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Unusual Medial Femoral Condyle Lesion in a Neonatal Quarter Horse Foal with MYH1-Associated Myopathy

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Case Report

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Abstract

Lesions of the medial femoral condyle (MFC) in neonatal foals represent a diagnostic challenge due to the variability and complexity of clinical, laboratory, and imaging findings. This case report describes an eight-day-old Quarter Horse foal presenting with acute, severe hindlimb lameness. Radiographic and ultrasonographic examination identified a lesion of the MFC characterised by subchondral irregularity and associated with the presence of a mineral-dense fragment, with medial collateral ligament (MCL) thickening and synovial reaction. Based on the clinical and imaging findings, the main differential diagnoses included osteochondral necrosis (ON) of the MFC and an enthesial avulsion injury at the femoral origin of the MCL. The foal had a concurrent history of omphalitis and was heterozygous for the MYH1 E321G mutation, suggesting potential predisposing factors including a possible haematogenous insult and altered neuromuscular function. A definitive diagnosis could not be established due to the absence of advanced imaging, arthroscopic evaluation, and histopathological confirmation. The foal was euthanised due to the severity of clinical signs and poor prognosis. This case highlights the diagnostic complexity of MFC lesions in neonatal foals. It also expands current knowledge by describing a similar presentation in a Quarter Horse foal, a breed in which such lesions have not been previously reported. The findings emphasise the importance of a comprehensive diagnostic approach integrating clinical, imaging, and genetic factors.

1. Background

The equine stifle is one of the most anatomically and functionally complex joints, whose proper function relies on the integrity of its supporting ligaments and soft tissues. The femoropatellar, medial femorotibial (FT), and lateral FT compartments act in a coordinated manner, functioning as a single integrated biomechanical unit [1–3]. Within this complex system, the medial femoral condyle (MFC) represents a critical load-bearing structure, particularly in foals, where incomplete skeletal maturation and ongoing endochondral ossification may predispose this region to a variety of developmental, infectious, and traumatic disorders [4]. A wide spectrum of pathologies affecting the MFC has been described in foals, including osteochondrosis, septic synovitis, physitis, osteomyelitis, and subchondral bone lesions [5]. In addition to these conditions, traumatic injuries involving periarticular soft tissues and ligaments may also occur, although they are less frequently reported in foals. For example, trauma to the lateral femorotibial joint (FTJ) has been described as resulting in a complex combination of lesions involving both osseous and soft tissue structures, including less commonly evaluated components such as the popliteal tendon. [6]. Among condylar disorders, osteochondral necrosis of the medial femoral condyle (ON) of the MFC has recently been recognised as an uncommon but severe condition, typically associated with acute onset of hindlimb lameness and joint effusion. The aetiology and pathogenesis of ON MFC remain poorly understood, with proposed mechanisms including developmental disturbances, vascular compromise, and septic processes [7]. Affected foals generally present between 2 weeks and several months of age, most commonly around 3–4 weeks [5].

The complexity and overlapping features of these lesions often make diagnosis challenging, frequently requiring a multimodal approach that may include advanced imaging techniques [5, 6]. Clinical findings are typically nonspecific, and both radiographic and ultrasonographic features may overlap among developmental, infectious, and traumatic conditions [8], making accurate lesion characterisation particularly challenging in foals.

In addition to local structural abnormalities, systemic or neuromuscular conditions may further influence the development and expression of musculoskeletal disease. Musculoskeletal abnormalities in foals are largely attributed to genetic predisposition [9]. Quarter horse (QH) and related breeds, are particularly susceptible to several muscle genetic disease for which testing is available, including hyperkalemic periodic paralysis (HYPP), Glycogen Branching Enzyme Deficiency (GBED) and Polysaccharide storage myopathy 1 (PSSM1) [10–12]. To the authors knowledge, relatively limited literature is available about clinical presentation and therapeutic management of genetic disease in neonatal QH foals, mainly because of the short clinical course and poor prognosis [13]. Myosin Heavy Chain Myopathy is a co-dominantly inherited myopathy associated with a missense mutation (E321G) in the *MYH1* gene. It encodes the myosin filament's heavy chain in type 2X myofibers and has been linked to immune-mediated and non-exertional muscle disorders [10, 14]. Although *MYH1*-associated myopathy typically presents with clinical signs such as muscular weakness or stiffness, subclinical cases have also been recognised. These mild or latent forms may subtly affect neuromuscular control and locomotor performance [10, 12, 15].

Despite the range of medial femoral condyle pathologies described in foals, some lesions remain poorly defined and may be challenging to interpret, particularly in neonatal animals. In addition, the interaction between complex orthopaedic lesions and underlying myopathic conditions has not been clearly defined.

This report describes a complex medial femoral condyle lesion in a neonatal Quarter Horse foal with MYH1-associated myopathy, and discusses the diagnostic challenges associated with lesion characterisation and interpretation.

2. Case presentation

2.1 Ethical approval

Owners provided informed consent for the veterinary treatment and for the foal data to be used for future publication.

2.2 Case History and Clinical Presentation

An eight-day-old QH colt was hospitalized for evaluation of severe acute-onset lameness of the left hindlimb and suspected flexural deformities. The mare was a six-year-old Trotter recipient of an intracytoplasmic sperm injection (ICSI) embryo, and the donor was a QH mare known to be heterozygous for the *MYH1* E321G mutation. The foal was born following an uneventful eutocic delivery, and the initial

clinical examination at birth was unremarkable. No episodes of trauma, slipping, or abnormal maternal behaviour had been observed, and the colt had remained confined to a box stall since birth. At five days of age, difficulty in standing and progressive lameness of the left hindlimb were noticed. Due to the worsening clinical condition and the development of stifle swelling, the colt was subsequently hospitalised in an Equine Perinatology Unit.

On presentation, the foal was bright and responsive, with a good suckle reflex. Clinical examination revealed tachycardia (140 bpm, RR 80-100bpm), mild tachypnoea (44 breaths/min, RR 20-40 breaths/min) and mild dilated nostrils. Rectal temperature (38.1°C), mucous membrane and capillary refill time were within normal limits. Auscultation of the thorax and abdomen was unremarkable. The umbilical stump was dry on palpation, but its volume was increased for the foal's age.

