Take Home Message: There are various manifestations of subchondral cystic lesions (SCLs). Treatment of clinically significant lesions revolves around Type 1 and Type 2 lesions. A consensus algorithm for treatment of SCLs of the medial femoral condyle (MFC) involves arthroscopic surgery and if the cyst has stable margins to a probe, and there is no penetration or collapse of the margins, intralesional corticosteroid injection is the first option. Cysts with collapsed margins are debrided. Failure to respond to intralesional corticosteroids leads to a recommendation of arthroscopic evacuation and debridement in an immature horse and arthroscopic debridement plus augmentation in an older horse. A recent option includes placement of a cortical bone screw across the lesion.

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I. INTRODUCTION

Subchondral cystic lesions and their pathogenesis and consequent treatment methods in particular, still incite controversy. The lesions were initially described as subchondral bone cysts; later, other authors described them as subchondral cystic lesions or osseous cyst-like lesions to avoid the implication that they are a true cyst. However, when examined pathologically, the author feels that they conform to most people’s definition of a cyst in that they have a lining (Fig. 1). They occur in a number of locations. Subchondral bone cysts were first reported as a clinical entity in 1968. In that report there were 12 cases in the phalanges and one in the radial carpal bone. A series was reported in 1982 in the stifle and distal phalanx. A third series of 69 cases with 64 horses was reported in 1970 under a modified name, osseous cyst-like lesions. In that series, there were 15 instances of cysts in the carpal bones, 10 in the third metacarpal bone, 3 in the radius, 5 in the proximal sesamoid, 6 in the proximal phalanx, 4 in the middle phalanx, 5 in the distal phalanx, 6 in the navicular bone, 12 in the femur, 2 in the tibia and 3 in the tarsal bones. Since that time, the most common site of clinical cases reported has been the medial condyle of the femur.

Fig. 1. A gross sagittal section revealing the appearance of a subchondral cystic lesion. Sclerotic bone is present around the cyst (a), dense fibrous tissue lines the cyst (b) and the center is often filled with gelatinous material (c). Reproduced with permission from Wallis TW, Goodrich LR, McIlwraith CW, et al. Arthroscopic injection of corticosteroids into the fibrous tissue of subchondral cystic lesions of the medial femoral condyle in horses: a retrospective study of 52 cases (2001-2006). Equine Vet J 2008;40:461–467.

II. WHAT IS A SUBCHONDRAL CYSTIC LESION AND WHAT ARE WE TREATING?

This discussion will address SCLs of the MFC in the medial femorotibial joint of the stifle as this is the most common
condition we deal with and studies on pathogenesis have been specific for this lesion. In early literature, cases of SCLs had lameness and radiographs usually showing an obvious lesion. Lameness is the usual reason that cases are presented to the veterinarian. Direct palpation of medial femorotibial joint effusion is uncommon but it has been reported that approximately 60% of cases will report with femoropatellar effusion. This is presumably related to known communication (at least the potential) between the medial femorotibial joint and the femoropatellar joint. The condition is confirmed by radiographs (Fig. 2). With the advent of digital radiographs and survey radiographs at yearling sales, more attention is now paid to subchondral defects and even flattening of the medial femoral condyle in addition to ‘traditional’ SCLs. These cases are usually asymptomatic.

Fig. 2. Radiographic appearance of two types of subchondral bone cysts as described by White et al 1988. A, Type I dome shaped lesion; B, Type II circular lucent defect.

With regard to radiographic appearance and classification, there has been an evolution from initially two types: Type I being a radiographically dome shaped lucent area which was confluent with a flattened joint surface and Type II had a circular lucent area within the condyle with a thinner radiographically lucent tract connecting the cysts to the articular surface of the condyle (Fig. 2). This early classification evolved into three types of SCLs. Type I – 10mm or less in depth, Type II – more than 10mm in depth and Type III – flattened or irregular contour of the subchondral bone. More recently, five different types have been described by Wallis et al (Fig. 3). Type 1 lesions were defined as being <10mm in depth and were usually dome shaped. Type 2A lesions were >10mm in depth and had a lollipop or mushroom shape with a narrow cloaca and a round cystic lucency. Type 2B lesions were >10mm in depth with a large dome shape extending down to a large articular surface defect. Type 3 lesions (noted incidentally on survey radiographs of yearlings) were defined as condylar flattening or small defects in the subchondral bone, usually noted in the contralateral limb to that of the clinically significant SCL. Type 4 lesions were defined as those that had lucency in the condyle with or without an articular defect, but had no radiographic evidence of a cloaca in the subchondral bone plate.

