

Meniscal Disruption Associated with Septic Arthritis in 3 Neonatal Foals

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Abstract

Objective: To report clinical characteristics, surgical management, and medium-term outcomes of 3 Arabian neonatal foals with meniscal disruption associated with septic arthritis of the lateral femorotibial joint. **Methods:** Three neonatal Arabian foals with septic arthritis of the lateral femorotibial joint (LFTJ), were diagnosed with lateral meniscal (LM) tears, based on persistent lameness despite improving synovial parameters, ultrasound (US) findings (protrusion of meniscal tissue beyond the level of the condyles, with hypoechoic regions), contrast Computed Tomography findings, and confirmed on arthroscopy. Treatment included arthroscopic debridement and lavage of the joint with debridement of the meniscal tear. Postoperative care included systemic and intra-articular antimicrobials, based on culture and sensitivity results. Two of the foals received intra-articular injections of autologous mesenchymal stem cells. **Results:** Grade III meniscal tears were observed in the LFTJ of the affected joints of all foals, involving the meniscal body (n=3) and caudal horn (n=1). Purulent material within the torn tissue, was debrided with a synovial resector. Foal 1 was lame-free as a yearling. Foal 2 was lame at walk at 7.5 months and euthanatized due to poor prognosis. Foal 3 showed mild lameness at trot in a straight line at 6 months. Disruption of the LM continued to be visible on US in both foals at these time-points. **Conclusion:** Meniscal disruption and infection should be considered a differential in neonatal foals with persistent femorotibial septic arthritis. In such cases, the LM could be the primary nidus of infection.

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Abstract

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Methods: Three neonatal Arabian foals with septic arthritis of the lateral femorotibial joint (LFTJ), were diagnosed with lateral meniscal (LM) tears, based on persistent lameness despite improving synovial parameters, ultrasound (US) findings (protrusion of meniscal tissue beyond the level of the condyles, with hypoechoic regions), contrast Computed Tomography findings, and confirmed on arthroscopy. Treatment included arthroscopic debridement and lavage of the joint with debridement of the meniscal tear. Postoperative care included systemic and intra-articular antimicrobials, based on culture and sensitivity results. Two of the foals received intra-articular injections of autologous mesenchymal stem cells.

Results: Grade III meniscal tears were observed in the LFTJ of the affected joints of all foals, involving the meniscal body (n=3) and caudal horn (n=1). Purulent material within the torn tissue, was debrided with a synovial resector. Foal 1 was lame-free as a yearling. Foal 2 was lame at walk at 7.5 months and euthanatized due to poor prognosis. Foal 3 showed mild lameness at trot in a straight line at 6 months. Disruption of the LM continued to be visible on US in both foals at these time-points.

Conclusion: Meniscal disruption and infection should be considered a differential in neonatal foals with persistent femorotibial septic arthritis. In such cases, the LM could be the primary nidus of infection.

Keywords

Horse, Foal, Meniscus, Septic arthritis.

INTRODUCTION

In the adult horse, traumatic meniscal injuries result in lameness, and are amongst the commonly reported soft tissue injuries of the femorotibial joints (De Busscher *et al.* , 2006; Nemery *et al.* , 2016; Ribitsch *et al.* , 2018). There are relatively few documented cases of soft tissue injuries of the stifle in foals, including cases of popliteal tendonitis (Gabriel and Marta, 2020), an avulsion of the combined origin of the long digital extensor tendon and fibularis tertius from the extensor fossa (Blikslager and Bristol, 1994), a complete tear of the vastus medialis muscle with patellar luxation (Ogden *et al.* , 2019), and a complex stifle injury involving the medial meniscus and cranial cruciate ligaments (Santschiet *et al.* , 2020). These lesions were believed to be traumatic in origin with no septic processes reported.

