EQUINE

Osteochondral fragments involving the dorsomedial aspect of the proximal interphalangeal joint in young horses: 6 cases (1997–2006)

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Objective—To determine clinical and diagnostic imaging findings in young horses with osteochondral fragments involving the dorsomedial aspect of the proximal interphalangeal (PIP) joint.

Design—Retrospective case series.

Animals—6 horses.

Procedures—Medical records were reviewed. Follow-up information was obtained through telephone conversations with owners or trainers or by examining race records.

Results—Horses were between 1 and 4 years old. Three had bilateral osteochondral fragments in the forelimbs (n = 2 horses) or hind limbs (1). Radiographically, all but 1 fragment seemed to originate from the dorsomedial aspect of the distal end of the first phalanx. Fragment size ranged from 6 × 9 mm to 11 × 21 mm. Three horses had lameness referable to the region of the affected joint; the other 3 horses did not have clinical signs referable to affected PIP joints. Two horses were euthanized shortly after diagnosis at the owners' request because of concerns that the horses would be unsuited for their intended athletic use. Two of the 3 horses in which fragments were incidental findings were able to race successfully, although 1 received intra-articular corticosteroid treatments; the third was retired because of unrelated orthopedic problems.

Conclusions and Clinical Relevance—Results suggested that osteochondral fragments involving the dorsomedial aspect of the PIP joint may be an incidental finding in young horses. Given the absence of clinical signs in 5 of 9 affected joints and the fact that 3 of 6 horses were affected bilaterally, a developmental origin of the fragments was suspected. (*J Am Vet Med Assoc* 2007;230:1498–1501)

Osteochondral fragments are infrequently encountered in the PIP joint in horses, and to our knowledge, only a few cases have been described in the literature.[1-3] Previously reported locations for such fragments include the dorsoproximal margin of the middle phalanx,[3] the palmar midsagittal aspect of the middle phalanx,[1] and the palmaromedial aspect of the middle phalanx.[2] Palmar fragments may represent partial avulsion fractures of the fibrous connective tissue attachments involving the PIP joint,[4] whereas dorsal fragments have been suggested to be developmental in origin.[3]

To our knowledge, osteochondral fragments involving the dorsomedial aspect of the PIP joint in young horses have not been described previously. The purpose of the study reported here was to determine clinical and diagnostic imaging findings in young horses with osteochondral fragments involving the dorsomedial aspect of the PIP joint.

Criteria for Selection of Cases

Medical records of horses examined at the Equine Teaching Hospital of the Norwegian School of Veterinary Science between January 1997 and January 2006 that had radiographic evidence of osteochondral fragments involving the PIP joint were reviewed. Horses with intra-articular fragments involving the dorsomedial aspect of the joint were included.

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Abbreviations

PIP	Proximal interphalangeal
CT	Computed tomography
OA	Osteoarthritis

Procedures

Information obtained from the medical records of cases included in the study consisted of clinical signs, diagnostic imaging findings, fragment size (estimated or measured), treatment, and outcome. Long-term follow-up information was obtained through telephone interviews with owners or trainers or through examination of race records from the national racing associations.

Because 2 of the horses identified in the initial search of medical records were determined to be related, radiographic screening of the pastern region was performed on all available offspring of the sire of these horses over a 6-year period.

Results

Six horses met the criteria for inclusion in the study. All were between 1 and 4 years old at the time the diag-

nosis was made. There were 4 Norwegian Coldblooded Trotters (a popular local harness racehorse breed), 1 Standardbred, and 1 Thoroughbred. There were 3 sexually intact males, 2 sexually intact females, and 1 gelding. All 6 horses were intended for racing, although 3 (horses 1, 2, and 4) were not yet in race training at the time of initial examination. The remaining 3 horses (horses 3, 5, and 6) were in race training at the time of initial examination. None of the horses had any grossly evident conformational abnormalities of the forelimbs or hind limbs.

