Veterinary Surgery, 22, 6, 515-523, 1993

Progression of Femoropatellar Osteochondrosis in Nine Young Horses

Clinical, Radiographic and Arthroscopic Findings

ROBIN M. DABAREINER, DVM, MS, KENNETH E. SULLINS, DVM, MS Diplomate ACVS, and NATHANIEL A. WHITE, II, DVM, MS, Diplomate ACVS

The clinical and radiographic progression, and arthroscopic findings for nine young horses (<1 year of age) with femoropatellar osteochondrosis (OCD) are presented. Horses had a 2 to 12 week history of bilateral (8 horses) or unilateral (1 horse) hindlimb lameness. The most consistent clinical signs included femoropatellar joint distention and bilateral hindlimb lameness. At the onset of clinical signs, radiographic lesions were not present (4 horses) or subtle (5 horses), but were easily identified on radiographs taken 4 to 24 weeks later. Arthroscopic surgery was delayed until radiographic changes became obvious. Surgical findings in 20 femoropatellar joints were most commonly osteochondral "flaps" located on the proximal lateral trochlear ridge of the femur and were larger than had been indicated by the radiographs. Eight horses were being used for their intended purpose, which was racing (3 horses were racing and 3 were in race training), dressage (1 horse) or pleasure riding (1 horse). One horse required a second surgery when similar lesions developed in the opposite stifle, and was euthanatized 2 months later because of persistent lameness. Once clinical signs are observed, osteochondrosis lesions of the distal femur can progress in foals younger than 9 months of age and the full extent of the radiographic lesion may take several weeks to develop.

CLINICAL AND RADIOGRAPHIC findings, and surgical management of equine femoropatellar osteochondrosis (OCD) have been reported.[1-8] The diagnosis of OCD has been based upon the presence of lameness or femoropatellar joint effusion, or both clinical signs, with radiographic lesions located most commonly on the lateral trochlear ridge (LTR) of the distal femur.*[6] Surgical intervention has been recommended to remove the diseased osteochondral tissue and to allow healing of the defect,[5] with 80% of horses becoming sound.*[4,6]

Some evidence suggests that femoropatellar OCD lesions can progress in young horses. In a study of 10 horses with femoropatellar OCD, two 8-month-old foals had surgery for unilateral LTR lesions but subsequently developed femoropatellar effusion and lameness in the contralateral limb; OCD lesions of the LTR were found in the untreated limbs of both foals at necropsy.[4] McIlwraith reported performing a second surgical procedure in one horse with femoropatellar OCD because of persistent effusion and lameness 5 months after the initial surgical procedure.[6] Osteochondral fragmentation of the distal aspect of the LTR was not present on radiographs taken before or after the first surgery but was evident on radiographs taken before the second surgical procedure.[6] These findings differ from an earlier study which reported gradual resolution of the LTR subchondral defect and disappearance of joint mice on subsequent radiographic examinations of horses with femoropatellar OCD.[2]

We report the radiographic progression of femoropatellar OCD in nine horses that were <1 year of age at the onset of clinical signs and describe the progression of clinical signs and the results of surgical treatment.

Case Histories

The nine horses described in this report had lameness and femoropatellar joint effusion, and were admitted to

* Foland JW, McIlwraith CW, Trotter GW. Equine femoropatellar osteochondritis dissecans: results of arthroscopic surgery in 153 horses. Vet Surg 1991; 19:335.

From the Marion duPont Scott Equine Medical Center, Virginia—Maryland Regional College of Veterinary Medicine, Leesburg, Virginia.
Reprints not available

the Marion duPont Scott Equine Medical Center between January 1985 and April 1991. Lameness was graded on a scale of 1 to 5.† Radiographic studies included lateral to medial (LM) and caudal to cranial (CaCr) projections of the stifle in all horses. Xeroradiographs were obtained at admission in four horses (Horses 4, 5, 8, 9) and a caudolateral to craniomedial oblique (CaL-CrM) projection was obtained in three horses (Horses 1, 4, 9) on clinician request.