A marked distension of the left stifle was present, associated with diffuse heat, pain on palpation, and severe functional impairment of the limb. The colt exhibited a non-weight-bearing lameness and maintained the limb in a flexed position, consistent with grade 5/5 of the AAEP scale (*Figure 1)* [16]. The right stifle appeared mildly distended on palpation but was not painful and did not affect gait. No external wounds or skin lesions indicating trauma were detected.

A bilateral flexural deformity of the forelimbs was noted, with mild swelling along the lateral aspect of both carpal regions. These findings were consistent with rupture of the common digital extensor tendons (*Figure 1)*. Additionally, physical examination revealed mild asymmetry and reduced development of the gluteal and epaxial muscle masses, while the semimembranosus and semitendinosus muscles appeared relatively preserved.

On admission, haematological evaluation revealed mild normocytic normochromic anaemia (HCT 25.8%, RR 28-43, haemoglobin 8.8 g/dl, RR 10-15 g/dl) and a leukocytosis (13.5 × 10^3/μL, RR 5.2.–11.9) characterised by neutrophilia (10.9 × 10^3/μL, RR 3.9-9.0. Platelet count was within normal limits. Serum biochemistry showed increased Creatin Kinase (CK) (499 U/L, RR 46-208 U/L) and increased serum amyloid A (SAA) (459 μg/mL, RR 0–10). Fibrinogen was within normal limits (3.7 g/L, RR 1.6–4.1). IgG concentration measured at admission (1.796 mg/dL) confirmed adequate passive transfer of immunity.

Further laboratory investigations included genetic testing. Genetic testing was performed by Polymerase Chain Reaction (PCR) on genomic DNA extracted from EDTA-anticoagulated whole blood and approximately 20 mane hairs with intact roots, submitted to a diagnostic laboratory (LABOKLIN, Bad Kissingen, Germany).

Ultrasonographic evaluation of umbilical remnants was carried out using a Philips CX50 ultrasound system (Philips Healthcare, Eindhoven, The Netherlands) equipped with a 13-3 MHz linear transducer at six short-axis (transverse) views of the umbilical structures at pre-determined points, according to a standardized protocol [17]. The ultrasound (US) of the umbilical remnants revealed an increased diameter of the umbilical vein (2 cm immediately cranial to the stump), with heterogeneous content;

increased diameter and heterogeneous echotexture of the right umbilical artery wall and increased overall diameter of the umbilical arteries and urachus (2.19 x 0.92 cm). The US evaluation was consistent with abnormal regression of the umbilical remnants, omphalophlebitis and right-side arteritis.

2.3 Diagnostic investigations and findings

2.3.1 Synovial fluid analysis

Arthrocentesis of the left medial FTJ was performed at admission under aseptic conditions.

The aspirated synovial fluid was slightly haemorrhagic and turbid, with increased viscosity. Laboratory analysis showed total nucleated cell count (TNCC) 2.417 cells/μL (RR 0–500) and total protein 3.5 g/dL (RR <2.0). Cytological examination revealed a revealed numerous red blood cells, predominance of non-degenerate neutrophils (approximately 80%), scattered mononuclear cells, and the absence of intracellular or extracellular bacteria. Aerobic and anaerobic bacterial cultures yielded no growth after 48 hours, ruling out septic arthritis.

2.3.2 Radiographic examination of the stifle

Radiographic examination of the left stifle was performed using caudal 60° lateral–craniomedial oblique (Cd60°L–CrMO) and caudal 10° proximal–craniodistal oblique (Cd10°Pr–CrDiO) projections, acquired with a portable X-ray generator and a direct digital system (Fujifilm, Tokyo, Japan). Images were obtained with a focus–film distance of 100 cm using standard exposure parameters (90 kVp, 3.2 mAs).

On the caudocranial (CdCr) projection, moderate soft-tissue distension was observed along the medial aspect of the femorotibial joint, most prominent at the medial recess, consistent with periarticular effusion. The medial femoral condyle (MFC) exhibited marked cortical and subchondral irregularity, extending from the intra-articular surface distally towards the physeal line. Within this region, a poorly marginated radiolucent area with a short, irregular transition zone was evident. Along the caudal aspect of the MFC, a distinct mineral-dense opacity was identified, closely opposed to the parent bone and measuring approximately 10–12 mm (short axis ≈3–4 mm) (Figure 2A).

On the Cd60°L–CrMO projection, the fragment was projected over the caudomedial aspect of the MFC. The cortical outline of the condyle appeared disrupted and mildly thickened. The surrounding soft tissues showed a diffuse increase in opacity consistent with local oedema. The distal femoral physis appeared normal in thickness and definition for age, with no evidence of asymmetry, widening, or step formation (Figure 2B). The subchondral contour of the MFC appeared irregular but remained partially continuous, and no additional mineralised bodies were identified within the joint space. The lateral condyle, femoropatellar joint, and tibial plateau were unremarkable.

Follow-up radiographs obtained six days later showed persistence of the fragment, with unchanged size and position, no evidence of further displacement, and absence of callus formation or periosteal new bone proliferation.

2.3.3 Ultrasonographic investigation of the stifle

A comprehensive ultrasonographic examination of the dorsomedial aspect of the left stifle was performed using a Philips CX50 ultrasound system (Philips Healthcare, Eindhoven, The Netherlands) equipped with a linear 7.5 MHz transducer (focal depth: 4 cm). The foal was examined in alternating lateral recumbency (left and right), to allow complete visualisation of the medial FT compartment. Longitudinal and transverse scans were obtained along the medial FTJ, from the distal femoral metaphysis to the proximal tibial metaphysis, and compared with the contralateral limb.

The medial FTJ recess was markedly distended by anechoic synovial fluid containing numerous fine echogenic strands and floating synovial villi, consistent with marked synovial reaction. The joint capsule appeared diffusely thickened, displaying loss of the normal, sharply marginated capsular outline (*Figure 3A and 3B*).