The stifle is a commonly-reported site for sepsis in foals (Wright *et al.* , 2017; O'Brien *et al.* , 2021). Compared to other stifle compartments, sepsis of the lateral femorotibial joint (LFTJ) is relatively rare (Reeves, Trotter and Kainer, 1991; Vacek, Ford and Honnas, 1992; Hennessy *et al.* , 2012). It has been estimated that up to 1% of all foals, and 13% of those in neonatal intensive care, are affected by septic arthritis and osteomyelitis (McIlwraith, 1983; Martens, Auer and Carter, 1986; Richardson DW and Stewart S, no date). Neonates are susceptible to septic arthritis due to their immature immune systems (Richardson DW and Stewart S, no date). Partial or complete failure of passive transfer of immunity is a common risk factor for hematogenous spread leading to septic arthritis (Richardson DW and Stewart S, no date). Rapid growth of bones and joints is associated with an increased vascular supply, which is believed to facilitate infectious foci becoming established in the physes and surrounding structures (Firth, 1983; Richardson DW and Stewart S, no date). Septic arthritis is commonly divided into 3 broad categories (Richardson DW and Stewart S, no date). S-type infections, of the synovial fluid and membrane, are usually seen in very young neonates (Richardson DW and Stewart S, no date). E-type infections affect the epiphyseal bone adjacent to the articular cartilage, and typically present in older foals (Richardson DW and Stewart S, no date). Finally, P-types are mainly seen in older foals, involving the physes of long bones, and may extend to the joint capsule (Richardson DW and Stewart S, no date). One limitation of this classification system is the lack of inclusion of soft tissue structure involvement, aside from the joint capsule and synovial membrane.

To the authors' knowledge, there have been no reports of meniscal involvement in cases of septic arthritis of the stifle of the foal. Furthermore, although septic arthritis associated with osteochondral lesions in the femoral condyle of the stifle joint has been reported, to date histopathological findings of such lesions is missing (Haggett *et al.* , 2012).

The objective of this short case series was to report the clinical characteristics, surgical management, histopathological findings and medium-term outcomes of 3 Arabian neonatal foals with meniscal disruption associated with septic arthritis of the LFTJ.

Case History

Three neonatal foals presented with lameness of variable duration: Foal 1, a 7-day old filly, 36 hours' duration; Foal 2, a 7-day old filly, 96 hours' duration; and Foal 3, a 17-day old colt, 6 hours' duration. All foals had received systemic antimicrobials and nonsteroidal anti-inflammatories (NSAIDs) prior to referral.

Clinical Findings

Foal 1: Physical examination revealed pyrexia (39.6°C). Static examination revealed marked effusion of both tibiotarsal and LFTJs, including the subextensorius recess (SER). The foal showed a non-weight-bearing left hindlimb (LH) lameness (American Association of Equine Practitioners (AAEP) grade 5/5).

Foal 2: Physical examination revealed pyrexia (39.7°C). Static examination revealed marked joint effusion of the radio- and inter-carpal, tibiotarsal, and femoropatellar joints, bilaterally, as well as both LFTJs. The foal was lame on all four limbs at walk, with the LH being the lamest limb (AAEP grade 4/5).

Foal 3: Physical examination revealed pyrexia (39.9°C). Static examination revealed heat, swelling and pain on palpation over the lateral right (R) stifle and tibia, with moderate effusion of the SER. A right hindlimb (RH) lameness was seen at a walk (AAEP grade 4/5).

Radiography

Screening radiographs of all effused joints were taken on admission. Findings were unremarkable (Figures 1 b & c).

Ultrasonography (US) of LFTJ on admission

Foal 1: Marked effusion of the left (L) LFTJ and SER was seen with hyperechoic synovial fluid and fibrin formation (Figures 2 a & b). The LM was abnormal in shape, protruding beyond the level of the tibial/femoral condyles, with a horizontal hypoechoic line running through it (Figures 2 a & b).

Foal 2: Moderate effusion of the L LFTJ and SER was seen, with fibrin formation in the caudal pouch and surrounding the LM (Figure 1 a). The synovium and joint capsule were thickened. The LM was abnormal in shape, protruding beyond the level of the tibial/femoral condyles, with a hypoechoic region within it (Figure 1 a).

Foal 3: Marked effusion of the R LFTJ and SER was seen, with fibrin accumulation at the distal aspect of the SER. The LM was largely protruding beyond the level of the tibial/femoral condyles, with a heterogenous appearance and a hypoechoic line running through it.