Historically, all horses had stifle lameness or femoropatellar joint effusion, or both clinical signs, at about 6 months of age and had been radiographed at the onset of clinical signs either by the referring veterinarian (Horses 1, 3, 5, 6) or at our hospital (Horses 2, 4, 7, 8, 9). All horses admitted to the Equine Medical Center within 1 to 2 months of their initial examination because of persistent clinical signs, were radiographed again, and had arthroscopic surgery of the affected femoropatellar joints.

After sedation with xylazine (0.5 mg/kg, intravenously [IV]) and guaifenesin (5%, IV to effect), general anesthesia was induced with ketamine (2.2 mg/kg, IV) and maintained using halothane and oxygen in a semi-closed system. Standard femoropatellar insertion of the arthroscope and instrumentation was used.[6] Phenylbutazone (2.2 mg/kg, orally, every 12 hours) was administered for 3 to 5 days after surgery, and perioperative penicillin (20,000 U/kg, intramuscularly, every 12 hours) was administered based on the surgeon's preference. Horses were confined to a stall for 2 months with daily handwalking beginning 2 weeks after surgery, and then pasture turnout was allowed in all horses except Horse 5. Follow-up information was obtained between 8 and 24 months after surgery by telephone conversation with the owner.

Results

Clinical and Radiographic Findings

Six Thoroughbreds, one Standardbred, one Quarter Horse, and one Warmblood-Thoroughbred cross comprised the group of seven males and two females (Table 1). The mean age of onset of clinical signs was 6 months (range, 4–9 months). The mean age on admission to the hospital was 6.5 months (range, 5–12 months). All horses had a history of bilateral hindlimb lameness of 2 to 12 weeks duration, except Horse 5 that had a unilateral lameness. Lameness was characterized by a shortened cranial phase to the stride and asymmetry of hip movement at the trot, or bilateral hindlimb stiffness with one limb affected more severely. The clinical signs on admission consisted of bilateral femoropatellar joint effusion in all but one horse (Horse 5), and hindlimb lameness that ranged from none to Grade 2 of 5 (Table 1). Horses had either subtle (5 horses) or no (4 horses) radiographic changes at the onset of clinical signs. Prominent subchondral bone lesions became evident in the lateral trochlear ridge (LTR) on radiographs taken 4 to 24 weeks after the initial radiographic examination.

At 9 months of age, Horse 1, a male Thoroughbred, had Grade 2 bilateral hindlimb lameness that was more severe in the left limb, and marked bilateral femoropatellar joint effusion which had been present for 8 weeks. Historically, the hindlimb lameness appeared 6 weeks before the femoropatellar joint effusion. No radiographic lesions were evident at the onset of clinical signs or 2 weeks later (Fig. 1A). However, after 6 weeks, bilateral LTR subchondral bone lucency and fragmentation were present and a lucent defect was apparent in the medial trochlear ridge (MTR) of both femurs (Fig. 1B and 1C).

At 6 months of age, Horse 2, a female Thoroughbred, had Grade 1 bilateral hindlimb lameness which had been present for 6 weeks. Femoropatellar joint effusion was not present and a subtle lucency was evident on the proximal aspect of the LTR of the left femur (Fig. 2A). No lesions were identified on radiographs of the right stifle. Four weeks later, the lameness was unchanged but bilateral femoropatellar effusion was present. Bilateral LTR subchondral irregularity and lucency with distal osseous fragmentation (Fig. 2B and 2C) was evident at this time.

Horse 3, a 12-month-old male Quarter Horse, had bilateral lameness of 12 weeks duration, which was more severe in the left limb. No abnormalities were identified on radiographs taken at the onset of clinical signs. On admission, the horse was Grade 2 lame in both hindlimbs with bilateral femoropatellar effusion. Subchondral bone lucency and concavities of both LTR's were identified.

At 5 months of age, Horse 4, a male Thoroughbred, had Grade 1 bilateral hindlimb lameness and femoropatellar effusion of 3 weeks duration. No lesions were identified on radiographs or xeroradiographs. Twelve weeks later, the bilateral hindlimb lameness was Grade 2 of 5 and more evident in the left hindlimb. There was a subchondral bone concavity and lucency of the proximal LTR of the left femur and flattening of the proximal aspect of the right LTR.