The medial collateral ligament (MCL) was diffusely thickened and hypoechoic throughout its course, with partial loss of normal fibrillar architecture, most evident in the proximo-cranial portions. The proximal ligament fibres, at the femoral insertion, appeared irregular and poorly defined relative to the adjacent cortical surface. Comparison with the contralateral limb confirmed increased cross-sectional area and reduced echogenicity of the left MCL.

The cranial and abaxial margins of the medial meniscus were smooth and sharply defined, with homogeneous echogenicity and normal triangular configuration; no discontinuity, deformation, or hypoechoic defects suggestive of meniscal tearing were identified (*Figure 3C)*.

The cortical bone surface of the MFC was severely irregular with loss of the normal smooth hyperechoic bone–soft tissue interface and increased subchondral echogenicity, extending caudally and proximally along the medial femoral epicondyle. A hyperechoic focus adjacent to the cortical margin corresponded to the osseous fragment identified radiographically (*Figure 3D*).

The distal femoral physis was visible as a regular hypoechoic band bordered by hyperechoic margins, consistent with age, with no evidence of thickening, irregularity, or step formation (*Figure 3E*).

The contralateral stifle was unremarkable, supporting the unilateral nature of the lesion.

2.4 Treatment and follow-up

The colt remained hospitalised for 11 days. On day 2 of hospitalization, based on the umbilical ultrasonographic findings, surgical intervention with omphalectomy was performed. Broad-spectrum antibiotic was administered, ampicillin (20 mg/kg IV q6h) and amikacin (20 mg/kg IV q24h) . Non-steroidal anti-inflammatory drugs (NSAIDs), (flunixin meglumine,1.1 mg/kg IV q12h) was administered, with gastroprotection based on clinical judgment.

Follow-up analysis performed six days later documented a marked increase in CK (1.732 U/L, RR 69-272 U/L) and AST 335 U/L (RR 112-266). SAA had decreased to 7 µg/mL (RR 0-10), consistent with resolution of the acute-phase response. Repeat haematology showed normalisation of white blood cell count (7.9 × 10^3/µL, RR 5.5–12.5) and a stable red cell profile (HCT 27.8%).

Because radiographs demonstrated a small, non-displaced avulsion fragment without radiographic signs of instability or articular incongruity, surgical intervention was not indicated, and conservative treatment was chosen. The foal was maintained on strict stall confinement on deep straw bedding to minimise limb loading. Periodic cold therapy and gentle manual mobilisation of the limb were employed to improve comfort and prevent joint stiffness.

In addition to the stifle lesion, the foal presented with bilateral flexural deformity of the forelimbs. To correct this, lightweight forelimb casts were applied bilaterally for 72 hours. The casts effectively improved the degree of flexion deformity, and carpal alignment appeared normal before discharge.

The stifle effusion decreased, and the colt became progressively more comfortable, showing partial weight bearing within the first week. At the time of discharge, lameness had improved from non–weight-bearing (AAEP 5/5) to grade 4/5, and the colt was able to bear partial weight on the affected limb while standing. The stifle remained moderately distended but with reduced heat and pain on palpation.

2.4.1 Results of genetic testing

Two weeks after the genetic testing at admission, the colt's genotype was revealed to be heterozygous (*My*/N), with autosomal dominant inheritance with variable penetrance, the same of the mare. The foal was confirmed to be not a carrier of the mutation responsible for the following genetic hereditary diseases of the Quarter Horse; deficiency in the glycogen branching enzyme deficiency (GBED) Type I Polysaccharide Storage Myopathy (PSSM Type I), Hereditary Equine Regional Dermal Asthenia (HERDA), Hyperkalemic Periodic Paralysis (HYPP).

2.5 Outcome

The foal was discharged with instructions for continued box rest for four additional weeks and with clinical and radiographic re-evaluation after that period. Approximately one week after discharge, despite attempts at continued management at home, the foal remained markedly lame. Given the persistence of severe lameness and the poor prognosis for future athletic soundness, the owner elected euthanasia. The prognosis is consistent with previously published case of ON or of severe traumatic injuries in foals of this age. As no post-mortem examination was permitted, long-term orthopaedic outcome could not be assessed.

3. Discussion

Lesions affecting the MFC in foals represent a significant diagnostic challenge, as overlapping clinical, laboratory, and imaging findings may hinder accurate characterisation of the underlying pathology [18].

In particular, the acute onset of severe lameness associated with FMTJ effusion may reflect a spectrum of conditions involving the osteochondral unit, ranging from developmental disorders [19], septic arthritis or osteomyelitis [20] to traumatic injuries [6]. In the present case, based on the available data, the main differential diagnoses included ON of the MFC and traumatic injury, such as avulsion fracture at the origin of the MCL.

ON of the MFC represents a highly plausible diagnosis in the present case and should be considered a primary differential in neonatal foals presenting with acute, severe hindlimb lameness and femorotibial joint effusion [5]. This condition, although uncommon, has been consistently associated with a characteristic clinical presentation involving marked lameness, joint distension, and progressive functional impairment, often developing within the first weeks of life [21]. Reported cases include foals as young as 9 days of age to 85 days of life, indicating that disruption of the articular epiphyseal cartilage complex (AECC) can occur extremely early in postnatal development and potentially even reflect perinatal or in utero insults [7].

A notable and diagnostically challenging feature of ON is the apparent discrepancy between the severity of structural pathology and the relatively mild synovial response. In both the present case and previous reports, synovial fluid analysis is typically nonseptic or only mildly inflammatory, with normal or only slightly increased total nucleated cell counts despite extensive osteochondral damage [5,7]. The presence of haemarthrosis, when detected, further supports disruption of the osteochondral interface and detachment of the AECC, consistent with the structural failure described in advanced lesions [7].

Radiographic findings in the present case are consistent with those described in ON, including subchondral irregularity, alteration in trabecular bone opacity, and the presence of an osteochondral fragment located in the caudo-abaxial region.

