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Management of subchondral cystic lesions of medial femoral condyle - an evolution based on research and clinical evidence

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Summary

Subchondral cystic lesions (SCLs) from the medial femoral condyle (MFC) are a common problem in horses, are usually located on the weight-bearing surface of the joint and may be clinical or non-clinical in nature. In early literature cases had lameness and radiographs usually showed an obvious cystic lesion. Most recently clinical lesions have been identified as 5 types, with type I being a dome shaped lucent area opening to the surface, type II being full cystic lesions with A and B sub-types depending on the opening at the joint surface, type III had flattening or irregular contour of the subchondral bone and type IV had an SCL with no radiographic evidence of a cloaca in the subchondral bone plate. With the advent of digital radiography equipment and survey radiographs at yearling sales, more attention has been paid to flattening and subchondral defects and even flattening of the medial femoral condyle. There has been an evolution of understanding in pathogenesis, diagnosis and treatment. Many SCLs are associated with osteochondrosis, but more recently trauma to the subchondral plate has been recognized as possibly leading to SCLs. In addition, the fibrous tissues of SCLs in horses produce various degradative mediators and are capable of recruiting osteoclasts. Diagnosis is straight-forward, but predictability of lameness in yearlings based on radiographs is difficult, but new studies correlating later performance with these sale radiographs is elucidating significance. Treatments have evolved from curettage via arthrotomy, to curettage via arthroscopic surgery and most recently, intra-lesional injection of triamcinolone acetonide via arthroscopic evaluation based on the newer findings regarding pathogenesis. Success rate has concomitantly improved. More exotic methods including the use of chondrocytes or mesenchymal stem cells and IGF-1 in fibrin with or without bone grafting have been used. The use of stem cells in fibrin has been successful in cases not responding to the intra-lesional corticosteroid technique.

Keywords: Cystic lesion, subchondral bone cyst, developmental orthopedic diseases, equine

Die Behandlung suchchondraler Knochenzysten des medialen Femurkondylus - Fortschritte auf der Basis von Forschung und klinischer Erfahrung

Subchondrale Knochenzysten (SCLs) des medialen femoralen Kondylus (MFC) sind ein recht häufig beobachtetes Phänomen bei jüngeren Pferden. Sie sind normalerweise an der gewichtstragenden Gelenkfläche situiert und können Zufallsbefunde sein, aber auch klinische Symptome hervorrufen. In frühen Berichten der Literatur wurden vor allem zystische Läsionen gezeigt und die Pferde zeigten Lahmheiten. In der neueren Fachliteratur werden 5 Formen von zystischen Läsionen unterschieden, nämlich Typ I) als kuppelförmige transparente Zyste , welche sich zu der Gelenkoberfläche hin öffnet, Typ II) als kreisförmige, volle zystische Läsion mit A und B als Sub-Typ, je nachdem wie sich die Öffnung zum Gelenkspalt hin gestaltet, Typ III) als Abflachung und/oder irreguläre Kontur des subchondralen Knochens am MFC und letztlich Typ IV) als subchondrale Knochenläsion ohne Vertiefung in der subchondralen Knochenplatte. Mit der Verfügbarkeit von digitalen Röntgenmaschinen und damit Übersichtsaufnahmen bei Jährlingsauktionen, konnte mehr Aufmerksamkeit auf die frühen Läsionen, wie Abflachung des MFC und/oder Knochenläsionen, gelegt werden als früher. Seit der Entdeckung des SCL konnte ein gewisses Verständnis zur Pathogenese, Diagnose und auch Behandlung erarbeitet werden. Viele SCL sind vergesellschaftet mit Osteochondrose-Läsionen, aber auch mit Trauma an der subchondralen Knochenplatte. Zusätzlich wurde herausgefunden, dass die fibröse Auskleidung des SCL selbst an der Produktion von degradativen Entzündungsmediatoren beteiligt ist und sowohl für die Rekrutierung und Aktivierung von Osteoklasten – und damit Knochenresoprtion – verantwortlich ist. Die Diagnose ist relativ einfach, aber die Vorhersagbarkeit für Lahmheiten von Jährlingen basierend auf Röntgenbildern ist immer noch schwierig. Neuere Untersuchungen, welche die Röntgenbilder der einjährigen Rennpferde zu ihrer späteren Leistung korrelierten, konnten da etwas Licht ins Dunkel bringen. Die klinische Behandlung entwickelte sich von der einfachen Kurrettage via Arthrotomie zur Kurretage via Arthroskopie und in den letzten Jahren zur intraläsionalen Injektion von Triamcinolone-acetonide via arthroskopischer Diagnostik aufgrund der Erkenntnis, dass SCL entzündlicher Natur sind. Entsprechend hat sich die Erfolgsrate der Behandlung von SCL verbessert. Mehr exotische Methoden beinhalten den Gebrauch von Chondrozyten oder mesenchymalen Stammzellen (MSC) und/oder IGF-1 in Fibrin mit oder ohne autologe Knochenaugmentation. Dabei zeigte sich die Verwendung von MSC in Fibrin als erfolgsversprechend in Fällen, welche keine Besserung nach intraläsionaler Injektion von Kortikosteroiden zeigten.