Computed Tomography

Foal 2: Plain computed tomography (CT) of the L stifle revealed marked effusion of the LFTJ with irregularity of the subchondral bone of the lateral femoral condyle (LFC) and the lateral tibial condyle (Figure 1 d). Contrast arthrogram revealed disruption of the LM caudally, with lateral displacement (Figure 1 e). Disruption of the meniscofemoral ligament was present at its attachment to the caudal horn of the LM (Figure 1 f). The cranial lateral meniscotibial ligament (MTL) was also irregular in appearance.

Foal 3: Marked effusion was present within all three compartments of the R stifle. The proximal, lateral articular margin of the tibia was mildly irregular. The LM was diffusely abnormal with several tears in multiple orientations within the lateral and caudal aspects (Figure 3 b). A large, pyramidal shaped tear of the axial aspect of the caudal horn of the LM was present (Figure 3 c). The caudal lateral MTL was not clearly identified and a central region of hypoattenuation was present in the cranial lateral MTL (Figure 3 d).

Laboratory findings

On presentation, Foal 1 had a neutrophilic ($13.3 \times 10^9/L$) leukocytosis ($18.2 \times 10^9/L$). Foal 2 had a marked neutrophilic ($15.74 \times 10^9/L$) leukocytosis ($18.34 \times 10^9/L$), with hyperlactatemia (4.21 mmol/L), hypoproteinemia (46 g/L) and hypoalbuminemia (24.3 g/L). Hematology in Foal 3 was unremarkable, with a mild hypoproteinemia (58 g/L) and hypoalbuminemia (23.5 g/L). IgG levels $< 400 \text{ g/dL}$, indicating failure of passive transfer, were found in Foals 1 & 2.

Synovial fluid analysis at admission of the LFTJ of Foal 1 revealed a WCC of $133 \times 10^9/L$; 93% polymorphonuclear cells (PMNs) and total protein (TP) 37 g/L . Synoviocentesis for foal 2 was unsuccessful, with only gelatinous-like fibrin obtained. For Foal 3, LFTJ synovial fluid WCC was $103 \times 10^9 \text{ cells/L}$, 97.1% PMNs, and TP 33 g/L .

LFTJ synovial fluid culture results were negative for bacterial growth in Foal 1. In Foal 2, *Micrococcus luteus* was cultured from the fibrinous tissue obtained; in Foal 3, *Staphylococcus schleiferi* was cultured; both had a broad antimicrobial sensitivity pattern.

Treatment

High-volume arthroscopic lavage and debridement of the affected LFTJs (including SERs) was performed. A cranial approach was taken and a synovial resector was introduced between the lateral patellar and lateral collateral ligaments to clear the visual field. Examination of the menisci revealed grade III tears in all cases (Figures 2 d & 3 f). In Foal 1, tearing of the cranial MTL was also apparent. In all cases, the meniscus appeared prolapsed and displaced craniolaterally (Figure 2 c; Figures 3 e & f). Fibrinous deposits were present adjacent to the tears, with purulent material apparent within the torn fibers (Figure 3 e). The unhealthy tissue and torn fibers were removed using a synovial resector. In all 3 Foals, the caudal aspect of the LFTJs were also arthroscopically examined, and in Foal 2, evidence of meniscal tearing could also be seen in the caudal horn. Where multiple joint involvement was present, other joints were treated similarly, with high volume arthroscopic lavage and debridement.

Postoperative treatment included a combination of broad-spectrum antimicrobials (parenteral and IA), based on culture and sensitivity results, where available; analgesia (NSAIDs and opioids, as indicated), and gastro-

protectants. Hyperimmune plasma was administered to Foals 1 & 2. Response to treatment was monitored based on comfort levels, US findings, and synovial fluid analysis.

Foal 2 and 3 required a second arthroscopic lavage and debridement of the L LFTJ 14 days, and 3 days respectively, after the initial surgery to remove further fibrin accumulations around the LM. In both cases, progression of the tearing and meniscal protrusion/displacement could be seen.