At 6 months of age, Horse 5, a female Thoroughbred, had Grade 1 right hindlimb lameness and femoropatellar effusion of 4 weeks duration. There was subchondral bone lysis of the proximal aspect of the LTR of the right femur. The left stifle had no clinical signs or radiographic lesions. Twelve weeks later, after right femoropatellar arthroscopic surgery, the horse had a Grade 2 bilateral hindlimb lameness and femoropatellar effusion, and there was subchondral lucency and fragmentation of the left LTR and MTR.

† Definition and classification of lameness. In: *Guide for Veterinary Service and Judging of Equestrian Events.* American Association of Equine Practioners. 3rd ed. 1984:24.

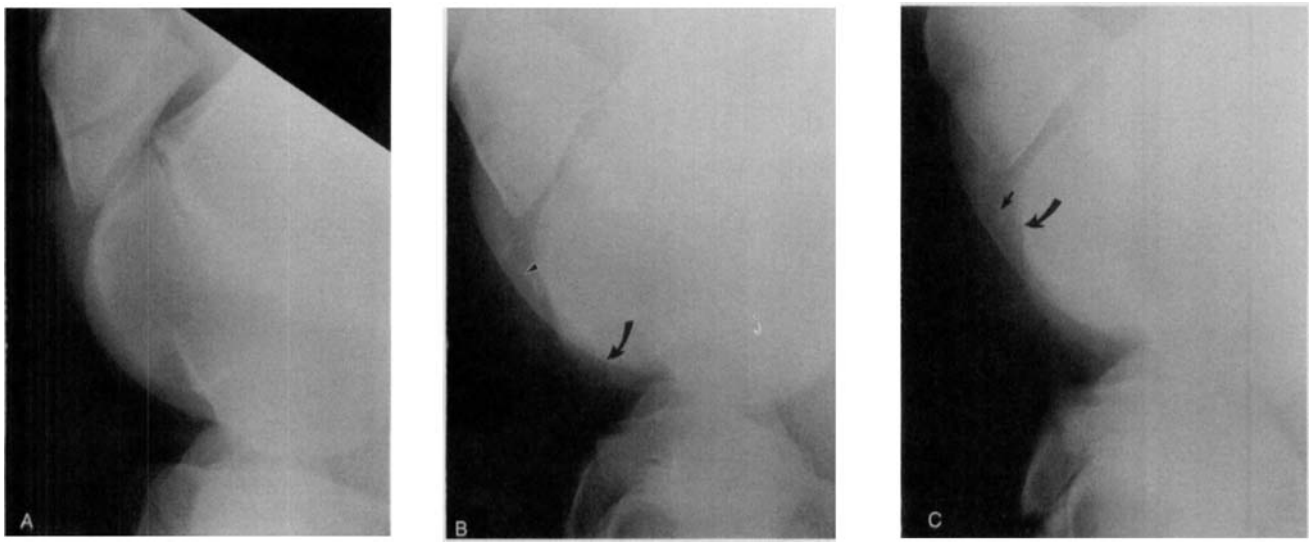

Fig. 1. Horse 1. Lateromedial projections. (A) Normal left stifle at 7.5 months of age. (B) At 9 months, generalized subchondral lucency of the LTR with osseous fragmentation distally (curved arrow) and subchondral lucency involving the MTR (arrowhead). (C) Right stifle. Flattening and fragmentation of the LTR (curved arrow) and a large subchondral lucency involving the MTR (arrowhead).

An irregular contour to the proximal aspect of the LTR with distal osseous fragmentation was observed on the right femur.

Horse 6, a 10-month-old male Thoroughbred, had Grade 2 left hindlimb lameness and bilateral femoropatellar effusion of 8 weeks duration. Abnormalities were not identified on bilateral stifle radiographs taken at the onset of clinical signs. Eight weeks later, bilateral LTR subchondral lucency and fragmentation were evident.

At 12 months of age, Horse 7, a male Thoroughbred, had a 12-week history of shifting hindlimb lameness and no femoropatellar effusion. On admission, the horse was sound but had right femoropatellar effusion. No abnormalities were noted on radiographs of the left stifle but there was fragmentation of the proximal aspect, and subchondral lucency of the distal aspect of the right LTR. At the owners request, the horse was treated conservatively by stall rest. At 18 months of age, the horse was examined again for progressive lameness and femoropatellar effusion. Clinically, the horse had a Grade 2 bilateral hindlimb lameness with marked bilateral femoropatellar joint effusion. Subchondral bone lucency was identified on both LTR's with osseous fragmentation of the proximal aspect of the right LTR.