Schlüsselwörter: Knochenzyste, subchondral, Lahmheit, Orthopädie, Pferd, medial, Femurkondylus

Introduction

Subchondral cystic lesions (SCLs) from the medial femoral condyle (MFC) are a common problem in horses and are usually located on the weight-bearing surface of the joint and may be clinical or non-clinical in nature (*Baxter* 1996, *McIlwraith* 2002). They have been graded or grouped by previous investigators on the basis of radiographic appearance (*White* et al. 1988), location (*Jeffcott* and *Kold* 1982), as well as depth and lesion size (*Howard* et al. 1995). They have been variously described as subchondral bone cysts, subchondral cystic lesions, or osseous cyst-like lesions (*McIlwraith* 1998). They have also had careful comparison to subchondral bone cysts secondary to osteoarthrosis in humans (*von Rechenberg* et al. 1998).

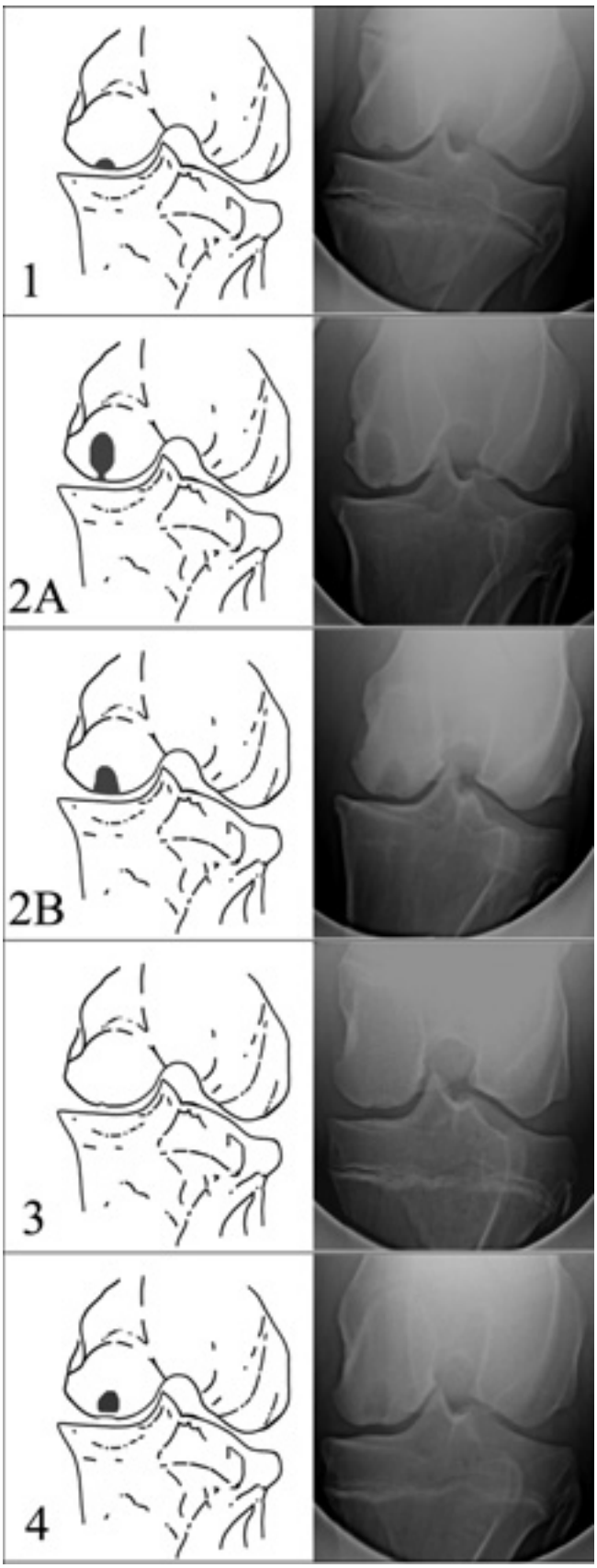

Fig. 1 Type I lesions were defined as being <10 mm in depth and were usually dome shaped. Type 2A lesions were >10 mm in depth and had a lollipop or mushroom shape with a narrow cloaca and a round cystic lucency. Type 2B lesions were >10 mm in depth with a large dome shape extending down to a large articular surface defect. Type 3 lesions 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. (Reprinted from *Wallis* et al 2008 with permission from Equine Vet. J.).

There has been an evolution of knowledge regarding pathogenesis, differentiation of clinical significance, as well as treatment in recent years and this paper reviews that new information.

What is a subchondral cystic lesion- cysts, defects and flattening

In early literature SCLs of the MFC were cases where lameness was present and radiographs showed an obvious cystic lesion in the MFC. The radiographic appearance was characterized as one of two types: type I had 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 cyst to the articular surface at the condyle (*White* et al 1988). The cyst was also the cause of lameness in these early reports based on a history of chronic intermittent or constant lameness most often characterized by a rear limb, flat-footed gait and shortened cranial stride, a combination of other lesions and, increasingly, by the use of intra-articular analgesia (*Jeffcott* and *Kold* 1982).