A structured radiographic grading system for ON lesions has recently been proposed by Marcos-Serralta et al. 2025 [5], to standardise lesion assessment based on the extent of subchondral and trabecular bone involvement and the degree of osteochondral separation. Within this framework, lesions progress from mild subchondral irregularities without detachment (Grade 1–2) to more extensive involvement of the condyle with partial (Grade 3) or complete (Grade 4) detachment of the osteochondral fragment.

In the present case, the radiographic appearance is most consistent with a Grade 4 lesion, characterised by widespread subchondral and trabecular abnormalities involving a substantial portion of the medial femoral condyle, associated with complete detachment and displacement of the osteochondral fragment. This grading reflects an advanced stage of structural failure of the osteochondral unit, with loss of continuity between the articular cartilage and underlying subchondral bone.

From a clinical standpoint, higher radiographic grades have been associated with more severe lameness and a poorer prognosis in affected foals, although a clear statistical correlation between grading and outcome has not been consistently demonstrated. In this context, the classification of the present lesion as Grade 4 further supports the interpretation of a severe and advanced osteochondral pathology,

reinforcing the diagnostic suspicion of ON and providing an objective framework for comparison with previously reported cases.

The aetiopathogenesis of ON of the MFC in foals remains complex and likely multifactorial, but three possibilities have been described: a developmental condition as a part of osteochondrosis [22]; a septic insult with bacterial colonisation of the articular epiphyseal cartilage complex (AECC; [7]) and osteonecrosis secondary to lack of vascular supply of the subchondral bone of unknown origin [21]. In particular, histopathological findings from the Pye et al. 2022 [7] case series demonstrated consistent osteochondral necrosis with detachment of the AECC, frequently accompanied by neutrophilic infiltration within cartilage canals, indicative of septic cartilage canal involvement. These findings suggest that bacterial colonisation, most likely via haematogenous spread during episodes of neonatal septicaemia, may lead to vascular occlusion, ischaemia, and subsequent necrosis of the epiphyseal cartilage and subchondral bone. This hypothesis is further supported by the frequent association between ON and concurrent or preceding systemic disease. In the Pye et al. 2022 case series, most foals had a history of infection or neonatal maladjustment syndrome [7], while in the Marcos-Serralta series, approximately 50% of cases had identifiable prior disease, including septicaemia or umbilical infection [5]. However, the occurrence of ON in foals without any documented history of systemic illness highlights that overt sepsis is not a prerequisite, and that subclinical bacteraemia or non-infectious vascular insults may also play a role.

From a pathophysiological standpoint, the vascular architecture of the distal femoral epiphysis appears to be a critical predisposing factor. The high density of cartilage canal vessels within the thick growth cartilage of the medial femoral condyle, particularly in the caudal weight-bearing region, may increase susceptibility to vascular compromise and bacterial embolisation. Occlusion of these vessels, whether due to septic emboli, thrombus formation, or endothelial disruption, can result in focal ischaemia, leading to necrosis of the osteochondral unit and subsequent structural collapse. The consistent localisation of lesions in the caudo-abaxial region of the MFC across studies supports this vascular vulnerability model [23].

In the present case, several of these pathogenetic mechanisms may be relevant. The acute onset of severe hindlimb lameness, the characteristic localisation of the lesion to the caudo-abaxial aspect of the MFC, together with the radiographic appearance of a fragment associated with marked subchondral irregularity, is consistent with the typical distribution and morphology of ON lesions described in the literature. In addition, the synovial fluid findings, which were not consistent with septic arthritis despite the presence of marked joint effusion, closely mirror those reported in ON, where synovial cytology often underestimates the severity of the underlying osteochondral pathology. The presence of haemarthrosis further supports disruption at the osteochondral interface, consistent with detachment of the articular epiphyseal cartilage complex.

Importantly, the documented history of omphalitis represents a clinically relevant factor, as umbilical infections are a recognised source of bacteraemia in neonatal foals and have been associated with the

development of osteoarticular lesions [24,25]. Haematogenous bacterial dissemination may result in colonisation of cartilage canals, vascular occlusion, and subsequent ischaemic necrosis of the osteochondral unit, even in the absence of overt systemic signs [20].

However, the early age at presentation represents a critical and atypical feature, as it is earlier than that commonly reported for ON of the MFC, and therefore does not fully support this diagnosis. In addition, it is noteworthy that all previously reported cases of ON of the MFC have been described in Thoroughbred foals [5,7,21], whereas the present case involves a QH. Therefore, it remains unclear whether this condition is under-recognised in other breeds or may show a breed-related predisposition, and this case may represent one of the first descriptions in a QH foal.

Despite all the elements supporting on as the most likely diagnosis, a definitive diagnosis could not be established. The absence of computed tomography (CT) limited detailed evaluation of the subchondral and trabecular bone architecture, including the extent of bone lysis and osteochondral separation [7,26]. Similarly, the lack of arthroscopic assessment precluded direct visualisation of the articular surface and confirmation of cartilage detachment, as well as evaluation of intra-articular structures [5]. These limitations prevented definitive differentiation between primary ON and other osteochondral or traumatic lesions.

An alternative differential diagnosis to consider in the present case is a traumatic lesion involving the MCL, specifically an enthesial avulsion at its femoral origin. The collateral ligaments (CLs) constitute the major stabilising components of the equine limb joints, primarily limiting motion to the sagittal plane [27]. CL injuries of the equine stifle are uncommon compared with meniscal or cruciate-ligament disease, but when they occur, they may represent a significant source of lameness [28]. Preliminary research identified the stifle as a frequent site of complex soft-tissue injury, with collateral-ligament lesions accounting for a minority of referred cases [29]. Most documented presentations involve partial desmopathy, periligamentous fibrosis, or meniscocapsular injury [27,30], whereas focal enthesial avulsion fractures have been rarely reported.