In Foals 2 & 3, following resolution of the septic arthritis and recovery, US and radiographic-guided bone marrow aspirates were taken from the sternbrae under general anesthesia, for production of autologous bone marrow-derived mesenchymal stem cells (BMSCs) (Figure 5 a). Briefly, the sternum was aseptically prepared, and the intersternbral spaces identified using US. A stab incision with a No. 11 scalpel was performed through the skin and a Jamshidi biopsy needle (11 gauge, 10 cm) introduced into the sternbrae, using digital x-ray to guide depth of needle introduction (Figure 5 a). A 60 ml syringe, preloaded with 1000 IU of heparin was used to aspirate the bone marrow. BMSCs were isolated aseptically and expanded in high glucose Dulbecco's modified eagle's medium (DMEM) supplemented with 10% v/v fetal bovine serum and 100 U/mL penicillin –100 mg/mL streptomycin (all Gibco, Biosciences, USA) at 20% pO₂ (Smith *et al.*, 2013). Following colony formation, BMSCs were obtained with accutase (Gibco, Biosciences, USA), counted (Countess II FL Automated Cell Counter, Thermo Fisher, USA), seeded at density of 5000 cells/cm² and expanded until Passage 3. A final cell suspension of 2-4 million cells per ml was obtained and suspended in saline for final injection in the patient.

Foal 2 received 3 US-guided IA injections of 10 million autologous BMSCs, in the L LFTJ, at 44 d, 65 d, and 97 d post-operatively (Figure 5 b). Foal 3 received 3 US-guided IA injections of autologous BMSCs, in the R LFTJ, at 19 d, 51 d and 86 d post-operatively.

Outcome

Foal 1

Foal 1 was discharged 23 days after admission. She was reported to be without lameness as a yearling and lost to follow-up thereafter.

Foal 2

At 6 months post discharge, Foal 2 was lame at a walk on the LH (AAEP 4/5). There was palpable thickening and fibrosis over the LM (Figure 4 c). Left gluteal muscle atrophy was apparent with resentment of passive global flexion of the limb. US revealed synovial effusion within the LFTJ and SER (Figure 4 b). The LM was displaced laterally, with an irregular outline, and areas of increased echogenicity suggestive of mineralization (Figure 4 b). Radiographs revealed multiple coalescing radiolucencies within the lateral femoral condyle (LFC), which appeared flattened (Figure 4 d). Mineralization was visible in the soft tissues in the region of the LM. A small radiolucent area was evident on the lateral tibial plateau, surrounded by sclerosis (Figure 4 e). Arthroscopic exploration of the L LFTJ and femoropatellar joint (FPJ) was performed under general anesthesia. The cranial horn of the meniscus was enlarged and fused with the joint capsule. It was displaced cranially, rigid and fibrotic on palpation. Three subchondral bone cysts with deep cloacae could be appreciated within the LFC. The lateral tibial plateau lesion could not be visualized. After a further 7 weeks, the filly's comfort levels had not improved any further. Repeat radiographs revealed significant deterioration. The filly was humanely euthanatized and submitted for post-mortem investigation.

Post-mortem macroscopic findings : Increased viscosity and volume of articular fluid were found in the left LFTJ. Edematous thickening of the joint capsule was observed with hypertrophy of synovial villi and increased yellow-ness of the synovial fluid (Figure 6 a). The LM showed marked roughness and edematous loosening/malacia and was significantly thinner than the medial meniscus (Figure 6 b). Examination of the proximal tibia revealed diffuse gray-white lesions in the cancellous bony tissue immediately below the articular cartilage, as well as multiple focal erosions on the caudal articular surface of the lateral condyle of the proximal tibia adjacent to the meniscus (Figure 6 c).

Post-mortem histological findings : Histological evaluation focused on the articular capsule of the left LFTJ, LM, articular cartilage of the proximal tibia, and the subchondral bone tissue beneath. Diffuse villous proliferation of the capsular synovium was observed. In the superficial layer of the synovium, slight hyperplasia of the epithelium covering the superficial layer of the synovium was noted (Figure 6 d). In the sub-synovial stroma, infiltration of inflammatory cells (neutrophils, lymphocytes, macrophages with hemosiderin deposition, and plasma cells), interstitial edema, and capillary angiogenesis were observed (Figure 6 d). In the LM, irregular arrangement of collagen fibers, multifocal coagulation necrosis, fibrin deposition in the collagen fiber tissue, infiltration of inflammatory cells (neutrophils, lymphocytes, and macrophages), and capillary angiogenesis, were detected (Figure 6 e).