In the first report of arthroscopic surgery to treat SCLs of the MFC in 1995, three types of SCLs were identified (*Howard* et al. 1995): type-I lesions were 10 mm or less in depth, appearing as shallow, saucer or dome-shaped defects in the weight-bearing surface of the MFC, type II lesions were more than 10 mm in depth and typically were domed, conical or spherical, type III lesions had a flattened or irregular contour of the subchondral bone at the distal aspect of the medial femoral condyle and were referred to as subchondral defects. Most recently *Wallis* et al. (2008) identified five different lesions as depicted in Figure 1, with type 1 lesions being the same as type I lesions as classified by *Howard* et al. (1995), type 2 lesions being divided into A and B based on the size of the opening at the articular surface, type 3 lesions being similar to type III lesions of *Howard* et al. (1995), but introducing the concept of condylar flattening in addition to a defect and type 4 lesions being a new description where there was no radiographic evidence of a cloaca in the subchondral bone plate (*Wallis* et al. 2008).

The acquisition of digital radiographs, increased ability to radiograph stifles and the introduction of repositories at both the Thoroughbred sales and cutting horse sales led to further scrutiny in examining radiographs and a spectrum of perception regarding the significance of flattening of the MFC. While the significance of SCLs in their various forms is usually obvious when there are clinical signs associated with the medial femorotibial joint, the prediction of unsoundness or soundness based on minor radiographic changes in yearlings is much more difficult. Indeed, radiographic lesions are not uncommon.