In the present foal, the acute onset of severe lameness without any apparent history of trauma, along with the localisation of swelling to the medial aspect of the stifle, the identification of a small, sharply marginated osseous fragment adjacent to the medial femoral epicondyle, together with ultrasonographic findings of proximal MCL thickening, reduced echogenicity, and altered fibre architecture, supports the possibility of an avulsion fracture. In addition, the presence of haemarthrosis further suggests a traumatic component, potentially related to disruption at the osteoligamentous interface.

Avulsion injuries at ligamentous attachment sites are tough to result from a failure of the osteoligamentous junction in response to tensile overload, where the fibrocartilaginous enthesis detaches with a fragment of cortical bone [31]. These injuries have been reported in several equine joints, illustrating the shared biomechanical principle of traction-induced enthesial failure [27]. Documented examples include avulsion of the long lateral collateral ligament of the tarsus at the calcaneal attachment [32], avulsion of the plantar aspect of the calcaneus with proximal intertarsal

subluxation [33], avulsion of the medial plantar eminence of the first phalanx secondary to collateral-ligament detachment [34], and avulsion of the round ligament of the femoral head [35]. The lesion observed in this colt was located at the femoral origin of the MCL, a site not previously reported as an avulsion point in the equine stifle. It consisted of a small, sharply marginated cortical fragment adjacent to the medial femoral epicondyle, with associated proximal MCL thickening and reactive synovitis.

However, in the present case, the absence of a clearly identifiable traumatic event and the extremely young age of the foal make a purely acute traumatic mechanism less likely. To the authors' knowledge, similar lesions have not been reported in foals of comparable age, further limiting interpretation.

It is therefore possible that, rather than representing a primary traumatic avulsion, the osseous fragment may reflect secondary mechanical failure of a structurally compromised osteochondral unit. In this context, altered biomechanics, reduced peri-articular stabilisation, or underlying tissue vulnerability may have contributed to local stress concentration at the ligament insertion [36]. The history of confinement since birth and the presence of bilateral flexural deformities may have further influenced limb loading patterns, potentially increasing strain on the medial stabilising structures of the stifle [37,38]. These elements, together with immature bone and ligament architecture, could have predisposed to traction overload at the MCL origin (Figure 4).

However, the absence of advanced imaging represents an important limitation in this case. CT and/or arthroscopic evaluation would have allowed more precise characterisation of the lesion and direct assessment of the ligament insertion, thereby facilitating differentiation between a primary ON and a true avulsion injury. In their absence, both hypotheses remain plausible.

Nevertheless, several elements favour ON over a primary avulsion lesion, including the extremely early age at presentation, the characteristic localisation of the lesion, the radiographic appearance consistent with osteochondral disruption, the nonseptic synovial fluid findings, and the history of omphalitis suggesting a possible haematogenous component. Conversely, the presence of a discrete osseous fragment, ultrasonographic evidence of MCL involvement, and haemarthrosis support a potential avulsion-type mechanism.

In addition to these factors, the underlying myopathy may have contributed to lesion development in this foal, potentially affecting both joint stability and the susceptibility of the osteochondral unit, and therefore influencing both the development of ON and a possible ligamentous injury. However, it remains unclear to what extent this condition may have influenced the present case, as osteochondral necrosis of the medial femoral condyle has not been previously described in QH, and similar avulsion lesions at this site have not been reported in foals of this age. For these reasons, the genetic background of the foal warrants particular consideration. The owner reported that the mare's genetic status was known before breeding; however, the mare was bred intentionally due to her high-performance genetic background despite being heterozygous for the *MYH1* E321G mutation. However, since the stallion's phenotype was unknown, breeding a heterozygous mare should be discouraged, as the probability of passing the altered the gene could range from 50 to 100%. Even if the clinical manifestation of this

muscular disease may occur in isolated episodes [39], triggering factors remains not yet fully understood. The reason why some heterozygotes do not develop disease is not known but it suggested to be a result of the disease exposure, the environment and the horse's genetic background [15]. In the American QH population, from 6% to 14% of QH have been reported to be heterozygous for the mutation [40], mainly in reining, working cow, and halter horses. The increased prevalence in high-performance horses is suggested to be due to common ancestors derived from limited genetic lines [40]. If compared to other genetic diseases such as HYPP, it is reported than the risk of having E321G MYH1 variant is greater in case of a random breeding of registered QHs in the population [40]. The presented case highlights the need of additional studies on the prevalence of genetic conditions in susceptible breeds. Genetic testing is crucial for a successful breeding program to avoid deleterious genetic variant. Preimplantation genetic testing (PGT) of embryos may allow to ensure healthier offsprings, particularly when applied to equine ICSI blastocysts prior to freezing or embryo transfer (ET) [41]. Equine embryos were reported to be analyzed and genotyped for genetic conditions including Tobiano white spotting pattern, Warmblood Fragile Foal Syndrome Type 1 (WFFST1) and GBED [41]. Given the risk of genetic disease, it is advisable for QH to reproduce via embryo transfer using embryos free of the genetic disorder. This selection enhances ET outcomes and has significant implications for economic value, biodiversity, and equine health [41]. Genetic diseases in QH may result not only in myopathy, but also in abortion, stillbirth and perinatal death, as described in foals with GBED [13].

In the present case, the colt's heterozygous *MYH1* E321G genotype, associated with myosin-heavy-chain myopathy, represented undoubtedly a predisposing factor for the muscleskeletal disease. The selective degeneration and inflammation of the type 2X myofibres potentially alters muscle tone and coordination in affected foals. In presence of the E321G mutation, the involved myofibers have a hyper-contractile phenotype that could contribute to MYHM development [11,12,42].

Although the mutation primarily affects muscle fibres, subclinical weakness or altered neuromuscular control could have further compromised limb stability [12,14,43]. The concurrence of a genetic myopathy, flexural deformities, and the severe MFC lesion in this foal highlights how genetic background and biomechanical factors can interact in the development of orthopaedic disorders, reinforcing the need to include genetic screening as part of the evaluation of breeding assessments [44]. Identifying carriers of MYH1 variants is crucial to preventing the propagation of genotypes associated with latent muscular weakness and compromised locomotor performance.