Digital radiographs and survey radiographs have led to increased scrutiny of radiographs. As part of a study to prospectively examine the significance of lesions in the medial femoral condyle of cutting horses there was further definition of subchondral defects and flattening of MFCs. In this publication to differentiate ‘lesions’ that were being noted as potentially significant by some veterinarians, the MFC was classified as smooth and continuously convex in contour (Grade 0); flattened without radiographic evidence of subchondral bone changes (Grade 1); a small defect or change in, without extension through, the subchondral bone (Grade 2); a shallow, crescent shaped subchondral lucency that is wider than tall and confluent with the joint surface (Grade 3); or a well-defined round or oval radiolucency in the middle of the MFC that communicated with the femorotibial joint (Grade 4). (In this classification Grade 3 is equivalent to the Type 1 of Wallis et al and Grade 4 equivalent to Grade 2 without subdivision into a and b).
III. PATHOGENESIS

Subchondral cystic lesions have been proposed as manifestations of osteochondrosis by a number of authors and it is the author’s opinion that with young horses and, particularly, in bilateral cases, this is the probable cause. On the other hand, there has been progressive recognition that subchondral bone cysts occur in older horses and some have been identified with an initial articular defect. In an early study by Kold et al, a subchondral bone cyst was produced experimentally in a pony by creating a linear cartilaginous defect in a central weight-bearing area of the medial femoral condyle. More recent work in the author’s laboratory did not duplicate this finding but showed that a subchondral bone defect could induce an SCL. A full thickness linear defect was created in the articular cartilage of the medial femoral condyle in 6 femorotibial joints in a group of exercised horses and, in all cases there was no formation of subchondral bone cysts. However, in the same study, concurrent elliptical cartilaginous and subchondral bone defects (5 mm diameter and 3 mm deep) in the medial femoral condyle resulted in the development of cystic lesions in 5 of 6 horses. This experimental finding, as well as anecdotal clinical evidence, lends support to the theory that direct mechanical trauma to the subchondral bone plays a role in the development of subchondral bone cysts.

Some work from a collaborative study, between the Orthopaedic Research Center (ORC) at Colorado State University (CSU) and the University of Zurich, has demonstrated that the fibrous tissue contents of SCLs in horses produce prostaglandin E2 (PGE2), nitric oxide (NO) as well as matrix metalloproteinases (MMPs). Of all the variables measured PGE2 concentrations were the highest in cystic tissue compared with synovial membrane and articular cartilage from normal joints and joints with chip fractures, indicating that this mediator may play an important role in pathological bone resorption associated with SCL. These findings were then supported by the observation that conditioned media of SCL tissue were capable of recruiting osteoclasts and increasing their activity. Such active bone resorption may play a role in the pathogenesis of subchondral bone cysts and may also be significant in the continued enlargement of cystic lesions well after the cessation of endochondral ossification. A second study investigated the potential association of interleukin-1beta (IL-1β) and interleukin-6 (IL-6) in SCLs in horses using in situ hybridization of paraffin sections of fibrous tissue of SCL and quantitative real-time PCR in fresh frozen tissue and undecalcified bone sections of SCL embedded in acrylic resin. Upregulation of mRNA of both cytokines could be demonstrated. Interestingly, mRNA of IL-1β was upregulated at the periphery of the cystic lesion adjacent to the normal bone, whereas IL-6 mRNA was upregulated within the fibrous tissue found within the center of the SCL. It was concluded that both cytokines are associated with the pathological bone resorption observed in SCLs and in combination with increased production of PGE2 may be responsible for the slow healing, maintenance or further expansion of the cystic lesions.

As an ‘intermediate’ hypothesis, it has been thought that compressive forces encountered in normal weight-bearing may encourage the formation of subchondral bone cysts by contributing to the deformation of thickened cartilage previously compromised by a disturbance in the endochondral ossification process. Associated with this observation is the observation that subchondral bone cysts tend to occur at the location in a joint subjected to maximal weight-bearing during the support phase of the stride.

More recently a new treatment technique for SCLs has been developed using a cortical bone screw where the originator of the technique proposes that altering strain on the SCL will promote trabecular bone formation and remodeling.

IV. TREATMENT BASED ON PATHOGENESIS

Treatment techniques have continued to be somewhat controversial but initially success was reported with conservative therapy (rest and antiinflammatory agents) by Jeffcott & Kold and Stewart & Reid. However, older horses have a worse prognosis; in the author’s experience some horses progress to athletic activity (including racing) without surgery but most have persistent problems.

