Acta Ortopédica Mexicana 2024; 38(3): May.-Jun. 179-187

Review

doi: 10.35366/115813

Arthrofibrosis of the knee in pediatric orthopedic surgery

Artrofibrosis de rodilla en cirugía ortopédica pediátrica

Faust TF,* Castañeda PG‡

Department of Research, Alabama College of Osteopathic Medicine. Alabama, USA.

ABSTRACT. Arthrofibrosis is a challenging complication associated with knee injuries in both children and adults. While much is known about managing arthrofibrosis in adults, it is necessary to understand its unique aspects and management strategies in the pediatric population. This paper provides an overview of arthrofibrosis in pediatric orthopedic surgery, focusing on its causes, implications, classifications, and management. This paper is a comprehensive review of the literature and existing research on arthrofibrosis in pediatric patients. Arthrofibrosis is characterized by excessive collagen production and adhesions, leading to restricted joint motion and pain. It is associated with an immune response and fibrosis within and around the joint. Arthrofibrosis can result from various knee injuries in pediatric patients, including tibial spine fractures, ACL and PCL injuries, and extra-articular procedures. Technical factors at the time of surgery play a role in the development of motion loss and should be addressed to minimize complications. Preventing arthrofibrosis through early physical therapy is recommended. Non-operative management, including dynamic splinting and serial casting, has shown some benefits. New pharmacologic approaches to lysis of adhesions have shown promise. Surgical interventions, consisting of arthroscopic lysis of adhesions (LOA) and manipulation under anesthesia (MUA), can significantly improve motion and functional outcomes. Arthrofibrosis poses unique challenges in pediatric patients, demanding a nuanced approach that includes prevention, early intervention with non-operative means, and improvements

RESUMEN. La artrofibrosis es una complicación difícil asociada con lesiones de rodilla tanto en niños como en adultos. Si bien se sabe mucho sobre el manejo de la artrofibrosis en adultos, es necesario comprender sus aspectos únicos y estrategias de manejo en la población pediátrica. Este documento proporciona una visión general de la artrofibrosis en la cirugía ortopédica pediátrica, centrándose en sus causas, implicaciones, clasificaciones y manejo. Este documento es una revisión completa de la literatura y la investigación existente sobre artrofibrosis en pacientes pediátricos. La artrofibrosis se caracteriza por una producción excesiva de colágeno y adherencias, lo que conduce a un movimiento articular restringido y dolor. Se asocia con una inmunorrespuesta y fibrosis dentro y alrededor de la articulación. La artrofibrosis puede ser el resultado de varias lesiones de rodilla en pacientes pediátricos, incluyendo fracturas de columna tibial, lesiones de LCA y LCP, y procedimientos extraarticulares. Los factores técnicos en el momento de la cirugía desempeñan un papel en el desarrollo de la pérdida de movimiento y deben abordarse para minimizar las complicaciones. Se recomienda prevenir la artrofibrosis a través de la fisioterapia temprana. La gestión no operativa, incluyendo el empalme dinámico y la fundición en serie, ha mostrado algunos beneficios. Los nuevos enfoques farmacológicos a la lisis de adherencias han demostrado ser prometedores. Las intervenciones quirúrgicas, consistentes en lisis artroscópica de adherencias (LOA) y manipulación bajo anestesia (MUA), pueden mejorar significativamente el movimiento y los resultados funcionales. La artrofibrosis plantea desafíos

* Department of Research, Alabama College of Osteopathic Medicine.
‡ Baylor School of Medicine, Department of Pediatric Orthopedic Surgery, Texas Children's Hospital.

Correspondence:
Taylor F Faust, BS MS
Department of Research, Alabama College of Osteopathic Medicine, 445 Health Sciences Boulevard, Dothan, AL 36303.
E-mail: faustt@acom.edu

Received: 01-09-2024. Accepted: 02-18-2024.

How to cite: Faust TF, Castañeda PG. Arthrofibrosis of the knee in pediatric orthopedic surgery. Acta Ortop Mex. 2024; 38(3): 179-187. https://dx.doi.org/10.35366/115813

in surgical techniques. Modern pharmacological interventions offer promise for the future. Customized interventions and research focused on pediatric patients are critical for optimal outcomes.

Keywords: erthrofibrosis, knee, child, surgery.

únicos en los pacientes pediátricos, exigiendo un enfoque matizado que incluye prevención, intervención temprana con medios no operatorios y mejoras en las técnicas quirúrgicas. Las intervenciones farmacológicas modernas ofrecen una promesa para el futuro. Las intervenciones e investigaciones personalizadas centradas en pacientes pediátricos son fundamentales para obtener resultados óptimos.

Palabras clave: artrofibrosis, rodilla, niño, cirugía.