Congenital flexural deformities may be associated with primary and secondary myopathic alterations or neuromuscular disorders [38]. In human medicine, the diagnostic workup of children with congenital contractures often includes a muscle biopsy. In foals with flexural deformity, the most frequently identified muscle disorders through muscle biopsy in a recent study included core-like myopathy, mild nonspecific myopathy, mitochondrial myopathy, polysaccharide storage myopathy, and congenital fiber-type disproportion [38]. The muscle biopsy was not performed in the present study, and this may represent an additional limitation. However, in the present case, the extent of muscle stiffness seemed to be related to the hindlimb lameness.

As high serum CK may be indicative of generalized muscle damage [10] and increased AST activity may also indicate muscle necrosis [38], the main causes of nonexertional rhabdomyolisis were considered in the differential diagnosis flowchart, including inflammatory/infectious disease.

Nutritional etiology was considered less probable, considering the ortophedic lesions.

The immune-mediated myositis (IMM) associated to the MYHM mutation, as well as systemic calcinosis, were excluded in the present case. IMM is considered as a distinct phenotype, and it is more common in older horses. The phenotypic heterogeneity related to MYHM mutation [13], related to the MYH1 dominant mode of inheritance with variable penetrance, may explain the concomitant presence of transient flexural limb deformities and LCs injuries in the present case. However, the above-mentioned uncommon association was not previously reported. In this foal, diagnostic imaging plays a central role in characterising stifle pathology and differentiating between osseous and soft-tissue involvement. Radiography remains the primary modality for detecting osseous fragments, whereas ultrasonography provides essential complementary information on soft-tissue integrity and synovial changes [45,46]. In the present case, while standard imaging was sufficient to identify the lesion, further diagnostic modalities could have refined the evaluation. As already outlined, advanced imaging modalities should have considerably expanded the diagnostic accuracy. In fact, CT and magnetic resonance imaging (MRI) provide information beyond the limits of radiography and ultrasonography, allowing more accurate lesion characterisation, prognosis, and therapeutic planning [47]. However, high-frequency ultrasonography allows visualisation of the ligament's echotexture, fibre alignment, and capsular relationships [48] and remains the most practical tool for detecting collateral-ligament injury in foals, where MRI is not often feasible. Ultrasonographic findings in these cases are often scarcely described in the literature; therefore, this report may provide useful additional imaging information. In addition to conventional imaging, diagnostic arthroscopy remains the reference standard for assessing intra-articular pathology of the equine stifle. Arthroscopy allows direct visualisation and palpation of the menisci, cruciate ligaments, and collateral-ligament attachments, enabling identification of subtle chondral and meniscal lesions that may not be apparent with other imaging modalities. Furthermore, it provides an opportunity to assess tissue viability and mechanical integrity under dynamic conditions and, where appropriate, to undertake immediate therapeutic intervention [49–51]. In the present case, the decision not to perform arthroscopy precluded direct confirmation of the lesion and evaluation of potential intra-articular damage, particularly to the meniscocapsular and cruciate complexes.

The absence of a post-mortem examination represents another significant limitation of the study.

The present case highlights the considerable diagnostic challenges associated with lesions of the MFC in neonatal foals, in which overlapping clinical, laboratory, and imaging findings may preclude definitive characterisation of the underlying pathology. In this foal, a conclusive diagnosis could not be established, and differentiation between ON and a primary traumatic lesion remained uncertain, largely due to the absence of advanced imaging, arthroscopic evaluation, and histopathological confirmation.

Despite this limitation, the case remains clinically relevant. It describes an unusual presentation in an extremely young foal, involving a breed in which similar lesions have not been previously reported, and documents the coexistence of multiple potential predisposing factors, including early-life infection, biomechanical alterations, and a genetic background potentially affecting musculoskeletal function. These elements collectively provide a unique clinical context that may contribute to a broader understanding of the multifactorial nature of stifle pathology in foals.

This report therefore underscores the importance of recognising that different pathological processes may overlap in neonatal patients, and that diagnostic findings should be interpreted within a comprehensive clinical framework. It also highlights the value of reporting atypical or inconclusive cases, as they may help expand current knowledge of disease expression across breeds, ages, and predisposing conditions, even in the absence of definitive diagnosis.

Further studies, including advanced imaging, arthroscopic evaluation, and longitudinal follow-up, are warranted to better define the pathogenesis, diagnostic criteria, and clinical significance of similar lesions in neonatal foals.

4. Conclusions

The present case highlights the significant diagnostic challenges associated with lesions of the MFC in neonatal foals, where clinical, laboratory, and imaging findings may be difficult to interpret and may limit accurate characterisation of the underlying pathology. In this foal, a definitive diagnosis could not be established, and differentiation between osteochondral necrosis and a primary traumatic lesion remained uncertain, largely due to the absence of advanced imaging, arthroscopic evaluation, and histopathological confirmation. Despite this, the case retains substantial clinical relevance. It describes an unusual presentation in an exceptionally young QH foal, a breed in which ON of the MFC has not been previously reported, thereby expanding the currently recognised spectrum of disease expression. Furthermore, it documents the coexistence of multiple potential predisposing factors, including early-life infection, biomechanical alterations, and a genetic background potentially affecting musculoskeletal function, highlighting the complex and likely multifactorial nature of such lesions. Importantly, this report emphasises that different pathological processes may overlap in neonatal patients, leading to atypical or difficult-to-interpret presentations, and that diagnostic findings should therefore be interpreted within a comprehensive clinical framework rather than in isolation. Even in the absence of a definitive diagnosis, the detailed clinical, radiographic, and ultrasonographic characterisation provided in this case contributes valuable information, particularly in an area where imaging descriptions remain limited.

For these reasons, the present report provides meaningful insights into the variability and complexity of stifle pathology in foals and underscores the importance of documenting atypical and inconclusive cases, which may ultimately contribute to improving diagnostic awareness and clinical decision-making. Further cases and studies, including advanced imaging, arthroscopic evaluation, and longitudinal follow-

up, are warranted to better define the pathogenesis, improve diagnostic accuracy, and clarify the clinical significance of similar lesions in neonatal foals.