In the lateral condyle of the proximal tibia, surface irregularities of the superficial layer of the articular cartilage were found, associated with the multifocal erosions seen grossly on the articular cartilage surface. In addition, septic cartilage canals, characterized by neutrophil/macrophage infiltration into the articular cartilage canals, and the presence of fibrin-like material within the canals were found (Figure 6 f). Multifocal-to-continuous bone necrosis/hemorrhage/inflammatory cell infiltration, and fibroblasts as well as angiogenesis, were present at the periphery of the bone tissue, immediately below the articular cartilage. These lesions were not detected within the deeper epiphysis.

Foal 3

At 6 months post discharge, Foal 3 showed a mild AAEP 3/5 RH lameness which was positive to global flexion. There was minimal palpable effusion of the right stifle, however there was a firm palpable swelling over the LM (Figure 4 c). Radiographs revealed mild soft tissue thickening over the R LFTJ. A radiolucent area, surrounded by a rim of sclerosis, could be seen on the lateral aspect of the tibial plateau, indicating development of a possible cystic formation (Figure 4 d). There was mild remodeling of the lateral tibial plateau and femoral condyle, which was flattened in appearance (Figure 4 e). Mineralization within the soft tissue in the region of the LM could be appreciated. Moderate hypoechoic effusion of SER was seen on US. The LM continued to protrude beyond the level of the condyles. Previously seen clefts were visible but had reduced in size, evidence of mineralization could be seen within the body of the meniscus which retained its triangular shape (Figure 4 b). Remodeling of the LFC could be appreciated, and a zone of irregular subchondral bone could be seen on the tibial plateau within the joint space.

Discussion

This case series describes meniscal involvement in 3 foals with septic arthritis of the LFTJs. To the authors' knowledge, this form of meniscal involvement has not been reported heretofore. Conventional classification systems of septic arthritis and osteomyelitis in horses do not make provision for involvement of IA soft-tissue structures, such as the menisci (Richardson DW and Stewart S, no date). This highlights the need for an additional classification subtype, to extend the S-type infection from involving not only the synovial membrane and fluid, but also to other soft tissue structures within the joint.

The progression of the pathophysiology in these cases is unclear. Given that the meniscal lesions were most marked at the site of attachment with the capsular synovium, it was theorized that extension of purulent inflammation occurred from the joint *via* the vascular network of the capsular synovium supplying the meniscus at this location (Arnoczky and Warren, 1982). Purulent osteomyelitis was found at the junction between articular cartilage and subchondral bone (Firth and Goedegebuure, 1988). The presence of septic canals below erosive articular cartilage lesions, in the absence of any lesions in the underlying epiphysis, suggests that the inflammation spread from the joint to the subchondral bone through septic cartilage canals (Wormstrand *et al.* , 2018). Therefore, the authors believe that, due partial/complete failure of passive transfer, the original IA septic focus established within the synovial membrane and fluid, and subsequently extended to the meniscus, articular cartilage and subchondral bone, leading to their disruption.

From the current series, it appears that a septic meniscus can act as a persistent nidus of infection perpetuating septic arthritis, and also leading to septic osteomyelitis and subchondral bone cyst formation. In these cases, once infection became established within the menisci, arthroscopic lavage and debridement of

IA fibrin alone was inadequate to address the sepsis within the meniscus, which did not resolve until the purulent focus within the meniscus itself was debrided. For this reason, in cases where foals with septic arthritis of the LFTJ relapse following arthroscopic debridement and lavage, the authors advocate paying close attention to the appearance and position of the meniscus using US, and CT where available, to examine for evidence of displacement or disruption. Meniscal disruption should also be considered in cases where synovial parameters resolve post-operatively following arthroscopic debridement and lavage for LFTJ septic arthritis in foals, however lameness persists for no apparent reason.

