Subchondral cystic lesions of the proximal extremity of the tibia in horses: 12 cases (1983–2000)

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Objective—To determine clinical and radiographic features of subchondral cystic lesions (SCL) of the proximal extremity of the tibia in horses that could be used to classify these lesions as being related to osteochondrosis or osteoarthritis and to evaluate results of surgical debridement.

Design—Retrospective study.

Animals—12 horses with 14 SCL.

Procedure—Medical records and radiographs obtained before and after treatment were reviewed.

Results—In 6 young horses (8 lesions), SCL were considered to be related to osteochondrosis; all involved the lateral tibial condyle. The remaining 6 horses were mature and had radiographic evidence of osteoarthritis in addition to SCL. Arthroscopic debridement was performed in 4 horses in which lesions were considered to be a result of osteochondrosis and in 3 horses with osteoarthritis. Three horses in which SCL were considered to be a result of osteochondrosis performed athletically after debridement. Two horses with moderate osteoarthritis returned to work after arthroscopic debridement but at a lower level of athletic performance. One horse with SCL related to osteochondrosis responded to medical treatment and went on to race.

Conclusions and Clinical Relevance—Results suggest that arthroscopic debridement of SCL is feasible in horses in which lesions involve the cranial portion of the lateral or medial tibial condyle, and that treated horses may be able to perform athleticism. (J Am Vet Med Assoc 2001;218:408–413)

Subchondral cystic lesions (SCL) are a well-documented cause of lameness and debility in horses. Although SCL, aneurysmal bone cysts, and unicameral bone cysts are grossly similar, the causes of these lesions differ. In addition, multiple causes may result in SCL in horses. The most prominent proposed causes of SCL in young horses are osteochondrosis and subchondral bone trauma. Experimental evidence suggests that trauma to the articular cartilage and subchondral bone can also result in formation of SCL in growing horses. In addition, SCL are encountered in horses as well as in humans with advanced osteochondrosis. In young horses, SCL can affect nearly any appendicular joint in horses; the medial femoral condyle is the site most commonly affected. Other described SCL locations include the distal extremity of the third metacarpal or metatarsal bone, the glenoid cavity, the proximomedial aspect of the radius, the distal extremity of the radius, and the phalanges. Lesions involving the tibia, however, have been reported infrequently.

The prognosis for horses with SCL involving the tibia is generally considered to be poor. Jeffcott and Kold described poor results following conservative treatment of horses with SCL involving the proximal extremity of the tibia. McIlwraith also suggested that the prognosis for affected horses was poor and that an intra-articular surgical approach to these lesions may not be feasible.

The purpose of the study reported here was to determine clinical and radiographic features of SCL of the proximal extremity of the tibia in horses that could be used to classify these lesions as being related to osteochondrosis or osteoarthritis and to evaluate results of surgical debridement.

Criteria for Selection of Cases

Medical records of all horses admitted to the Cornell University Hospital for Animals between 1987 and 2000, the University of Florida Veterinary Teaching Hospital between 1983 and 1988, and the Randwick Equine Centre between 1994 and 2000 that had SCL involving the proximal extremity of the tibia were reviewed.

Procedures

Medical record analysis and lesion categorization—Signalment, history, severity of lameness, results of all diagnostic tests (including results of intra-articular anesthesia, radiography, nuclear scintigraphy, surgery, histologic evaluation of biopsy specimens, and necropsy), other pertinent medical abnormalities (eg, history of neonatal infectious disease or injury or disease involving the affected stifle joint), and outcome were obtained from the medical records. Long-term follow-up information was obtained directly from the client or referring veterinarian whenever possible. Radiographs of the affected joint were reviewed. In most instances, lateral-medial and caudocranial radiographic projections of the stifle joint had been obtained; however, in some horses, flexed lateromedial and oblique projections had also been obtained to better illustrate the lesion.

Lesions were considered to be a result of osteochondrosis in young horses with solitary cystic lesions of the proximal extremity of the tibia that did not have evidence of OA. Lesions were considered to be a result...
of OA in the remaining horses, all of which were mature animals and had moderate or severe OA of the affected joint with a more variable lesion appearance.