Horse 8, a 6-month-old male Thoroughbred, had bilateral femoropatellar effusion of 4 weeks duration but

Fig. 2. Horse 2. Lateromedial projections. (A) Lucent defect (curved arrow) proximal left LTR at 6 months of age. (B) Defect (curved arrow) is larger at 7 months. (C) Right stifle. A lucent defect (curved arrow) is identified in the proximal LTR and there is osseous fragmentation (large arrow) distally.

TABLE 1. *Age, Radiographic and Surgical Findings, and Outcome of Nine Young Horses with Femoropatellar Osteochondrosis*

Horse	Age (months)			Radiographic Findings at Admission for Surgery	Surgical Findings	Outcome
	Admission for Surgery	Previous Radiographic Examinations	Lesion Identified			
1	9	7, 7.5	9	L—lucent defect and fragmentation involving proximal LTR	L—4 cm osteochondral fragment on proximal LTR	2 year old in training
				R—flattening proximal LTR with 1 cm osseous flap; subchondral lucency proximal MTR.	R—2 × 2 cm cartilage flap proximal LTR, 2 × 2 cm subchondral defect in MTR	
2	7	6	6 (L)	L—2.5 × 2 cm lucent defect proximal LTR	L—2 × 4 cm osteochondral fragment proximal LTR	Racing
				R—3 × 1 cm proximal LTR lucency with distal osteochondral fragment	R—3 × 1 cm cartilage flap with undermined subchondral bone proximal LTR, small distal LTR fragment	
3	12	9	12	L—Subchondral bone lucency proximal LTR	L—3 × 3 cm undermined defect on proximal LTR, fibrilled MTR cartilage	Pleasure
				R—concavity and disruption proximal LTR	R—2 × 2 cm osteochondral fragment proximal LTR, subchondral bone defect	
4	8	5	8	L—concavity and subchondrial bone lucency proximal LTR	L—2 × 4 cm osteochondral fragment proximal LTR, 1 cm cartilage erosion distal patella	2 year old in training
				R—flattening proximal LTR	R—1 × 2 cm cartilage flap proximal LTR	
5	6 (R)	6	6 (R) 9 (L)	R—(6 mo.) subchondral lysis and disruption of proximal LTR	R—(6 mo.) 2 cm dimple of cartilage proximal LTR with soft subchondral bone underneath	Euthanatized
				L—concavity and osseous fragmentation proximal LTR; irregular border to MTR	L—1.2 cm cartilage flap on LTR; 2 × 3 cm cartilage flap over proximal MTR	
				R—(9 mo.) disruption of contour of proximal LTR; 2 distal LTR fragments	R—(9 mo.) lesions from previous surgery were covered with fibrous-looking repair tissue; 2 osteochondral fragments distally.	

6	10	8	10	L—disrupted border proximal LTR, flattened distal LTR	L—2 × 5 cm cartilage flap subchondral bone defect on proximal LTR	Racing
				R—concavity and osseous fragment on proximal LTR	R—3 cm osteochondral fragment and subchondral bone defect on proximal LTR	
7	18	12	12 (R) 18 (L)	R—(12 mo.) subchondral lucency distal LTR; 2 cm linear osteophyte proximal LTR		Race training
				L—concavity and lucency proximal LTR	L—1.5 cm of fibrillated cartilage with subchondral defect on proximal LTR.	
				R—(18 mo.) subchondral lucency and fragmentation proximal LTR; subchondral lucency distal LTR	R—2.5 cm osteochondral fragment on proximal LTR; 2 cm cartilage defect over subchondral defect on distal LTR.	
8	7	6, 7	6, 11	L—(6 mo.) 2 mm lucent defect in proximal LTR		Racing
				R—(6 mo.) subtle lucent defect and flattening proximal LTR, 1 cm medial condylar bone cyst.		
				L—(7 mo.) 5 mm defect proximal LTR	L—Loose cartilage proximal LTR	
				R—(7 mo.) 8 mm irregularity proximal LTR; 1 cm medial condylar bone cyst	R—Loose cartilage proximal LTR	
				L—(11 mo.) no change from post-op films.		
				R—(11 mo.) 1.5 cm × 5 mm subchondral cyst LTR, 1.5 cm medial condylar bone cyst	R—1.5 cm subchondral lysis proximal LTR, medial subcondylar bone cyst	
9	9	8	9	L—2 × 3 cm lucency proximal LTR	L—2 × 3 cm undermined cartilage fragment proximal LTR	Dressage show horse
				R—2 × 2 cm lucency proximal LTR, distal patellar lysis	R—2 × 3 cm undermined cartilage fragment proximal LTR; 2 × 3 cm medial distal patella osteochondral fragment	