Declarations

Competing interests

The authors declare no competing interests.

Declaration of Interest Statement

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Data availability

All data generated or analyzed during this study are included in this published article.

Ethics in Publishing Statement

All authors confirm that the manuscript adheres to the highest standards of ethical publishing. No experimental procedures were conducted for the purposes of this study. All data were obtained during routine clinical veterinary examinations. Written informed consent was obtained from foal owners prior to data collection and inclusion in the study. The authors affirm that the work is original, has not been published elsewhere, and is not under consideration for publication by any other journal.

Author Contribution

I.I. and F.M. wrote the main manuscript text and editing . R.R., J.M., A.L. reviewed and contributed to editing. R.R., J.M. and A.S. provided supervision. All authors read and approved the final manuscript.

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Figures

Figure 1

Clinical presentation of the colt at admission. The colt shows a non-weight-bearing stance of the left hindlimb with the stifle flexed and held off the ground. A bilateral flexural deformity of the carpi is also evident, characterised by mild carpal contracture.

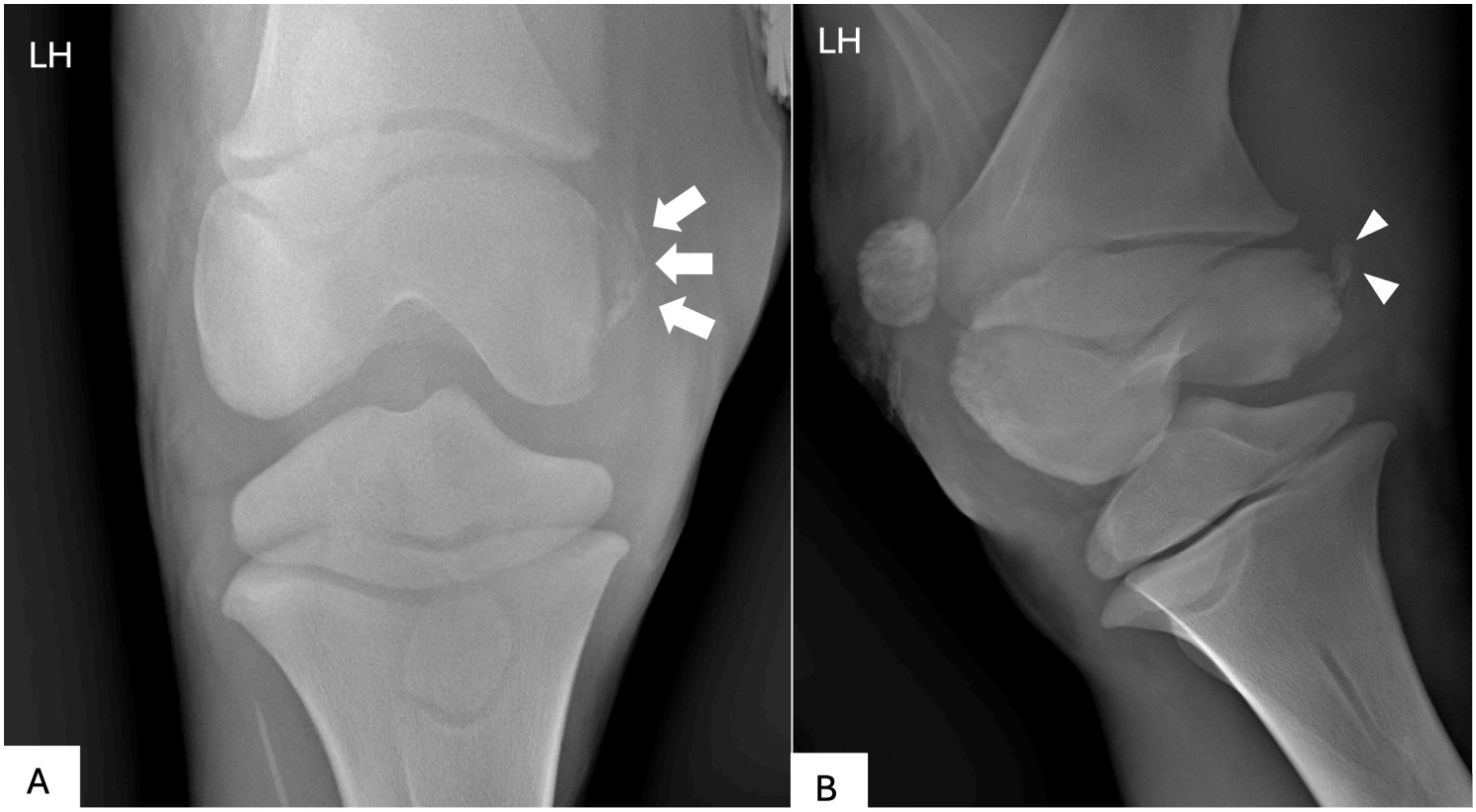

Figure 2

Radiographic examination of the left stifle: Images are displayed with the lateral side to the right. (A) Caudocranial (CdCr) and (B) caudal 60° lateral–craniomedial oblique (Cd60°L–CrMO) views showing marked irregularity and subchondral lysis of the MFC, associated with a mineral-dense fragment (arrows in A; arrowheads in B)