Some form of surgical intervention has generally been recommended when athletic activity is required. Criteria for ‘success’ have varied and some horses with conservative treatment can go on to alternative and less demanding careers. For instance, we have seen horses that are bred to race but end up being riding horses. The criterion for success, at least with athletes, is generally that the horse returned to the complete activity that it was undertaking before or for which the horse was bred and at the same level. Historically there have been a number of surgical treatments. In 1975-1978 the author attempted surgical treatment using an extra-articular approach and packed the defect with cancellous bone graft in similar fashion to the technique used in phalangeal cysts by Kold & Killingbeck. None of the initial 6 cases treated in this fashion achieved athletic soundness. This was attributed to inadequate curettage at the edge of the cyst and lack of penetration of the sclerotic bone but of course this was based on the SCLs being considered inert structures that just required increase in healing without any awareness of progressive inflammation and osteolysis. Similarly, a periarticular approach with debridement and packing was considered successful by White & Prades.

From 1979 to 1998 an intraarticular approach was used with arthroscopy into the medial femorotibial joint between the middle and medial patellar ligaments with the joint flexed. Of 48 horses that did not have evidence of osteoarthritis (OA) prior to surgery, all improved and 35 were not lame when returned to athletic activity. Of four with OA signs prior to surgery, two remained lame and two improved. At that stage surgery was recommended for cases that had not responded to at least 3 months rest; however, a long delay did not necessarily have detrimental effects because horses that had been lame for a year or more before surgery became sound after surgery.

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Surgery using arthrotomy and packing of cancellous bone graft into the curetted cavity with the rationale that this would improve healing did not show any benefit of curettage alone.\textsuperscript{,7,28} Because curettage and leaving the cavity open had superior results this was the technique adopted by most.\textsuperscript{12}

In 1989 the author started using arthroscopic surgery as described by Lewis to treat these lesions.\textsuperscript{8} Excellent visualization of the cystic lesion can be obtained and a follow-up study reported on outcome for 41 horses with SCLs of the MFC treated by arthroscopic surgery.\textsuperscript{5} There were 17 Quarter horses, 15 Arabians, 8 Thoroughbreds and 1 Holsteiner with 28 (68\%) of the horses being 1-3 years old. For all horses the initial complaint was mild to moderate hindlimb lameness or an altered gait. Bilateral radiographic abnormalities were detected in 27 horses and 19/27 horses had lesions identified at arthroscopic surgery. In addition to the SCL, 13 joints in 11 horses had an OCD lesion on the articular surface of the medial femoral condyle that extended from the opening of the SCL. Surgical debridement via arthroscopy was the only treatment for 37 lesions of 23 horses. Debridement followed by drilling of the defect bed was performed in 23 lesions of 18 horses. Complete follow-up information was obtained for 39 horses; 22 (56\%) had a successful result and 17 (44\%) had an unsuccessful result. However, in a separate analysis excluding horses with unsuccessful results because of factors not directly attributable to the SCL of the MFC (so-called censored analysis), 23/31 (74\%) horses had a successful result and 8/31 (26\%) horses had an unsuccessful result. Within this group of horses the prognosis for a successful result was not associated with age, sex, size of lesion, unilateral vs. bilateral lesions, whether the lesion was drilled, the presence of OCD associated with the SCL or whether the lesion enlarged after surgery. Compared with Thoroughbreds and Arabians, Quarter Horses had a poorer prognosis for success. Follow-up radiographs were available for 14 horses. In 9 of these 14 horses the SCL had enlarged after surgery. Postoperative cystic enlargement was associated significantly with drilling of the lesion bed at the time of surgery.\textsuperscript{5} Subsequent to these findings we ceased using subchondral bone drilling and also added the intralesional injection of 40mg of methylprednisolone acetate\textsuperscript{4} in the regimen. The latter technique was based on extrapolation from the treatment of unicameral bone cysts in humans. Since commencing the use of corticosteroids at the time of curettage we did not see any cysts increase in size.

Other authors have reported on results with arthroscopic debridement and certain limitations have been identified. In a study in Kentucky of 150 cases, 70\% of horses with a surface defect <15mm started a race after surgery whereas only 54\% with >15mm of surface defect diameter started a race after surgery.\textsuperscript{29} In another study in the UK 25/39 (64\%) of horses aged 0-3 years of age returned to soundness whereas 16/46 (35\%) of horses >3 years returned to soundness.\textsuperscript{30} More recently, a retrospective study identified concurrent or sequential development of medial meniscal tears and SCLs within the medial femorotibial joint in horses.\textsuperscript{31} In this study 21 horses (9.1\% of all horses undergoing MFC arthroscopy during that period) were identified to have both a medial meniscal injury and SCL of the MFC. Thirteen horses had the abnormalities concurrently. Six developed a meniscal lesion subsequent to SCL debridement and this led to a hypothesis that the rim of a debrided SCL could cause damage to the medial meniscus or medial meniscotibial ligament. Two horses also developed an SCL subsequent to meniscal injury. Only 4/19 horses were classified as successful and returned to intended use.