All horses were admitted to the hospital because of lameness (horses 1, 2, 3, 4, and 6) or a history of lameness (horse 5). In 3 horses (horses 1, 2, and 4), the underlying cause of the lameness was localized to the pastern region on the basis of results of medial abaxial sesamoid perineural anesthesia (2 mL of 2% mepivacaine hydrochloride; cases 1 and 4) or anesthesia of the PIP joint (5 mL of 2% mepivacaine hydrochloride; cases 2 and 4). These 3 horses also had palpable abnormalities of the affected limbs at the time of initial examination, including thickening of the pastern region and joint effusion. Three horses (cases 2, 4, and 5) were affected bilaterally, but 2 of these horses (cases 2 and 4) had only unilateral lameness and the third (case 5) was not lame at the time of initial examination. One horse (case 3) was referred for treatment of an apical proximal sesamoid bone fracture of the right forelimb; mild (grade 1 of 5) right hind limb lameness was also identified, and a fragment was identified in the right hind PIP joint radiographically. Two horses (cases 5 and 6) did not have any clinical signs referable to the affected joints at the time of initial examination. In one of these horses (case 5), long plantar ligament desmitis was diagnosed on the basis of results of palpation and ultrasonography, and bilateral hind limb PIP joint fragments were identified radiographically. The other horse (case 6) had subtle right forelimb lameness that was alleviated by anesthesia of the middle carpal joint (10 mL of 2% mepivacaine hydrochloride); third carpal bone sclerosis was identified radiographically, along with 2 PIP joint fragments. None of the 6 horses had any physical or historical evidence of acute trauma to the affected limbs.

Radiography of the pastern region was performed in all 6 horses, and CT was performed in 2 (cases 5 and 6). In all but 1 horse (case 1), radiographs of both forelimbs (cases 3, 4, and 6) or both hind limbs (cases 2 and 5) were obtained. In 1 horse (case 1), radiographs were obtained only of the affected right forelimb. Radiographic projections that were obtained included lateromedial, dorsopalmar or dorsoplantar, dorsomedial-palmarolateral or dorsomedial-plantarolateral oblique (made 45° lateral to the dorsopalmar or dorsoplantar line), and dorsolateral-palmaromedial or dorsolateral-plantaromedial oblique (made 45° lateral to the dorsopalmar or dorsoplantar line) projections. Osteochondral fragments originating from the dorsomedial aspect of the distal end of the first phalanx were identified in 9 joints (6 forelimb and 3 hind limb joints). Fragments were best visualized on the dorsopalmar or dorsoplantar projection and on the dorsolateral-palmaromedial or dorsolateral-plantaromedial oblique projection. Displaced fragments (5 joints) were also clearly visible on the lateromedial projection.

Radiographically, all fragments appeared rounded without any evidence of reactive bone proliferation or subchondral bone sclerosis involving the fragment bed. In 8 of the 9 joints, the fragments appeared to originate from the region of the dorsal aspect of the medial condyle of the proximal phalanx (**Figures 1 and 2**) and involved various portions of the dorsal articular surface of the distal end of the bone. Fragment location was verified in 2 joints (cases 5 and 6) by use of CT and in 4 joints (cases 2 and 4) by means of postmortem examination. In 1 joint (case 5; left hind limb), the fragment originated from the dorsomedial aspect of the proximal end of the middle phalanx, as determined by means of radiography and CT. This fragment involved part of the

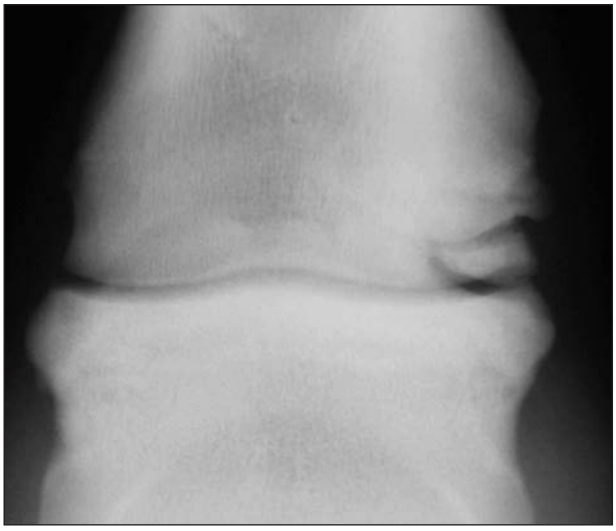

Figure 1—Dorsopalmar radiographic projection of the left forelimb in a horse. Notice the large osteochondral fragment originating from the dorsomedial aspect of the distal end of the proximal phalanx.