In a recent study, radiographs of yearlings and 2-year-old Quarter Horses were obtained from a radiograph repository

at a private farm. Of the 458 included horses, 408 (89.1%) had normal radiographic findings (*Contino* et al. 2009). Horses had the most abnormalities reported in the tarsus (304; 69.4%) followed by the stifle (202; 44.5%). Of the horses with stifle abnormalities, 188 (93.1%) were in the medial femoral condyles. In this study the grading system was modified again so that careful definition of subchondral flattening and defects in the MFC could be related to later performance (*Contino* 2009). In this system the MFC was graded as 0 when it was smooth and continuously convex, grade 1 when flattened without subchondral bone changes, grade 2 when small defects of changes in subchondral bone (but the change not extending through the subchondral bone) and grade 3 when there was shallow, crescent shaped, subchondral lucency extending through the subchondral bone equivalent to type 1 of *Wallis* et al (2008). These defects are illustrated in Figure 2. Of the 454 stifle radiographs examined, 266 (58.6%) were normal, 98 (21.6%) had flattening, 37 (8.1%) had a small defect, 30 (6.6%) had a crescent shaped lucency and 23 (5.1%) had a subchondral bone cyst. Follow-up examination to assess the significance of these lesions is currently progressing and indications at this stage are that there is no significance with grade 1 (flattening) change (*Barrett* and *McIlwraith*, unpublished data).

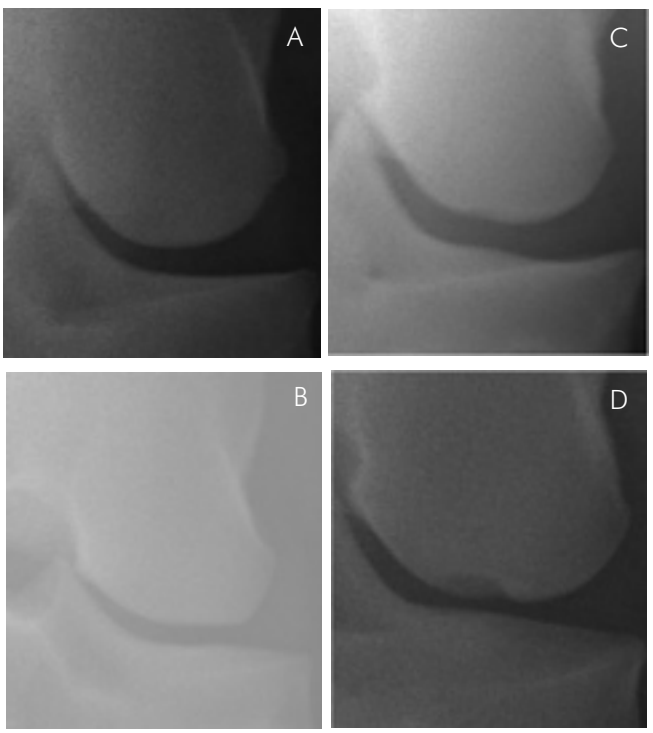

Fig. 2 Grades of MFC defects based on classification of *Contino* et al 2009. A. 0- normal MFC; B. grade 1; C. grade 2; D. grade 3 (see text for further details).

A study of survey radiographs taken at Thoroughbred yearling sales had examined the significance of subchondral lucencies. They defined subchondral lucencies at the distal aspect of the MFC as shallow, having a greater width (lateral to medial) than height (proximal to distal) and being crescent shaped. This was to distinguish them from MFC cysts as rounded lucencies within the condyle and usually seemed to communicate with the medial femorotibial joint (*Whitman* et al. 2006). They reported that 25 horses had MFC cysts (6 bilateral, 3 in left limb only, 16 in right limb only), and 27 horses had subchondral lucencies (8 bilateral, 6 left, 13 right). There was no significant difference in the ability to race between yearlings with MFC cysts at sale time (88% raced) and control horses (90% raced). They contrasted this with data from the report of *Sandler* et al. (2002) where 64% of horses with MFC cysts (after arthroscopic debridement) had raced. They considered the difference probably related to the horses having surgery, being lame and, although lameness examinations were not performed on the yearlings that had survey radiographs in this study, they considered it unlikely that many had clinical lameness. What was most interesting was the percentage of horses that had started at least one race from age 2-4 years of age, with 93% of horses with subchondral lucencies compared to 90% of control horses.