LTR = Lateral trochlear ridge of femur; MTR = Medial trochlear ridge of femur.

no lameness. A subtle 2 mm lucency was identified on the left LTR and there was subtle flattening with a lucent area on the proximal aspect of the right LTR (Fig. 3A). A subchondral bone cyst was also identified in the right medial femoral condyle. The right femoropatellar joint was injected with hyaluronic acid and one month of stall rest was recommended. The foal returned at 7 months of age because of continued femoropatellar effusion and bilateral Grade 2 hindlimb lameness. The radiographic lesions seen on both LTRs had increased in size (Fig. 3B) and bilateral arthroscopic debridement was performed. The horse was re-examined 16 weeks after surgery for right hindlimb lameness and femoropatellar effusion. A subchondral bone cyst, approximately 1.5 cm in length, was identified in the proximal aspect of the right LTR (Fig. 3C) and there was enlargement of the medial condylar bone cyst. Arthroscopy was performed again and the LTR subchondral bone cyst was located adjacent to the original lesion. The LTR and the medial condylar cyst were debrided surgically.

At 6 months of age, Horse 9, a male Warmblood-Thoroughbred cross, had a stage 1 flexural deformity[9] of the right front coronopedal joint, which was treated by distal check ligament desmotomy, corrective shoeing, and stall confinement. At 8 months of age the horse was re-admitted for bilateral femoropatellar joint effusion and a two-week history of having difficulty rising. Stifle radiographs and xeroradiographs were taken of both stifles; no lesions were noted in the left stifle and a subtle irregularity was noted in the proximal aspect of the right LTR. Both femoropatellar joints were injected with 50 mg hyaluronic acid and 4 weeks of stall rest was recommended. After 4 weeks the horse was admitted again for a Grade 2 bilateral hindlimb lameness and persistent femoropatellar joint effusion. Lysis of the distal border of the left patella and subchondral bone concavity and lucency of the left and right proximal LTR was evident on radiographs. Arthroscopic exploration revealed bilateral osteochondral fragmentation of the proximal aspect of the LTRs. Additionally, a 2 cm × 3 cm osteochondral lesion was debrided on the medial aspect of the distal articular surface of the right patella.

As a group, Horses 1, 3, 4, and 6, had no radiographic abnormalities at the initial examination but had changes evident on radiographs taken 4 to 12 weeks later. Horses 2, 8 and 9 initially had subtle flattening of one or both lateral trochlear ridges that progressed to large subchondral lucencies with fragmentation bilaterally within 4 weeks. Horses 5 and 7 initially had unilateral lesions with radiographic changes evident 16 to 24 weeks later in the opposite limb.

Arthroscopic Findings

Arthroscopic surgery was performed on 20 femoropatellar joints (Horse 5 and Horse 8 had right femoropatellar joint surgery twice). In general, direct visualization of the articular surface at surgery revealed more abnormalities than was anticipated from the pre-surgical radiographic examination. The lesions were confined to the proximal region of the lateral trochlear ridge of the distal femur in 11 joints. The proximal and distal regions of the lateral