During the time period of this study the findings of von Rechenberg et al\textsuperscript{21,22} led to development of a technique of injecting corticosteroids directly into the fibrous lining of the cystic lesion under arthroscopic guidance.\textsuperscript{13} This is a good example of laboratory bench research leading to an improved clinical technique. The rationale for intralesional injection of triamcinolone acetonide possibly improving results compared to previous studies using debridement included shorter convalescence, less articular disruption, less potential for cystic enlargement, and less potential for causing a subsequent meniscal lesion. Although the injection technique can be performed under radiographic or ultrasonographic monitoring, the arthroscopic technique has been preferred by the author to debride any loose chondral or osteochondral flaps (as was earlier seen during arthroscopic debridement\textsuperscript{1}). In a retrospective study on the results of arthroscopic injection of corticosteroids into the fibrous tissue of SCLs of the MFC in 52 cases, the inclusion criteria were lameness and radiographic evidence of an SCL. The results showed that 35/52 (67\%) horses were successfully treated. An additional 5 horses giving a total of 40/52 (77\%) were considered sound at veterinary recheck examination, but for various reasons were not performing their intended athletic discipline. When no other variable was considered other than the presence of an SCL, 56/73 SCLs (77\%) treated by this procedure resulted in a sound leg. Of the SCLs from unilateral cases 28/31 (90\%) were considered successful and of the bilateral SCLs, 28/42 (67\%) were considered successful. This equated to 25/31 horses with unilateral SCLs (81\%) classified as successful vs. 10/21 horses with bilateral SCLs (48\%) (p=0.01). A change in radiographic size of SCLs was recorded in 22 SCLs that had pre and postoperative radiographs available. Seven/nine (78\%) SCLs revealed a decrease in size and were classified as successful while 9/13 (69\%) SCLs did not reveal a change in size but were still classified as successful. Only 1/22 SCLs had evidence of enlargement postoperatively and this horse remained lame following treatment. There was no significant association found between success and age when separated into age groups of 3 years or less and >3 years of age. Forty-three/55 (78\%) SCLs in horses aged 0-3 years and 13/18 (72\%) SCLs in horses aged >3 years were classified as successful. This equated to 27/39 (69\%) horses aged 0-3 years and 8/13 (62\%) aged >3 years classified as successful. Radiographic findings of osteophytes were found on preoperative radiographs or listed in the radiograph report of 16/61 SCLs (26\%). There was a significant association between absence of these osteophytes and success (p=0.04). Ten/16 limbs (63\%) with these radiographic signs were classified as successful as compared to 39/45 (87\%) without any radiographic osteophytes being classified as successful.
V. ALGORITHM FOR CASE MANAGEMENT

A consensus algorithm for treatment of SCLs of the medial femoral condyle is depicted in Figure 4. Arthroscopic evaluation of the medial femorotibial joint is performed. If the cyst has stable margins to a probe and there is no penetration or collapse of the margins intraslesional corticosteroid injection of the SCLs are recommended. On the other hand, if the cyst has unstable or collapsed margins then debridement is recommended, realizing that there are other consequences as previously described.

![Algorithm for Case Management](image)

If the case fails to respond to intraslesional corticosteroids, the author recommends arthroscopic evacuation and debridement in an immature horse but if the horse is 3 years of age or older the author will go ahead with arthroscopic debridement plus augmentation.

A number of options are available for augmentation including fibrin containing chondrocytes and growth factors, fibrin and stem cells, packing with bone substitute together with superficial augmentation of fibrin, growth factors, chondrocytes or stem cells.

VI. USE OF A TRANSCONDYLAR SCREW

This is a new technique that has been developed with the hypothesis that altering SCL strain is believed to promote trabecular bone formation and remodeling. A single 4.5mm cortical screw is placed in lag fashion across the condyle. It is placed cranial to the medial collateral ligament and proximal to the articular surface, angled in a slight cranial-caudal direction. Individual cases of bone healing have been reported with correlative improvement in lameness and resolution of lameness. The screw is generally left in place for horses in active training and cases can perform with the screw in place.

Eighty-percent success rate in returning horses to their intended discipline has been reported. In a recent report, 26 limbs were treated. Nine horses (11 limbs) had autologous adjunctive biologics placed into the SCL. Lameness was reduced by 1-2 grades 60 days after surgery in 18 horses and was eliminated in 15 horses 150 days post-surgery at which time the SCL area had decreased greater than 50% and work had resumed without lameness (mean follow-up 12 months).

The author has not performed this technique in any horses at this stage and is waiting for more data on screw implantation. However, it is a reasonable option for a previously failed treatment.

REFERENCES AND FOOTNOTE


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