It has been reported that sepsis of the LFTJ usually occurs as an extension of infection from the FPJ, as the two communicate in 1-17% of cases (Reeves, Trotter and Kainer, 1991; Vacek, Ford and Honnas, 1992; Hennessy *et al.*, 2012). In this case series, only one of the three foals was presented with additional joints affected in the stifle (Foal 2), with the other two foals having only LFTJ involvement. Furthermore, in adults, meniscal tears occur three times more often in the medial meniscus than the LM (Walmsley, Phillips and Townsend, 2003; Cohen *et al.*, 2009), which was not the case in this series.

In adults, advanced imaging of the stifle is challenging due to size limitations, and thus arthroscopy has traditionally been relied on for the diagnosis of pathology of menisci and meniscal ligaments (McIlwraith CW, Nixon AJ, Wright IM, no date). US does not always accurately predict meniscal tears in adults (Cohen *et al.*, 2009). The small size of the patients in this series allowed for CT to be performed in 2 of the foals, providing valuable additional information regarding IA soft tissue involvement in these cases.

In adult horses, only limited portions of the meniscus are visible with arthroscopy, requiring examination of the cranial and caudal pouches of the femorotibial joints from separate approaches (McIlwraith CW, Nixon AJ, Wright IM, no date). Grade 3 tears, which extend beneath the femoral condyle, are typically inaccessible (Walmsley, Phillips and Townsend, 2003; McIlwraith CW, Nixon AJ, Wright IM, no date). Furthermore, according to McIlwraith *et al.*, accessibility to the LFTJ can be difficult due to the IA location of the long digital extensor and popliteal tendons (McIlwraith CW, Nixon AJ, Wright IM, no date). In the current series, the authors found the foal LFTJs relatively easy to maneuver within, most likely because of laxity due to their young age, which facilitated better access to a greater extent of the tears for debridement.

Regenerative therapies, including IA platelet rich plasma (PRP) and interleukin-1 receptor antagonist protein (IRAP) have been proposed as adjunctive therapies for meniscal injuries (Fowlie JG, Richardson DW, Ortved KF, no date). A clinical study in adult horses has shown that IA administration of BMSCs may promote meniscal regrowth and decrease the subsequent development of osteoarthritis (Ferris *et al.*, 2014). Similarly, caprine models have demonstrated significant regeneration of medial meniscal tissue, with implanted cells shown to be incorporated within newly formed tissue, following IA delivery of adults BMSCs (Murphy *et al.*, 2003). The use of BMSCs for treatment of similar injuries in foals has not been previously reported until now.

Prognosis for grade III meniscal tears in adults horses is poor, with only 6% of horses returning to previous athletic function (Walmsley, Phillips and Townsend, 2003). In adults, grade III tears preclude complete removal of torn tissue (McIlwraith CW, Nixon AJ, Wright IM, no date). The results of the current series suggest that the short-medium term prognosis in foals with meniscal disruption associated with septic arthritis, is also guarded to poor for return to soundness, with only 1 Foal lame-free out of 3 after 1 year. The prognosis in foals may be more favorable than adults, due to their greater healing potential, low weight and easier access to the tears by arthroscopy due to their small size, allowing more complete debridement. It has been hypothesized that dystrophic mineralization of the meniscus, visible on radiographs and US, may lower the prognosis for return to athleticism (Fowlie JG, Richardson DW, Ortved KF, no date). Early signs of mineralization within the soft tissue lateral to the LFTJ, and within the LM, in Foals 2 and 3, seen at 6 months, may indicate a poorer long-term prognosis for soundness.

Conclusion

Meniscal disruption and infection should be considered in neonatal foals with persistent femorotibial septic arthritis. In such cases, the meniscus could be a nidus of infection. Meniscal disruption in neonatal foals,

associated with LFTJ sepsis, is associated with a guarded to poor short-medium term prognosis for return to soundness.