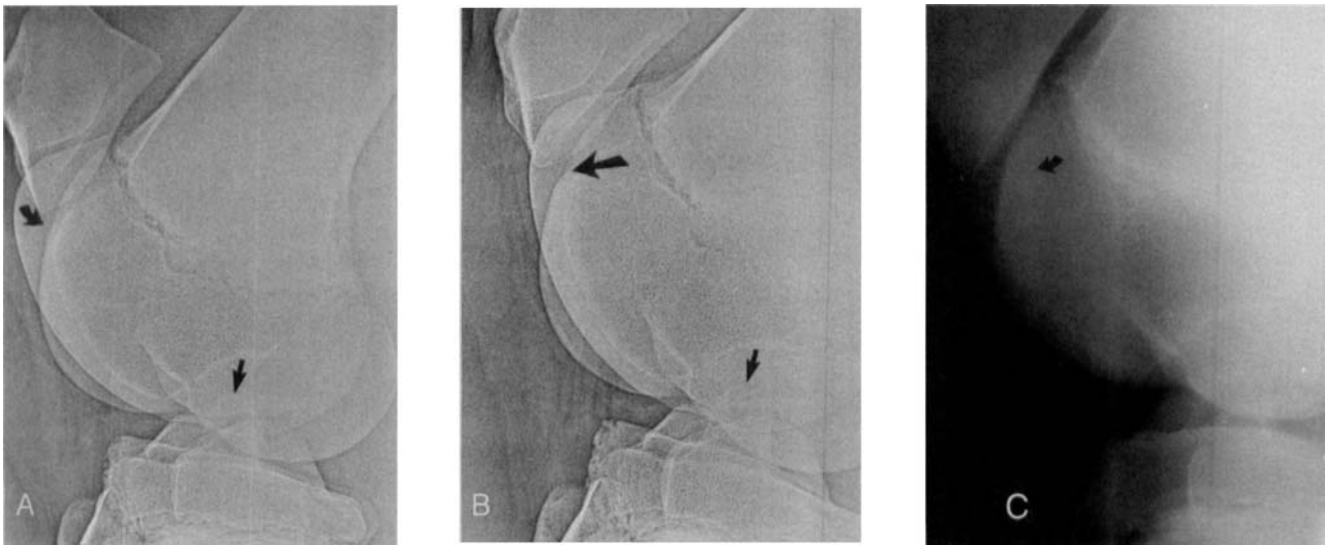

Fig. 3. Horse 8. Lateromedial projection. (A) At 6 months of age there is a 2 mm focal radiolucency (curved arrow) and subtle flattening of the right LTR. A subchondral bone cyst (short arrow) is identified in the medial condyle. (B) 7 months of age, there is a concave defect (large arrow) with fragmentation in the right proximal LTR. The bone cyst in the medial condyle (short arrow) is still present. (C) At 11 months of age there is a 1 cm cystic lesion (curved arrow) in the proximal LTR.

trochlear ridge were involved in three joints and both medial and lateral trochlear ridges were affected in the joints. Two of these joints had a concurrent lesion on the distal articular surface of the patella. One joint had a lesion in the proximal region of the lateral trochlear ridge and a subchondral bone cyst in the medial femoral condyle. Osteochondral fragmentation was found in nine joints, separation of cartilage only was observed in seven joints, and four joints had undermined, dimpled, or fibrillated cartilage with subchondral osteomalacia. Post-operative radiographs were obtained 2 to 7 months later in four horses (Horses 2, 3, 5 and 8) and sclerotic remodeling with smooth contours to the LTR borders were noted at the surgical sites (Fig. 4).

Based on follow-up information, eight horses were being used for their intended purpose (Table 1). Three horses were racing successfully, three were in race training, one was being used for dressage and one horse was used for pleasure riding. Horse 5 was euthanatized 2 months after the second surgical procedure. Necropsy confirmed satisfactory debridement of the LTR lesions however, there was bilateral eburnation of the articulating patellar and LTR surfaces.

Fig. 4. Horse 2. Seven months after surgery. Sclerotic remodeling and a smooth but irregular contour is apparent on the proximal LTR.

