7) all had moderate to severe synovial effusion, bony enlargements around the antebrachiocarpal joints and joint stiffness of the

Table 1. Data of the 7 mares included in the study.

Horse	Breed	Age (years) at first examination	Number of examinations	Age (years) at euthanasia	Specimen
1	SWH	15	10	18	yes
2	SWH	13	12	17	yes
3	SWH	18	7	22	yes
4	mix	21	2	21	
5	Arab	21	4	23	yes
6	pony	26	3	29	
7	mix	23	3	26	

SWH = Swedish Warmblood Horse; mix = mixed breed.

carpal joints, at the first examination. Horse no 4 was slaughtered soon after the second examination due to severe lameness and nos. 5, 6 and 7 all developed more severe symptoms with increased lameness and joint stiffness.

In the most severe case (no. 1), the joint stiffness only permitted 50 degrees of flexion. For 4 years, this mare was used for breeding, but the signs deteriorated to the point that when the mare rested, she always lay on her side, and euthanasia was strongly suggested by the clinician.

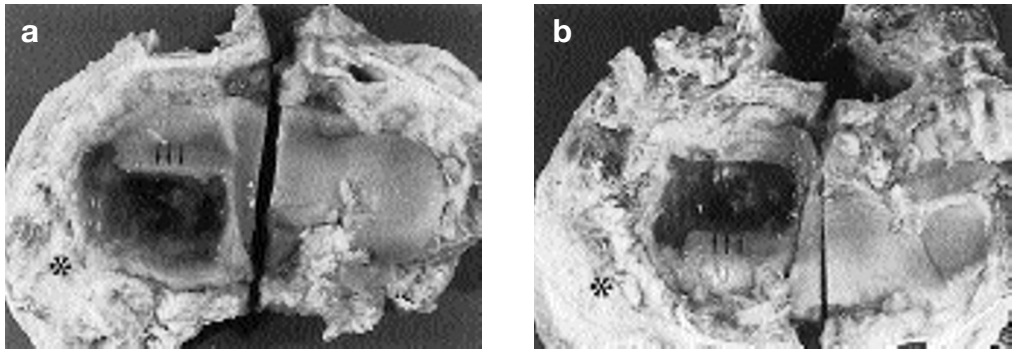
The second mare (no. 2) was treated with hyaluronic acid and corticosteroids in the initial stage with mild synovial effusion and mild bilateral frontleg lameness. In spite of a long period of rest, and being used only for breeding, the horse developed severe osteoarthritis of the antebrachiocarpal joints during a 4-year period. The severe joint stiffness did not allow the mare to rest on the chest in a natural way and euthanasia was elected. These 2 mares (nos. 1,2) had moderately bucked knees and were wide at the knees at the time of euthanasia. A tendency to develop these specific faulty limb confirmations was also found in the other horses. There was no history of broken limb axis prior to the lameness.

Macroscopical and radiological examination

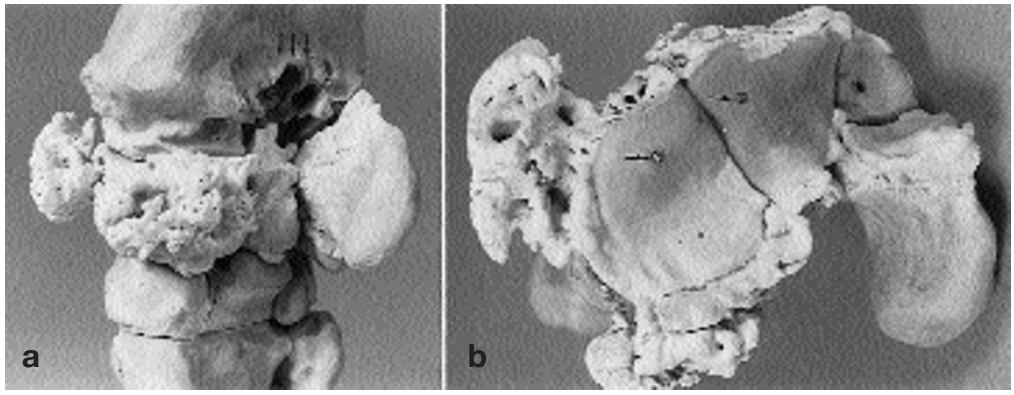
All (nos. 1, 2, 3, and 5) antebrachiocarpal joints that were examined showed severe villi hypertrophy of the synovial membrane and severe fibrosis of the synovial capsule.