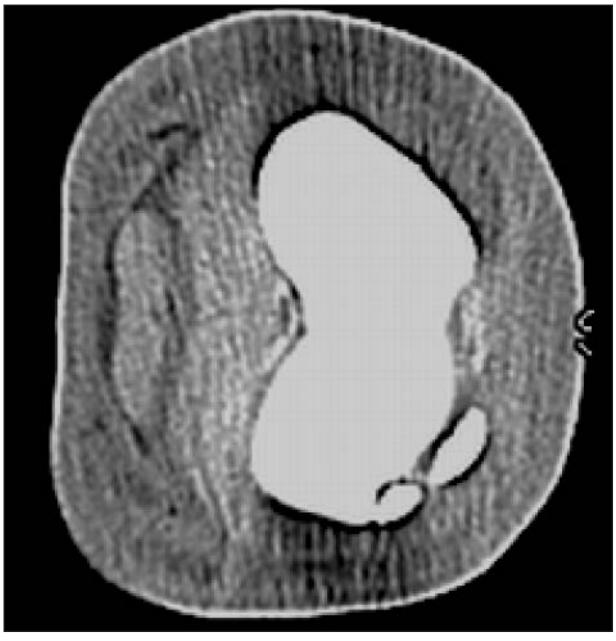

Figure 2—Transverse CT image of the distal end of the proximal phalanx in a horse. Notice the 2 osteochondral fragments originating from the dorsomedial aspect of the proximal phalanx.

joint surface. Mild to moderate OA was identified radiographically in all horses and by means of CT in 2 horses (cases 5 and 6). Marginal osteophytosis, enthesiophytosis, and periarticular periosteal reaction were present medially in 8 of the 9 joints at the time of initial examination.

Fragment size was measured at the time of surgery (case 2) or necropsy (cases 2 and 4) or was estimated on the basis of radiographic (cases 1 and 3) or CT (cases 5 and 6) appearance. Fragments ranged from 6 × 9 mm to 11 × 21 mm.

In 1 horse (case 2), the fragment was surgically removed from the lame limb, but was not removed from the limb without clinical evidence of lameness; the excised fragment measured 15 × 11 mm. This horse had evidence of OA at the time of surgery that continued to progress over the next 5 months despite fragment removal. Seven months after surgery, the horse developed clinical evidence of cervical vertebral malformation and was euthanized. In 2 horses (cases 3 and 5), treatment consisted of intra-articular administration of betamethasone (12 mg) in the affected joints. One of these horses (case 3) did not have any further lameness involving the affected limb, whereas the other (case 5) never had any clinical signs referable to the PIP joint fragments and was still racing successfully 5 years later. This horse was evaluated for unrelated lameness several times after the initial examination but did not have clinical signs related to the PIP joint fragments or progression of radiographic signs of OA in the affected joints. Two horses (cases 1 and 4) were euthanized shortly after diagnosis at the owners' request because of concerns that the horses were unsound for their intended athletic use. The remaining horse (case 6) was treated for unrelated lameness in the affected limb (intra-articular medication of the middle carpal joint) and was in active race training 8 months after the initial examination without any signs of lameness referable to the PIP joint in the affected limb.

A necropsy was performed on 2 horses (cases 2 and 4) that were euthanized. Histologic examination of the fragments revealed morphologically normal trabecular bone covered with articular cartilage; the fragments were attached to parent bone by fibrous tissue. Necropsy also confirmed the OA changes in the joints that had been identified radiographically and cervical vertebral malformation in 1 horse (case 2). At the owner's request, a necropsy was not performed on the remaining horse (case 1) that was euthanized.

Forty-seven progeny of the sire of 2 horses in the present report (cases 1 and 2) were identified, of which 15 were available for radiographic examination. None of these 15 horses had radiographic evidence of osteochondral fragments involving the PIP joints.

Discussion

The PIP joint is a high-load, limited-motion joint with strong periarticular soft tissue structures that provide stability and prevent hyperextension.[4] Thus, traumatic chip fractures of the PIP joint are rare and, when identified, most commonly affect the palmar aspect of the middle phalanx.[5] Trauma cannot be excluded as the initial cause of the fragments identified in the horses described in the present report. However, none of the horses had any history of acute lameness or an acute traumatic incidence involving the affected limbs, nor was there any radiographic evidence of callus formation or reactive bone proliferation involving the fragment bed.

Osteochondral fragments of developmental origin have been extensively described in the veterinary literature.[6] In horses, such fragments are most commonly seen in the tibiotarsal and femoropatellar joints,[7] whereas only a few cases of developmental lesions involving the PIP joint have been reported. Schneider et al[3] described small osteochondral fragments originating from the dorsoproximal aspect of the middle phalanx in 3 horses and suggested that these fragments were of developmental origin. Nixon[8] stated that osteochondrosis of the PIP joint with intra-articular fragmentation does occasionally occur, although subchondral cystic lesions involving the distal articular surface of the proximal phalanx is the more common presentation of the disease. Subchondral radiolucencies of the distal end of the proximal phalanx with concurrent severe OA were described in 6 horses by Trotter et al[9] and classified as osteochondrosis. Pool[10] later interpreted these findings as juvenile arthrosis. Subchondral lucencies were not identified in any of the horses described in the present report, and none of these horses had the severe clinical or radiographic abnormalities typically associated with juvenile arthrosis.