Introduction

The restriction of knee range of motion (ROM) after injury or surgery of the knee poses a formidable challenge, stemming from both single and multi-ligament injuries or post-reconstruction consequences. In adult orthopedics, knee injury recognition and management have reached relative prominence, yet there needs to be more information in the pediatric population. The disparity in managing these issues in children necessitates a comprehensive understanding of their requirements, distinct from the adult population.

The extent of knee motion loss varies, contingent on the inciting injury mechanism. Typically, a more severe loss in motion signifies more significant ligament damage, often involving multiple ligaments and high-energy injury events. These injuries can result from either mechanical trauma or pre-existing degenerative conditions. Risk factors for motion loss encompass numerous variables, including the timing of surgical intervention, differences in postoperative rehabilitation, genetic predisposition, concurrent infections, surgical errors, and complications, among others.

Arthrofibrosis manifests as a symptomatic reduction in knee ROM compared to the contralateral knee. This enigmatic condition emerges as a complication following surgical procedures or knee injuries, culminating in the thickening and scarring of the joint capsule and surrounding tissues. These changes give rise to restricted knee motion, often accompanied by varying degrees of pain. Among pediatric patients, arthrofibrosis can arise following knee surgery, such as reconstruction or fracture management. Its manifestations range from focal to diffuse involvement of the knee and its adjacent structures. Arthrofibrosis is clinically defined as a lack of 10º from full extension or less than 90º of knee flexion three months after the initial procedure.[1,2]

Enhancing our understanding of arthrofibrosis is crucial for refining techniques to prevent motion loss and achieve more successful post-surgical outcomes. In cases where preventive measures fall short, exploring the most effective treatment strategies becomes imperative, including static or dynamic bracing, manipulation under anesthesia, and arthroscopic or open debridement.[3]

Understanding normal knee motion is multifaceted, encompassing various motions across different axes. Planes of motion include longitudinal, rotational, varus and valgus, flexion, and extension movements. In particular, the range of knee flexion and extension is divided into three categories: terminal extension, active function, and passive flexion.[3] Terminal extension signifies the limit of passive extension and is often minimal, around 10º to 5º of hyperextension. It plays a role in quadriceps muscle relaxation during the stance phase and is not frequently encountered during normal gait. The arc of functional motion ranges between 10º and approximately 120º, encompassing everyday activities like squatting and ascending or descending stairs. Passive flexion extends to around 120º and continues under applied external force. Gender differences exist, with men typically achieving 140º and women 143º.[3]

Implications of flexion loss

For most daily activities that do not significantly impact gait, a knee flexion of up to 125º is essential. Inadequate flexion beyond this point can impede squatting, and even slight deficits in flexion may adversely affect the performance of athletes. Severe flexion deficits, falling below 90º, render even the least active individuals unable to sit and climb stairs.[3]

Challenges posed by extension loss

Compared to the loss of flexion, a loss of knee extension is less tolerable and presents a more challenging clinical scenario. A mere 5º loss of extension can induce a limp during ambulation, leading to quadriceps strain and patellofemoral pain. In weight-bearing situations with a flexed knee, the quadriceps muscle is crucial for stability, bearing 7% of the load at 15º of flexion, 210% at 30º, and 410% at 60º.[4] This increased load results in elevated joint contact pressure and may lead to patellofemoral arthrosis.[3]

Measuring motion loss

Precise assessment of knee motion loss in patients with recent ligamentous knee injuries or following reconstruction necessitates a fine-tuned approach. The current technique employs a goniometer placed on the lateral knee joint line in a midsagittal position, using the greater trochanter and lateral

malleolus as landmarks. This method exhibits high inter- and intra-observer reliability.[5] An alternative method employs heel-height measurement differences with patients in a prone position, with a 1 cm heel-height disparity equating to a 1º knee flexion contracture. This method is particularly useful for detecting subtle degrees of motion loss below 10º.[6]

Classification of motion loss

Defining the true incidence and character of motion loss in the knee is a complex endeavor. Several classification schemes are available, with the most recent one introduced by Shelbourne et al.,[7] which compares motion loss on the affected side to the contralateral limb. This classification system delineates four distinct types based on flexion and extension losses, with or without patella-infera. Type 1 is normal flexion with extension loss at < 10º; type 2 is normal flexion and extension loss > 10º; type 3 is flexion loss of > 25º and extension > 10º, and type 4 is flexion loss > 30º and extension loss > 10º with patella-infera.