Surgical technique—Lesions were surgically debrided, using an arthroscopic approach, in horses in which lesions were located in the cranial third of the tibial plateau. In horses with lesions involving the lateral tibial condyle, the lateral aspect of the femorotibial joint was approached via a medial portal, as described, with the arthroscope inserted between the middle and medial patellar ligaments. Lesions were typically identified cranial and immediately lateral to the lateral tubercle of the intercondylar eminence, and although the lesions typically communicated with the articular surface, the stoma was obscured by the cranial ligament of the lateral meniscus in some horses. In these horses, the ligament was bluntly divided with a probe, parallel to its fibers, to expose the stoma. Lesions were curetted to the level of healthy subchondral bone in standard fashion. Any associated femoral cartilage damage and fraying of the meniscal border was gently debrided. Fibers of the cranial ligament of the meniscus appeared minimally disrupted once instruments were removed and were not reappraised. Following curettage of the lesion, the joint was lavaged and closed routinely.

In horses in which the lesions were located medial to the intercondylar eminence, the medial aspect of the femorotibial joint was approached through a lateral portal, as described, with the arthroscope inserted caudal to the lateral patellar ligament. In these horses, lesions were identified by probing through the fibers of the cranial ligament of the medial meniscus in a manner similar to that described for lesions lateral to the intercondylar eminence.

Results

Signalment—Twelve horses met the criteria for inclusion in the study. Nine were male, and 3 were female. There were 6 Thoroughbreds, 2 Quarter Horses, 1 Standardbred, 1 Arabian, 1 Warmblood, and 1 Belgian. Breed distribution reflected the distribution of breeds examined at the participating hospitals. In 6 horses, lesions were considered to be a result of osteochondrosis. Mean age of these horses was 12.3 months (range, 6 to 24 months). In the remaining 6 horses, lesions were considered to be a result of OA. Mean age of these horses was 9.3 years (range, 2 to 12 years).

Clinical and radiographic appearance—Severity of lameness ranged from 0 to 3 on a scale from 0 to 5. Mean duration of lameness was 6.4 months (range, <1 month to >2 years). The lameness in all horses was exacerbated by flexion of the stifle joint. Stifle joint effusion was evident in 6 horses; however, records did not clearly document which joint pouch was palpably abnormal. Two horses in which SCL were considered to be a result of osteochondrosis had bilateral lesions. The left and right limbs were nearly equally affected (6 left, 8 right). Intra-articular anesthesia was performed in 6 horses; the lameness improved in 4 horses and was unchanged in the other 2, both of which had extensive deep lesions of the lateral tibial condyle. Specific information on number of minutes before evaluation of response to intra-articular anesthesia was not available from the medical records.

All 6 horses in which lesions were considered to be a result of osteochondrosis had solitary lesions involving the lateral tibial condyle that did not appear to be associated with other signs of joint disease (Fig 1). Radiographically, all lesions appeared to communicate with the surface of the subchondral bone. In 2 of the 8 affected joints, osteochondrosis lesions involving the lateral trochlear ridge of the femur were also evident. One of these horses was not lame and had been referred because of osteochondrosis of the lateral trochlear ridge discovered during a prepurchase radiographic examination. In this horse, the SCL was subsequently detected on preoperative radiographs at the referral institution.

Radiographically, 5 of the 6 horses in which lesions were considered to be a result of OA had well-defined SCL of the medial condyle of the tibia and signs of mild to marked OA, including remodeling of the proximal extremity of the tibia, osteophyte formation on the medial aspect of the tibia and femur, and subchondral bone sclerosis (Fig 2). Joint space narrowing was evident in 2 horses, and radiographic signs suggestive of cranial cruciate ligament injury, including osteophytes at the intercondylar eminence, were apparent in 1 horse. One horse had bilateral SCL of the medial femoral condyles, and the tibial lesion evident in 1 joint appeared to have arisen secondarily. The remaining horse in this group had multiple loculated lesions involving the lateral tibial condyle and extending into the metaphysis. In general, lesions in the horses in which SCL were a result of OA were larger than lesions in the horses in which SCL were a result of osteochondrosis. All lesions related to osteochondrosis were located in the lateral tibial condyle, whereas 5 of 6 lesions related to OA were located in the medial tibial condyle. In 3 horses in which results of physical examination did not localize the cause of the lameness, nuclear scintigraphy was performed. Focal uptake of radioisotope was evident in the proximal extremity of the tibia in all 3 horses, and radiographic evaluation of this area was performed on the basis of these findings.