Discussion

Osteochondrosis is a disease affecting the normal maturation and differentiation of the cells in growing cartilage.[10] This results in failure of endochondral ossification and persistence of hypertrophied cartilage.[10] The early changes in the development of OCD have been reported in other species.[11-13] Initially, the chondrocytes are degenerative and disorganized at the osteochondral junction causing failure of endochondral ossification with focal cartilage retention and thickening.[13] Pressures or strain within the joint may create fissures in this damaged cartilage.[15] These cracks or fissures through the thickened cartilage allow debris from the deeper necrotic layers to gain access to the joint and result in clinical signs of synovitis and pain.[10,15] Additionally, changes in intraosseous pressure can result in subchondral bone pain and subsequent lameness.[10,14] The unaffected cartilage in the surrounding epiphysis continues to expand and ossify leaving a concavity in the subchondral bone that corresponds to the defect. The partially mineralized osteochondral fragments and irregular border of the subchondral epiphyseal bone of the LTR are observed radiographically as the disease progresses. Pascoe and coworkers suggested that the radiolucent defects observed in the LTR of the horse resulted from the continued ossification at the healthy osteochondral junction surrounding the focal area of delayed endochondral ossification.[4] A 30% to 70% loss or gain in bone density is required before changes are apparent radiographically.‡ The horses in this study were probably radiographed after the OCD lesions resulted in the clinical signs of pain and synovitis but before bony changes became radiographically apparent.

The age of the majority of horses affected with femoropatellar OCD is reported to range from younger than 3 years of age.[8,16] Generally, these horses can be divided into two groups according to their clinical and radiographic features. The first group is comprised of horses 2 years of age and older that develop an intermittent hindlimb lameness that commonly occurs when the horse begins training. The actual duration of the OCD is usually unknown. One report suggested that there was a higher frequency of LTR fragmentation in these horses.[4] The second group consists of horses that are one year of age or less. These horses are described as having difficulty rising from recumbency and having a bunny hop gait in the hindlimbs when traveling at higher speeds.[6] Clinical features include marked femoropatellar distention and lameness that is more noticeable in one hind limb. One group reported a higher frequency of subchondral lysis with less fragmentation in this age group;[4] but in another report there was no difference in

‡ CF Reid, DVM, personal communication, December 1992.

lesion type between age groups.[5] The clinical severity and prognosis is generally considered to be worse in the younger age group.[16] In the present study, horses ranged from 4 to 9 months of age at the onset of clinical signs. Lesions were either subtle (5 horses) or not observed (4 horses) at the initial radiographic examinations. The lameness and joint effusion were unchanged (Horses 1, 6, 9) or worsened (Horses 2–5, 7, and 8) with stall or paddock confinement suggesting continuation or progression of the pathologic process during this period. All horses had progressive bony changes on follow-up radiographic examinations. The rate of development of the radiographic lesions could not be determined because horses were re-examined at different times however, lesions were evident between 4 and 24 weeks after the initial examination in all horses.

Radiographic examination in this study included the LM and CaCr projections in all horses. Xeroradiographs were obtained at admission for Horses 4, 5, 8 and 9, because a lesion was suspected from the clinical signs but was not evident on the initial radiographs. The lateral (CaL-CrM) oblique projection was obtained in only three horses (Horses 1, 4, 9) and confirmed the radiographic findings identified on the LM and CaCr radiographic projections, but did not add additional information. The CaL-CrM projection was taken at clinician request and may have proved more useful had it been obtained in all horses. The CaL-CrM oblique projection is beneficial in characterizing the depth of the LTR lesions, although the location and length of the lesion are similar to that seen on the lateromedial projection.[17] The radiographic progression of the LTR osteochondrosis lesions in the horses in the present study could be easily identified on the LM projection.

Our findings of progressive radiographic changes with OCD of the LTR differ from the previously reported findings of decreasing subchondral lesion size over a one to two year period of repeat radiographic examinations.[7] Due to the poor correlation between surgical and radiographic findings, it has been suggested that the need for surgery should not be based solely on the radiographic examination,[5] as previously recommended.[8] For this reason, arthroscopic evaluation has been recommended for all horses with femoropatellar OCD.[5] Our experience supports the usefulness of arthroscopy as a diagnostic aid for femoropatellar OCD. Arthroscopic surgery at the time of onset of joint effusion may reveal cartilage lesions that are not detected radiographically and early removal of the focus of joint inflammation could decrease the extent of subsequent joint damage.[5,6,10] Alternatively, lesions that are still forming may not be seen at surgery and could progress with time as observed in Horses 5 and 8, necessitating a second surgery. Pascoe and coworkers reported that six of 15 operated femoropatellar joints had focal radiolucent regions in the subchondral bone of the LTR 6 weeks to 24 months postoperatively, suggesting that complete removal of the original lesion had not been achieved.[4] The age of these horses at the time of surgical intervention was not specified.[4] Findings from our study indicate that when minimal radiographic changes are evident in horses <9 months of age, with femoropatellar effusion and hindlimb lameness, that there maybe sufficient bony change within 4 to 6 weeks to permit radiographic identification of OCD. The optimum time for surgical intervention is unknown; however, eight of the horses in the present study were subsequently used as intended, when they had surgery after radiographic lesions became apparent.