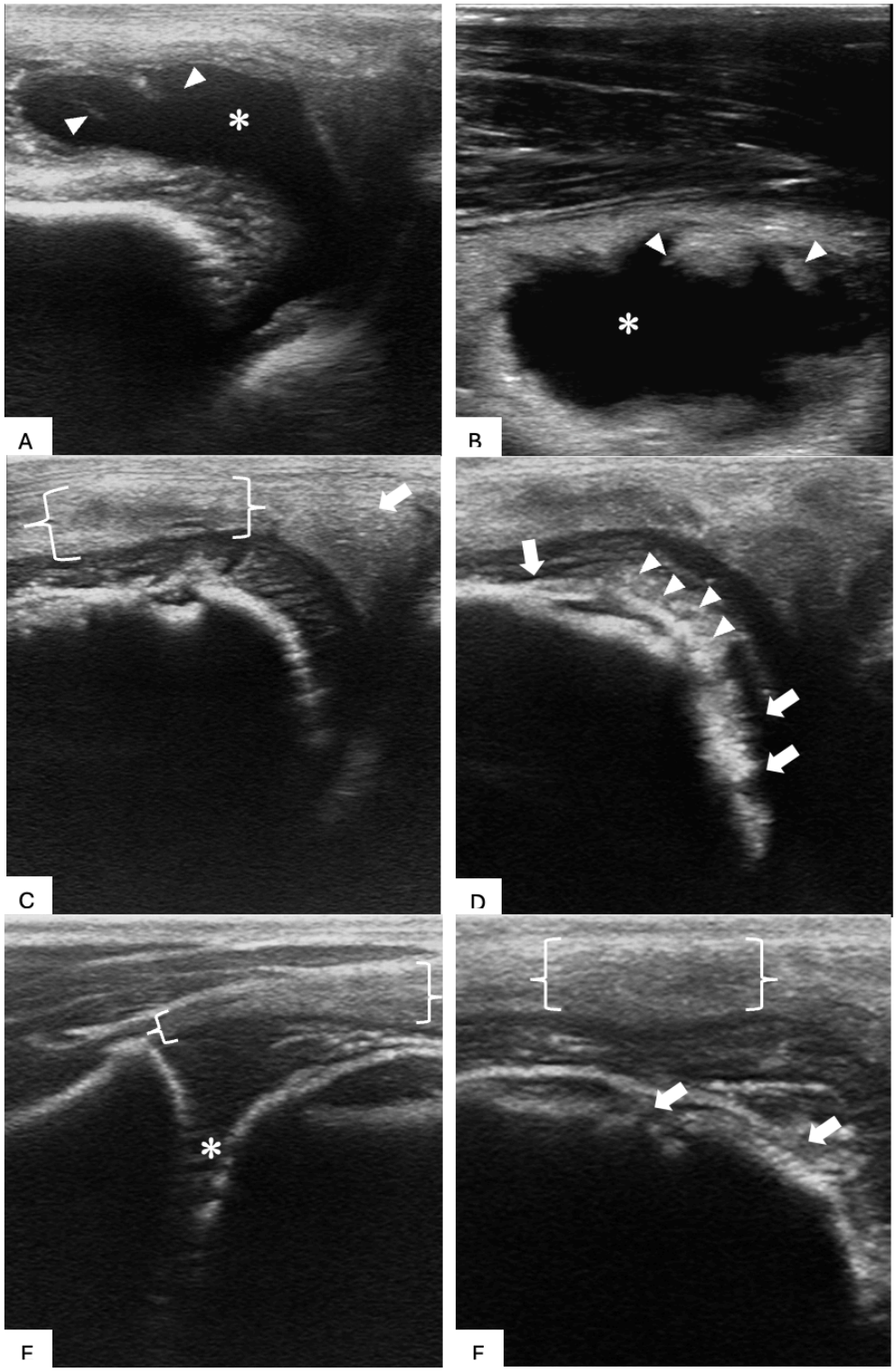

Figure 3

Ultrasonographic evaluation of the left medial femorotibial joint: Images are displayed with the medial/cranial side to the right.

(A–B) Longitudinal and transverse scans of the medial femorotibial recess showing severe distension by anechoic effusion (asterisk) and villous thickening of the synovial membrane (arrowheads). The synovial

lining appears irregular and hyperechoic, and fine echogenic filaments are visible within the joint fluid, consistent with marked synovial reaction.

(C) Longitudinal view of the medial collateral ligament (MCL) showing diffuse thickening and loss of fibrillar pattern (brackets). The normal appearance of the medial meniscus can also be observed (arrow)

(D) Longitudinal scan at the level of the medial femoral condyle demonstrating irregular and heterogeneous cortical-subchondral bone surface (arrow) and a hyperechoic focus with acoustic shadowing (arrowheads) compatible with fragmentation.

(E) Longitudinal scan of the distal femoral physis showing a regular hypoechoic physeal line (asterisk) bordered by smooth hyperechoic margins. Thickened MCL at its enthesis is also visible (brackets)

(F) Longitudinal scan at the level of the proximal MCL and medial femoral epicondyle, confirming hypoechoic thickening of the ligament (bracket). The cartilage and subchondral bone show marked irregularity and heterogeneous echogenicity (arrows).

FEATURES	OSTEOCHONDRAL NECROSIS	AVULSION FRACTURE OF THE MEDIAL COLLATERL LIGAMENT
VERY YOUNG AGE AT PRESENTATION	+	-
ACUTE ONSET OF SEVERE LAMENESS	±	+
ABSENCE OF WITNESSED TRAUMA	+	-
TYPICAL CONDYLAR LOCATION	++	+
EXTENSIVE SUBCHONDRAL/CORTICAL IRREGULARITY	++	±
DISCRETE MINERALISED FRAGMENT	+	+
PROXIMAL MCL THICKENING AND FIBRE DISRUPTION	-	++
NON-SEPTIC SYNOVIAL FLUID PROFILE	++	++
HAEMARTHROSIS	+	++
CONCURRENT OMPHALITIS / POSSIBLE HAEMATOGENOUS COMPONENT	+	-
POSSIBLE CONTRIBUTION OF ALTERED BIOMECHANICS / MYH1-ASSOCIATED MYOPATHY	+	+

Figure 4

Comparative diagnostic features supporting osteochondral necrosis (ON) versus enthesial avulsion injury of the medial collateral ligament (MCL) of the medial femorotibial joint (MFTJ). The figure summarises the relative diagnostic weight of the main clinical, imaging, and contextual findings considered in the differential interpretation of the medial femoral condyle lesion. Overall, the pattern of evidence favoured ON, although an enthesial avulsion component could not be excluded.