Pathogenesis

Proposed mechanisms for development of SCLs include osteochondrosis (*Rejno* and *Stromberg* 1978, *Poole* 1993) and trauma (*Verschooten* and *DeMoor* 1982, *Baxter* 1996). The author feels that with young horses, and, particularly, in bilateral cases that osteochondrosis is the probable cause. On the other hand, there has long been recognition that SCLs occur in older horses (*McIlwraith* 1998) and some have been identified associated with an initial articular defect (*Yovich* and *Stashak* 1989, *McIlwraith* 1993).

The trauma theory was initially explored experimentally by *Kold* et al. (1986) in which a subchondral bone cyst was induced 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 our laboratory did not duplicate this finding, but showed interesting data. A full-thickness linear defect was created in the articular cartilage of the MFC in 6 femorotibial joints in a group of exercised horses, and in all cases, there was no formation of SCLs. However, in the same study, concurrent elliptical cartilaginous and subchondral bone defects (5 mm diameter and 3 mm deep) in the MFC resulted in the development of cystic lesions in 5 of 6 horses (*Ray* et al 1996). This experimental finding, in addition to 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. Because of these findings, the author is cautious about debriding subchondral bone in the central weight-bearing area of the MFC when doing arthroscopic surgery.

More recent work from a collaborative study between the Orthopaedic Research Center at CSU and colleagues in Zurich has demonstrated that the fibrous tissue of SCLs in horses produce nitric oxide (NO), PGE2, and neutral metalloproteinases (MMPs). In addition, conditioned media of SCL tissue was capable of recruiting osteoclasts and increasing their activity (*von Rechenberg* et al 2000). Further work with in situ hybridization of sections of fibrous tissue of SCLs, as well as quantitative PCR with fresh frozen fibrous tissue and undecalcified sections of SCLs showed upregulation of mRNA of interleukin-1 (IL-1) and interleukin-6 (IL-6) in the fibrous tissue of SCLs (*von Rechenberg* et al 2001). From this research it was concluded that intralesional fibrous tissue of SCLs plays an active role in the pathologic process of bone resorption

occurring in SCLs in horses and may be partly responsible for the maintenance, slow healing rate and expansion of these lesions. This research was the basis of developing a technique of intralesional injection of triamcinolone acetonide (discussed below). As an 'intermediate hypothesis', it has been thought that compressive forces encountered in normal weight-bearing may encourage the formation of SCLs by contributing to the deformation of thickened cartilage previously compromised by a disturbance in the endochondral ossification process (*Kold* et al 1986, *Bramlage* 1993). Associated with this hypothesis 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 (*Nixon* 1990, *McIlwraith* 1993).

Diagnosis

Diagnosis is typically straight-forward, but the main diagnostic challenge is the significance, or potential significance, of lesions found on survey radiographs of yearlings. When associated with lameness, the degree of lameness varies from mild to severe and the onset can range from an insidious presentation of lameness to fairly acute (*Goodrich* and *McIlwraith* 2008). Commonly the lameness develops at the commencement of training in young horses (a typical example being a 2-year old Thoroughbred going into training), but in middle age to older horses the incidence of a traumatic event or correlation to ongoing intra-articular inflammation may be associated with the onset of the SCL (*von Rechenberg* et al. 1998). In a series of 41 horses affected with SCL, a degree of lameness was recorded grade 1/5 or 2/5 in most cases, but was recorded as 3/5 for 2 horses (*Howard* et al 1995). Slight or mild effusion was noticed in 15 femoropatellar joints, 2 medial femorotibial joints and thickening over the medial collateral ligament was detected in 2 joints. There have been attempts to correlate SCLs causing lameness with nuclear imaging. In a case report of bilateral SCLs in the MFC in a yearling foal, follow-up radiography and bone scintigraphy after surgical debridement revealed continued radiographic enlargement, but there was no demonstration of increased radiopharmaceutical intake (*Squire* et al 1992). In the right femorotibial joint of this case, it was demonstrated that persistent lameness was associated with the joint, but despite scintigraphy being unremarkable. Similarly in a study where SCLs were induced experimentally, scintigraphic uptake was inconsistent and most commonly negative despite progression of the size radiographically (*Ray* et al 1996).