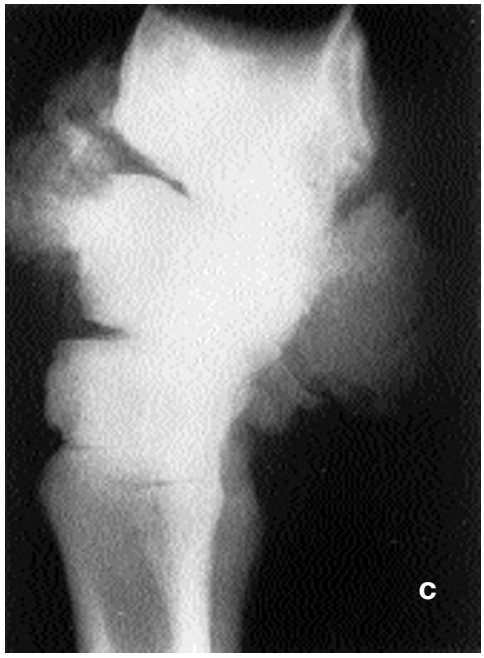
The radial facet of the distal radius and the proximal part of the carpal radial bone presented large areas in the palmar parts with loss of articular cartilage and denuded eburnated bone surfaces (Figs. 1a and b). Areas of articular cartilage fraying were found adjacent to the denuded bone and in the middle carpal joint. The fraying was most severe in the palmar parts of the third, fourth and second carpal bones with "kissing lesions" in the corresponding articular surfaces of the distal parts of the ulnar, intermediate and radial carpal bones. Three mares (nos. 1, 2, 3) also had cartilage ulcerations in the palmar aspect of the third carpal bone with corresponding ulcerations in the third metacarpal bone, with small marginal osteophytes of these bones.

Severe marginal osteophytes were seen in the distal radius, the radial, intermediate, ulnar and accessory carpal bones (Figs. 2a, b and c). The osteophytes were larger in the medial parts of the joints. The more prominent proliferations were found in the radial carpal bones between



Figures 1a and b. Horse no 1. Specimens of a) distal radius and b) the proximal row of the carpal bones from the left carpal joint. Note large areas with loss of articular cartilage in the medial distal radius (arrows) and the proximal radial carpal bone (arrows) and a severe thickening of the joint capsule (*).





Figures 2a, b and c. Horse no. 1. a) Macerated specimen from right carpus, with severe periostal bony proliferations of the radial and accessory carpal bones. A severe bony destruction is also present in the distal palmar region of radius (arrows). b) Macerated specimen of the proximal row of carpal bones (right carpus) with periosteal proliferations of the radial, intermediate, ulnar and accessory bones (the bones are fixed with central nails (see arrow). c) Radiograph of the left carpal bones, post-mortem. The severe bony proliferations are obvious.

the tendons of radial carpal extensor and the oblique carpal extensor muscles and behind the medial collateral ligament. The bone proliferations were extending over the joint cavity creating a fusion in some areas between the distal radius and the radial carpal bones. This ankylosis explained the severe joint stiffness recorded on clinical examination. An area of bone destruction was also evident in the distal palmar regions of radius (Fig. 2a) outlined by the insertions of 2 ligaments connecting the radial carpal bone and distal radius. Subchondral bone sclerosis was present in the radiographed slabs of the distal radius and in the proximal area of the radial carpal bones of horses nos. 1 and 3.

Histological examination

On histologic examination of the left distal radius and the proximal radial carpal bones from 2 horses (nos. 1, 3), the articular cartilage fraying, ulcerations and loss of cartilage were verified. The subchondral bone showed thick trabeculae with lamellar bone (bone sclerosis). Areas of woven bone outlined some of the trabeculae, where also osteoblast and osteoclast activity was increased and an intertrabecular fi-

brosis was present. The synovial membrane of the proximal carpal joint of these horses was characterised by villi proliferations, marked proliferation of synoviocytes and mild to moderate subsynovial accumulations, mostly perivascular, of lymphocytes.