Treatment, surgical findings, and outcome—In 4 of the 6 horses in which SCL were considered to be a result of osteochondrosis and in 3 of the 6 horses in which SCL were considered to be a result of OA, lesions were debrided surgically. Separation of the fibers of the cranial ligament of the meniscus was necessary in 5 horses (Fig 3). The stoma of the lesion was visible immediately caudal to the cranial ligament of the meniscus in the other 2 horses (Fig 4). Entry to the lesion was confirmed in 3 horses by use of intraoperative radiography. Areas of trochlear-ridge flattening evident on preoperative radiographs in 3 horses led to arthroscopic examination of the femorotibial joint; however, dissecting osteochondral flaps were not found. One horse had a dissecting cartilage flap overlying the cystic lesion in the tibia.

Three of the 4 horses with SCL related to osteochondrosis that underwent surgery went on to perform athletically (mean follow-up time, 2.8 years; range, 6
weeks to 9 years). The fourth horse developed a femoral neck fracture 6 weeks after surgery and was euthanatized.

In 2 horses with SCL related to osteochondrosis, medical management was attempted. For 1 of these horses, treatment consisted of stall rest for 8 weeks, systemic administration of anti-inflammatory agents for 2 weeks, and IM administration of pentosan polysulphate (3 mg/kg [1.4 mg/lb] of body weight, every 7 days) for 4 weeks. The horse eventually underwent arthroscopic debridement at 1 year of age because the lameness had persisted and the lesion had enlarged radiographically. This horse is currently performing well in race training as a 2 year old. The second horse that underwent medical management had bilateral lesions and was treated with 3 weekly intra-articular injections of polysulfated glycosaminoglycan solution in the femorotibial joints and stall rest for 8 weeks. This horse improved clinically and radiographically and went on to an 8-year racing career with intermittent hindlimb lameness. A third horse did not receive medical or surgical treatment, remained lame for 2 years, and was subsequently destroyed.

Follow-up radiographs were obtained for 3 of the 6 horses in which SCL were considered to be a result of osteochondrosis, including 2 that had been treated surgically and 1 that had been treated medically. Bony filling of the lesions was evident in all 3 horses; there was no radiographic evidence of OA.

All 3 of the horses with SCL related to OA that underwent surgical debridement had only moderate OA. At surgery, generalized erosion of the cartilage of the opposing femoral condyle with fibrillation was evident in 2 horses, and evidence of cranial cruciate ligament injury was also apparent in 1 of these horses. The third horse that underwent arthroscopic debridement had tearing of the cranial ligament of the medial meniscus, and following curettage of the SCL, an autogenous cancellous bone and cartilage graft was placed in the defect. This horse was sound at a walk 12 weeks after surgery. The other 2 horses with SCL related to OA returned to athletic activity, although at a lower competitive level. Follow-up radiographs were obtained on 2 of the horses that underwent arthroscopic debridement. Three and 6 months after surgery, bony filling of the lesion was evident in both horses; the radiographic evidence of OA was unchanged from the preoperative radiographs.

Of the remaining 3 horses in which SCL were considered to be a result of OA, 1 underwent extra-articular transcortical debridement of the lesion but was subsequently euthanatized because of wound dehiscence and synovial fistula formation. The other 2 were considered unsuitable for surgery; 1 was treated with nonsteroidal anti-inflammatory agents and lost to follow-up, and the other was euthanatized without treatment.
Histologic evaluation—Four lesions were evaluated histologically. One lesion obtained at the time of surgery consisted of a cartilage flap overlying a subchondral defect (Fig 4); the histologic diagnosis was osteochondritis dissecans. The other 3 were obtained at necropsy and were characterized as being lined with connective or granulation tissue that contained some inflammatory cells and a few vascular and chondro-osseous elements.