When suspected, SCLs are most often confirmed radiographically (*McIlwraith* 1998). Occasionally SCLs may not be obvious using conventional radiography but imaging modalities such as nuclear scintigraphy, ultrasound, MRI or CT scan may be helpful in confirming the diagnosis. There have been 2 reports describing definition of SCLs in the distal limb, but MRI is not practical for the stifle (*Barrett* and *Zubrod* 2008, *Mair* and *Sherlock* 2008).

Treatment

The ideal treatment has long been the subject of debate. Success with conservative treatment (rest and anti-inflammatory agents) has been reported (*Jeffcott* and *Kold* 1982, *Stewart* and *Reid* 1982); however, older horses had a worse prognosis (*Stewart* and *Reid* 1982). In the author's experience, at the time some horses progress to athletic activity (including racing) without surgery, re-develop, particularly at the athletic level.

Debridement of SCLs through arthrotomy was reported in 1982 and in 42 of 51 horses; lameness was reduced or eliminated (*White* et al 1988). Of the 42 horses in this case series, 35 were sound. The conclusion was that arthrotomy was successful in reducing or eliminating lameness in 82% of all horses, which seems on reflection very high. Subsequent studies with arthroscopic surgery have not produced the same results. *Kold* and *Hickman* (1984) described the surgical treatment with arthrotomy with 10 SCLs in the MFC where cancellous bone graft was used in 9 cases and two-component acrylic bone cement used in the case of an extremely large cyst. Eight of the 10 cases were considered to make a satisfactory clinical recovery. More recently an experimental study examining the effect of compacted cancellous bone grafting in the healing of experimental subchondral bone defects (12.7 mm diameter and 19 mm deep) in the MFC concluded that there was no significant difference between grafted and ungrafted defects with respect to lameness, radiographic score or percent bone fill (*Jackson* et al 2000). Histologically grafted defects were characterized by the presence of dead graft and secondary cyst formation in 4 defects. Ungrafted defects filled with fibrous tissue and no cyst formation was identified. The author has never used cancellous bone grafts in the treatment of SCLs based on an initial apprehension of bone graft getting into the femorotibial joint, but based on both anecdotal experience, as well as the paper of *Jackson* et al. (2000), there appears to be little support for continued use of this technique.

Proceeding to arthroscopic surgery as the technique for debridement of SCLs was logical and in 1995 the results of arthroscopic surgery for SCLs of the MFC in 41 horses was published (*Howard* et al 1995). Surgical debridement performed via arthroscopy was the treatment for 37 lesions in 23 horses. Debridement followed by drilling of the defect bed was performed in 23 lesions of 18 horses. Complete follow-up information was obtained in 29 horses; 22 (56%) had a successful result and 17 (44%) had an unsuccessful result. In a separate analysis excluding horses with unsuccessful results because of factors not directly attributable to the SCL of the MFC (censored analysis), 23 of 31 (74%) horses had a successful result and 8 of 31 (26%) horses had an unsuccessful result. Interestingly, compared with Thoroughbreds and Arabians, Quarter Horses had a poorer prognosis for success. Follow-up radiographs were available for 14 horses and in 9 of these 14 horses the subchondral cystic lesion has enlarged after surgery. Post-operative cystic enlargement was also associated significantly with drilling of the lesion bed at the time of surgery. This technique was therefore subsequently abandoned.

Smith et al (2005) reported on a series of 85 horses with SCL of the MFC treated with arthroscopic debridement. In that study, 64% of horses age 0- 3 years returned to soundness, while only 34% of horse's age greater than 3 years returned to soundness. In another report of arthroscopic treatment of SCLs of the MFC by debridement in 150 clinically lame TB