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FIGURE LEGENDS

Figure 1: US images of Foal 2 (a) on admission with moderate effusion (*) and fibrin formation of the L LFTJ. The LM was abnormal in shape, protruding beyond the level of the tibial/femoral condyles, with a diffuse hypoechoic region within it (arrowhead). Screening radiographs of the L stifle of Foal 2 were taken on admission, findings were unremarkable (b & c). Plain CT of the L stifle of Foal 2 revealed marked distension

of the LFTJ with irregularity and sclerosis of the subchondral bone of the LFC and the lateral tibial condyle (d). Contrast CT-arthrogram revealed disruption of the LM caudally, with lateral displacement (e). At its attachment to the caudal horn of the LM, disruption of the meniscofemoral ligament could be seen (Figure 2 f). F = lateral femoral condyle; POP = popliteus tendon; T = lateral tibial condyle.

Figure 2: US images of Foal 1 on admission (a) with marked effusion and fibrin formation within L LFTJ (*). The LM was abnormal in shape, protruding beyond the level of the tibial/femoral condyles (covered by epiphyseal cartilage), with a horizontal hypoechoic line running through it (arrowheads). Figure (b) shows normal R LFTJ for comparison. Figure (c) shows arthroscopic image of the cranial aspect of the L LFTJ of Foal 1, the LM can be seen prolapsed and displaced cranio-laterally, with fibrin accumulation (*), meniscal hyperemia and synovial proliferation. Figure (d) shows probe revealing Grade III tear of LM (#) extending beyond the margin of the lateral femoral condyle. F = lateral femoral condyle; T = lateral tibial condyle.

Figure 3: CT images of Foal 3, left (normal) stifle (a) for comparison with right (abnormal) stifle (b). The LM was diffusely abnormal with multiple tears in multiple orientations, primarily within the lateral and caudal aspects (Figure 3 b). A large, pyramidal shaped tear of the axial aspect of the caudal horn of the LM was present, resulting in marked irregularity of the axial margin of the meniscus (Figure 3 c). There was a small tear of the distal substance of the cranial cruciate ligament. The margin of the caudal cruciate ligament was minimally fibrillated (Figure 3 d). Figure (e) shows an arthroscopic image of R LFTJ of Foal 3, the LM can be seen prolapsed and displaced cranio-laterally, with fibrin accumulation. Figure (f) shows Grade III tear (arrow) of LM extending beyond the margin of the lateral femoral condyle (F – femoral condyle; T – tibia).

Figure 4: US images of Foal 3 R LFTJ 3 days after initial presentation (a) and 6 months after initial presentation (b). US 6 months after initial presentation revealed increased synovial effusion within the LFTJ (*) (Figure 4 b). The LM position continued to be displaced laterally and was abnormal in appearance with areas of increased echogenicity suggestive of forming mineralization (arrows) (Figure 4 b). There was a marked palpable thickening and fibrosis over the region of the LM (Figure 4 c). Radiographs of the L stifle 6 months after initial presentation showed the LFC was flattened in appearance with remodeling of the lateral tibial condyle (Figure 4 d). A small radiolucent area (4 x 3 mm) was evident on the lateral tibial plateau, surrounded by a rim of sclerosis (arrow) (Figure 4 e).

Figure 5: Radiograph showing a Jamshidi needle approaching the sternebra for bone marrow aspiration performed under general anesthesia. This is the first step for production of autologous bone marrow-derived mesenchymal stem cells (BMSCs) (a). Foals 2 & 3 received 3 US-guided injections of autologous BMSCs, in the SER of the affected LFTJs post initial discharge. T = tibia.

Figure 6: Gross post-mortem images of Foal 2 showing left stifle joint (a). Image showing ventral/tibial surface of the menisci of L LFTJ (b), with medial meniscus (left side) and LM (right side). The LM showed marked thinning with loosening of collagen fibers, especially caudally (b). Figure (c) shows the posterior articular surface of the lateral condyle of the proximal tibia, revealing the presence of diffuse gray-white lesions in the subchondral bone, immediately below the articular cartilage. Figure (d) shows synovitis with diffuse villous proliferation and infiltration of inflammatory cells in the villous stroma. Figure (e) shows meniscitis with irregular arrangement of collagen fibers, infiltration of inflammatory cells and capillary angiogenesis. Figure (f) shows a septic cartilage canal with inflammatory cell infiltration and deposition of fibrin-like material within the cartilage canal of the articular cartilage. Bar=200 µm.