Discussion

Results of the present study suggest that the proximal extremity of the tibia is an uncommon site for SCL in horses. We were able to identify only 12 cases by reviewing medical records for a 13-year period at 1 referral institution and for 5- and 6-year periods at 2 other institutions. Because of this low number of cases, results of the present study can only be used as a guide for determining outcome of horses with SCL involving the proximal extremity of the tibia. However, it is apparent that a categorically poor prognosis for horses with these lesions is unwarranted, particularly for young horses in which lesions are apparently a result of osteochondrosis and concurrent joint abnormalities are not evident. The arthroscopic approach provided adequate access to these lesions, and 3 of the 6 horses in which SCL were considered to be a result of osteochondrosis returned to athletic performance. Conversely, horses with large lesions or lesions associated with OA or other intra-articular abnormalities, which typically were mature horses, appeared to have a more guarded prognosis. Arthroscopic debridement of lesions in the medial tibial condyle in 3 horses with moderate OA resulted in a fair clinical outcome, with 2 horses able to return to some athletic function and the third horse progressing well in early convalescence. These 3 horses were the least affected of all the 6 horses with concurrent OA and do not represent the entire population of horses with SCL of the proximal extremity of the tibia and concurrent OA. Therefore, we believe that most horses with OA that have these types of lesions are not good candidates for surgery and deserve a guarded prognosis regardless of treatment.
Although it is well-accepted that SCL may be a sequela of OA in humans, the veterinary community has only recently acknowledged that OA may induce development of SCL. Most reports published in the veterinary literature prior to 1982 described populations of young horses with lesions at a few characteristic sites. As such, the idea of SCL as a uniform clinical entity that represented a manifestation of osteochondrosis alone was quite common. However, more recent case reports include descriptions of mature horses with SCL in various anatomic locations, of varied gross appearance, and in association with other evidence of trauma or OA. To add to the controversy, the horses in the present report in which SCL were considered to be a result of osteochondrosis had a history of neonatal infectious disease: 1 had pneumonia at 6 weeks of age, associated with Rhodococcus equi infection (no synovitis was reported in association with the pneumonia, however) and the other had presumptive septic osteomyelitis at 4 weeks of age at a site distant from the proximal extremity of the tibia. Bone and joint sepsis have previously been recognized as possible causes of SCL and warrant further consideration in the pathogenesis of these lesions.

Although we postulated that SCL in horses with OA were a result of degenerative subchondral bone trauma, the exact chronology of joint disease in these horses is essentially unknown. The SCL may have been primary lesions that incited secondary osteoarthritic changes rather than a result of cartilage degeneration secondary to OA. All 3 of the horses with OA that underwent surgery had lesions involving the medial tibial condyle in combination with damage to the cranial cruciate ligament, the medial meniscus, or the cranial ligament of the meniscus. It is possible that destabilization of the medial aspect of the femorotibial joint secondary to loss of meniscal or ligamentous integrity resulted in abnormal weight-bearing and subchondral bone trauma. Alternatively, it is possible that whatever traumatic event caused these structures to be damaged may have caused direct mechanical trauma to the subchondral bone, resulting in formation of the SCL. It is also possible that the SCL formed secondarily to the degeneration of articular cartilage associated with OA. With exposure of the subchondral bone, erosion canals may develop, and extrusion of synovial fluid to the deeper bony layers may exert enough pressure to cause bone resorption, resulting in a cystic lesion. In the horses in which we considered SCL to be related to osteochondrosis, we also acknowledge that this was often a presumptive diagnosis, as most horses lacked a definitive histologic diagnosis. The lesions of these young horses may also have resulted from trauma rather than an inherent cartilage defect such as osteochondrosis. The horses described in the present report lend further support to the idea that even in the case of a common inciting event (ie, subchondral bone perforation), the events surrounding development of SCL are apparently variable. Development of SCL in association with OA in humans is a contentious topic despite the availability of a large number of reported cases and the more extensive histologic investigations of these lesions.

The pathogenesis of SCL in horses, as in other species, may involve developmental, traumatic, degenerative, infectious, and vascular factors. Clearly, further histologic studies on the formation of SCL are needed.

In 7 of 12 horses in the present report, arthroscopic debridement was possible, because the lesion was located in the cranial third of the tibial plateau. However, many of the lesions in horses with OA developed in the central weight-bearing portion of the proximal extremity of the tibia and would have been inaccessible arthroscopically. Therefore, we suggest that careful scrutiny of lateromedial radiographic views is essential prior to attempting intra-articular arthroscopic debridement.

References
21. McIlwraith CW. Subchondral cystic lesions in the horse—