horses with a total of 214 cystic lesions of the MFC, 86 of the 150 lame horses (57%) had unilateral lesions and 64 (43%) had bilateral lesions. A total of 96 (64%) of the horses that were operated on raced whereas 77% of their siblings raced (*Sandler* et al 2002). Twenty-eight percent (42) of the horses that were operated on raced as 2-year olds, 61% (79) of the horses raced as 3-year olds and 51% (55) of the horses raced as 4-year olds. The difference was established based on the width of the surface defect in that 91 (60.6%) of horses with ≤ 15 mm of surface debridement and 59 (39.3%) horses with > than 15 mm surface debridement started a race. The conclusion of this study was that the amount of cartilage surface affected seemed to be a better predictor of success than lesion depth. Recently we have reported on an association between debridement of medial femoral condylar defects and medial meniscus or meniscal ligamentous injury (*Hendrix* et al. 2010). While the study did not demonstrate this, it seems logical that a debrided edge of bone could cause trauma to the opposing cranial horn of the meniscus or cranial ligaments of the medial meniscus. Because of the studies of *Hendrix* et al (2010) and *Sandler* et al (2002) the conclusion has been made that retention of surface cartilage over SCLs is optimal in the treatment technique.

Based on the previous reported findings that the fibrous tissue of SCLs produced inflammatory mediators such as nitric oxide, prostaglandin E2 and neutral MMPs, which activate osteoclasts and enhance bone resorption (*von Rechenberg* et al. 2000), as well as knowledge that methylprednisolone acetate was used to treat unicameral bone SCLs in human subjects successfully (*Scagellitti* et al 1982), an arthroscopic technique involving injecting the fibrous tissue of SCLs with corticosteroid under arthroscopic guidance was developed (*Wallis* et al. 2008). Hypothesized advantages of this technique when it was developed earlier included a similar increased chance of success as compared to debridement, shorter convalescence, and lower risk of cystic enlargement and minimal disruption of the articular surface. A retrospective study of 52 horses with clinical and radiographic evidence of SCL in the MFC has been reported (*Wallis* et al. 2008). Thirty-five of the 52 (67%) cases were classified as successful, involving 73 SCLs of which 56 (77%) were classified as successful. Significantly more unilateral SCLs compared to bilateral (28/31: 90% SCLs) were classified as successful and bilateral (28/42: 67%). There was no significant association between age group (age ≤ 3 years versus >3 years) and outcome or cystic configuration and outcome, which contrasts with the findings of *Smith* et al (2005). There were significant differences in outcome based on the surgeon operating the case (but this seemed based on recommended lay-up time with shorter lay-up being more successful) and there was a negative association between pre-existing radiographic findings of osteophytes and negative outcome.

Results of a similar technique to that used in our study were presented by *Vandekeybus* et al. (1999) and showed favorable results. In that report the use of a combination of intra- and extra-articular approaches resulting in 11 of 21 horses returning to athletic use.

There was also a trend towards differences between different breeds and success ($p = 0.07$). Further investigation is needed to determine if there is a potential difference in prognosis for various breeds such as the American Paint Horse (only 5 of 10 of that breed were successful). It was also noted that 13 of 16 (81%) of young racing Thoroughbreds (8 being 2-year olds and 16 ≤ to 3-year olds) were classified as successful and 15 of 16 (94%) were classified as sound. Triamcinolone acetonide was used in 15 of 16 with the 16th horse being injected with triamcinolone diacetate due to availability of triamcinolone acetonide and remained lame.

Chondrocytes mixed with IGF-1 and fibrin implanted into subchondral cystic lesions have been described by Nixon in the authors arthroscopy book (*McIlwraith* et al 2005), as well as by *Fortier* and *Nixon* (2005). The chondrocyte-IGF-1-fibrin graft is placed over cancellous bone grafts. *Nixon* described clinically applying the chondrocyte grafting technique to OCD or SCLs of the fetlock in 14 horses and stifle in 43 horses and said the overall results have been generally good. However, some problems have been encountered with resorption of the cancellous bone graft and consequent sinking of the composite graft (*Nixon* personal communication 2005). The use of bone marrow-derived mesenchymal stem cells in fibrin (without bone grafting) has also been made successfully in 2 cases of SCLs that did not respond to intra-lesional injection of triamcinolone acetonide (*Frisbie* unpublished data 2008).

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