Osteochondrosis is a multifactorial disorder of epiphyseal cartilage, with genetic factors, dietary factors, conformation, trauma, and exercise playing important roles in the clinical appearance of the disease.[11] Lameness associated with osteochondrosis is often not seen until a horse is being trained at 2 to 3 years of age.[12] There is considerable confusion in the literature about the etiology of osteochondrosis, possibly because most studies of osteochondrosis have involved horses in the chronic stage of the disease, when lesions were morphologically complicated and the initial causative insult was impossible to determine.[11] Vascular cartilage canals in the epiphyseal cartilage have been shown to have an important role in the pathogenesis of the disease, with lesions in these areas believed to occur secondary to a defect in vascular supply.[13] These vascular cartilage canals are no longer present at the usual predilection sites for osteochondrosis after 7 months of age,[13] and classification of lesions in older animals (ie, after 7 months of age) may be challenging because of the chronicity of lesions. Therefore, the value of histologic findings for horses in the present report is questionable. However, the histologic appearance of these fragments resembled findings described by Grøndahl and Dolvik[14] in connection with fragmentation of the distal intermediate ridge of the tibia and the appearance of fragments of the lateral trochlear ridge of the femur in horses > 1 year old described by Rejnö and Strömberg.[12]

Fragmentation of the distal intermediate ridge of the tibia has a strong genetic correlation in several breeds, with estimates of heritability ranging from 0.26 to 0.52.[14,15] The number of cases in the present study was far too small for any analysis of heritability, and the

fact that 2 horses were related may be purely coincidental. However, it was interesting that the Norwegian Coldblooded Trotters were all affected in the forelimbs, whereas the Standardbred and the Thoroughbred were affected in the hind limbs. If the lesions truly are developmental, a genetic influence seems logical and further investigation, preferably in younger animals, is warranted. However, such studies may be difficult because of the uncommon occurrence of these fragments.

Osteoarthritis is characterized as degradation of the articular cartilage, often accompanied by periarticular new bone formation at the joint margins, synovitis, capsulitis, and subchondral bone sclerosis.[16] Osteoarthritis of the PIP joint is a common finding in horses with lameness referable to the pastern region[5] and may arise from a range of insults, such as conformational defects, trauma, repeated stress, and septic arthritis.[17] A base-wide and toe-in or toe-out stance is considered to predispose horses to OA of the medial aspect of the PIP joint as a result of abnormal biomechanical forces exerted on this region.[5] Mild to moderate periarticular new bone formation was seen in 8 of the 9 affected joints in the present report. However, although signs of OA were more pronounced medially, conformational abnormalities were not identified. Nixon[8] states that development of OA in the PIP joint may be relatively slow, even with large intra-articular osteochondral fragments. This corresponds with our findings for 2 of the cases in the present report, in which long-term follow-up failed to identify any substantial progression of OA in affected joints. Fragments in these 2 cases were comparable in size to fragments in the other cases; however, these fragments may have been more stable than in the other cases.

Techniques for arthroscopic surgery of the dorsal and palmar or plantar pouches of the PIP joint have been described.[18] Owing to the size and abaxial location of the fragments in the horses described in the present report, arthroscopic fragment removal as described by Schneider et al[3] was not considered feasible. In 1 horse, the fragment was removed by means of an arthrotomy; however, this horse continued to be lame after surgery owing to progression of OA. Surgical removal of fragments was not considered appropriate for horses without evidence of lameness referable to the pastern region; however, in some of these horses, the joints were treated by means of intra-articular corticosteroid administration because of the presence of radiographic signs of OA. Arthrodesis of the PIP joint[19,20] may be considered in horses with lameness and advanced OA changes, as this procedure has been shown to potentially relieve lameness, but was not attempted in any of the horses described in the present report because of a lack of clinical signs (3 cases) or financial constraints (3 cases).

The absence of clinical signs for 5 of the 9 affected joints described in the present report, the fact that 3 of the 6 horses were affected bilaterally, the lack of any history of acute trauma, and the fact that all 6 horses were young suggest that the osteochondral fragments that were identified may have been of developmental origin. This coincides with the conclusion drawn by Schneider et al,[3] who suggested that osteochondral fragments located at the dorsoproximal margin of the middle phalanx were manifestations of osteochondrosis. However, as pointed out by Pool[10] and by Ekman and Carlson,[11] it is not possible to determine on the basis of gross, radiographic, or histologic appearance whether osteochondral fragments are of developmental origin or a result of a traumatic incident. Further studies are warranted to elucidate the nature of these fragments.

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