Rapid progression of OCD can occur, making the prediction of lesion size difficult until after one year of age. Successful results were obtained in eight of the nine horses in this study when they were treated surgically after detection of radiographic lesions. We could find no reports that would allow comparison of outcome in horses with similar signs that had surgery before radiographic manifestation of the disease or with similar lesions that were managed conservatively. Further studies on the progression of femoropatellar OCD are needed to identify the optimal stage for treatment in horses younger than 1 year of age.

References

1. Moore JN, McIlwraith CW. Osteochondrosis of the equine stifle. Vet Rec 1977;100:133–136.
2. Stromberg B. Osteochondrosis dissecans of the stifle joint in horses: A clinical, radiographic and pathologic study. J Am Vet Rad Soc XVII, 1976;3:117–124.
3. Trotter GW, McIlwraith CW, Norrdin RW. A comparison of two surgical approaches to the equine femoropatellar joint for the treatment of osteochondritis dissecans. Vet Surg 1983;12:33–40.
4. Pascoe JR, Pool RR, Wheat JD, O'Brien TR. Osteochondral defects of the lateral trochlear ridge of the distal femur of the horse: Clinical, radiographic, and pathological examination and results of surgical treatment. Vet Surg 1984;13:99–110.
5. McIlwraith CW, Martin GS. Arthroscopic surgery for the treatment of osteochondritis dissecans in the equine femoropatellar joint. Vet Surg 1985;14:105–116.
6. McIlwraith CW. *Diagnostic and Surgical Arthroscopy in the Horse.* 2nd ed. Philadelphia: Lea and Febiger, 1990:139–153.
7. Stromberg B, Rejno S. Osteochondrosis in the horse 1. A clinical and radiographic investigation of osteochondritis dissecans of the knee and hock. Acta Radiol Suppl 1978;358:139–152.
8. Wyburn RS. A degenerative joint disease in the horse. NZ Vet J 1977;25:321–332.
9. Wagner PC. Flexural deformity of the distal interphalangeal joint. In: White NA, Moore JN, eds. *Current Practice of Equine Surgery.* Philadelphia: J.B. Lippincott, 1990:472–476.
10. McIlwraith CW. Diseases of joints, ligaments, and related structures. In: Stashak TS, ed. *Lameness in Horses* 4th ed. Philadelphia: Lea and Febiger, 1987:396–419.

11. Paatsama S, Rokkanen P, Jussila J, Sittnikow K. A study of osteochondritis dissecans of the canine humeral head using histological, OTC bone labelling, microradiographic and microangiographic methods. J Small Anim Pract 1971;12:603–611.
12. Hill MA, Ruth GR, Hilley HD, Hansgen DC. Dyschondroplasias, including osteochondrosis, in boars between 25 and 169 days of age: Histologic changes. Am J Vet Res 1984;45:903–916.
13. Kato M, Onodera T. Early changes of osteochondrosis in medial femoral condyles from rats. Vet Pathol 1987;24:80–86.
14. Olsson SE. Morphology and physiology of the growth cartilage under normal and pathologic conditions. In: Summer-Smith G, ed. *Bone in Clinical Orthopedics.* Philadelphia: W.B. Saunders, 1982:159–196.
15. Trotter GW, McIlwraith CW. Osteochondrosis in horses: Pathogenesis and clinical syndromes. Proc Am Assoc Equine Practnr 1981;27:141–159.
16. Jeffcott LB, Kold SE. Stifle lameness in the horse: A survey of 86 referred cases. Equine Vet J 1982;14:31–39.
17. O'Brien TR, Baker TW, Koblik P. Stifle radiology: How to perform an examination and interpret the radiographs. Proc Am Assoc Equine Practnr 1986;32:531–552.