Discussion

This paper describes a severe chronic osteoarthrosis (OA) of the antebrachiocarpal joints from 7 mares.

Considering the severe joint lesions, it took a surprisingly long time before the horse-owners noticed the lameness. This can be explained by the bilateral lameness present prior to the lameness being dominant in 1 leg. The first sign of injury in the antebrachiocarpal joints of the horses in this study was a short stride with decreased shoulder action. A clinical examination revealed bilateral lameness with marked reaction after flexion and marked synovial distension. When a frontleg lameness is present in racehorses or riding horses, it usually originates from the middle carpal joint even if the radiological lesions are more severe in the antebrachiocarpal joint (Ingela Liwång, personal communication). Hence, it has been suggested that the threshold of pain is higher in the antebrachiocarpal joint compared to the middle carpal joint.

The destruction of articular cartilage, subchondral bone sclerosis, marginal osteophyte formation with large periosteal bony proliferation and subsequent anchylosis resulted in pain and joint dysfunction. The most striking signs were loss of normal joint motion and a severe pain at flexion. Surprisingly, all the horses in this study were mares. This is perhaps a random effect, but it is also more acceptable to keep a lame mare for breeding.

Repeated micro trauma and/or an impact load to the joint tissues can be the central etiologic concept in the degenerative changes in these joints. The initial cartilage lesions are often disruption of the superficial zone with fraying, erosion and loss of cartilage. The progression of the cartilage lesions is linked together with the subchondral bone sclerosis creating a stiff inelastic tissue (Norrdin et al. 1998). The degenerative debris will cause low-grade synovitis, which will contribute to the progression of the OA (*Palmer & Bertone* 1994). OA in the antebrachiocarpal joints of horses can also develop due to trauma (*Bertone et al.* 1989), subchondral bonecysts (*Specht et al.* 1988) and chip fractures (*McIlwraith et al.* 1987).

In the present material, the OA was bilateral and the destruction of the articular cartilage was most severe at the most weight-bearing parts in the antebrachiocarpal joint. Together with the lack of a history of predisposing disorders such as septic arthritis, articular fractures or subchondral bonecysts, this suggests a repetitive joint trauma in a high-load-high-motion joint as a cause of the OA. The fact that the OA was bilateral in all 7 mares indicates that a single traumatic injury is unlikely as an etiologic factor. However, a previous subchondral bonecyst may be a factor in the pathogenesis of the OA of the antebrachiocarpal joints. All the horses had been used for all-round riding performance in riding schools and none had been competing in jumping or dressage.

The cause of the severe OA of the antebrachiocarpal joints in these old mares is not clear. None of the owners did report lameness from the 7 mares early in the disease, which may suggest that the antebrachiocarpal joint have a high threshold for pain, which must be important to consider in equine clinical practise.

Acknowledgements

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Sammanfattning

Osteoarthros i övre karpalleden från sju ridhästar.

Sju äldre ridston med en svårartad, bilateral osteoarthros i radiokarpalleden har undersökts kliniskt upprepade gånger under åren 93-98. Symtomen dominerades av en tilltagande ledstyvhet, smärta vid sammanböjning, konturstörning framtill runt leden samt hälta. Trots vila förvärrades ledbesvären och samtliga hästar avlivades efter en tid. Vid makroskopisk och radiologisk undersökning postmortalt påvisades förlust av ledbrosk distalt på radius och proximalt på radiokarpalbenet, ledrandsosteofyter, subkondral benscleros samt förtjockad ledkapsel med villiproliferationer. I samtliga fall har det dröjt länge innan ägarna noterat hälta. Detta kan tala för att smärttröskeln i radiokarpalleden är högre jämfört med den i interkrapallederna. Orsaken till ostearthrosen i dessa fall har ej kunnat fastställas och uppgifter om predisponerande faktorer saknas.

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Stina Ekman, Department of Pathology, Faculty of Veterinary Medicine, Swedish University of Agricultural Sciences, Box 7028, S-750 07 Uppsala, Sweden. E-mail: Stina.Ekman@pat.slu.se, fax: 46-18-67 35 32.