Incidence of motion loss

Previous studies have reported postoperative loss of knee motion in approximately 35% of adult patients who underwent anterior cruciate ligament (ACL) reconstruction or repair.[8] Thanks to advances in surgical techniques and rehabilitation protocols, this incidence has decreased from 0 to 4%.[9,10,11,12] High-energy multi-ligament injuries are more predisposed to motion loss than single-ligament, low-energy injuries. A study investigating the early treatment of motion complications after ACL reconstruction found a 23% incidence of motion loss in patients undergoing concomitant ACL reconstruction and medial collateral ligament (MCL) repair.[13] Traumatic knee dislocations, causing various ligamentous instabilities, are also associated with increased motion loss. Unfortunately, data specific to a pediatric population is more difficult to ascertain.

Pathophysiology

Arthrofibrosis is characterized by excessive collagen production and adhesions, resulting in restricted joint motion and pain. This condition, which may affect various joints,[14] is known by different names, such as frozen shoulder, adhesive capsulitis, joint contracture, stiff knee, and stiff elbow.[15] Sterile arthrofibrosis, another variant, arises from chronic or repetitive injuries or surgeries, triggering a dysregulated immune response and fibrosis within and around the joint.[16] Fibrotic scar tissue formation in the joint's extracellular matrix (ECM), primarily composed of collagen, is the hallmark of this condition. Fibrosis represents the final common pathway in the context of chronic inflammatory injuries, transcending multiple organs.[17,18] Normal tissue injuries trigger the release of local inflammatory cytokines, initiating a complex cascade that attracts immune cells to the affected area. Under the influence of inflammatory cytokines, fibroblasts differentiate into myofibroblasts, producing the repair of extracellular matrix proteins.[19] Transforming growth factor beta (TGF-β) is pivotal in arthrofibrosis pathophysiology, regulating immune responses and wound healing.[20] It stimulates myofibroblast proliferation, increases disorganized extracellular matrix production, inhibits myofibroblast apoptosis, and impedes collagen degradation.[19] Other factors also play a role, including TNF-α, platelet-derived growth factor, and IL-1,6,17. The myofibroblasts are critical as the TGF-β influences them to produce the dense ECM. The ECM comprises mainly nonelastic type 1 collagen that forms connections to adjacent tissues. There are 10-fold higher amounts of alpha-smooth muscle actin containing myofibroblast in patients with arthrofibrosis following ACL-reconstruction revision than in patients with primary ACL-repair.[17] Genetic factors may also contribute to susceptibility to arthrofibrosis, potentially involving genetic predispositions related to musculoskeletal tissues.[21] Early intervention targeting these genetic variants could enhance patient outcomes.[20] A better understanding of the pathophysiology of arthrofibrosis, along with the discovery of genetic involvement, promises to refine management strategies, especially in pediatric cases where heightened awareness and tailored management techniques are paramount.

Causes of arthrofibrosis in pediatric patients following knee injuries

Knee trauma as a precursor to arthrofibrosis

Injuries, such as avulsion fractures of the tibial eminence, often result from falls and sports-related accidents. Non-displaced or minimally displaced fractures are typically treated conservatively, while open reduction and fixation are indicated for displaced fractures.[22] It is crucial to note that open reduction with arthrotomy, historically used for displaced fractures, has been associated with prolonged rehabilitation and increased morbidity.[23] In contrast, arthroscopic reduction and fixation have emerged as preferred techniques due to reduced hospital stays and morbidity, expediting rehabilitation.[24] Nevertheless, complications, including extension loss and quadriceps weakness, can occur in displaced and non-displaced tibial eminence fractures, especially when sutures or screws are employed.[22] Skeletally immature patients are particularly susceptible to postoperative knee stiffness.[25] The primary treatment goal is the restoration of knee motion through anatomic reduction, preserving ligament tension, and preventing extension loss. It is imperative to discuss possible complications with patients before surgery, considering the acute inflammatory phase's role in arthrofibrosis pathogenesis. The development of an intense postoperative rehabilitation program is essential for minimizing the risk of postoperative motion loss.

Risk factors of arthrofibrosis in tibial spine fractures

Knee arthrofibrosis in pediatric patients can follow a tibial spine fracture, both after cast immobilization and arthroscopic treatment.[26] A retrospective multicenter study by Vander Have et al.[22] found that 10% of children with a tibial spine fracture experienced arthrofibrosis after surgical treatment.

Tibial spine fractures in pediatric patients are relatively rare, but identifying the risk factors associated with arthrofibrosis development is essential for mitigating the risk. Several risk factors have been identified:

1. Concomitant ACL injury: pediatric patients with tibial spine fractures who also have an ACL tear are more than seven times more likely to develop postoperative stiffness.[25]
2. Traumatic injuries unrelated to athletics: traumatic injuries not associated with athletic activities, such as motor vehicle accidents, bicycle accidents, falls from heights, and horseplay/fighting, significantly increase the risk of arthrofibrosis development.[25]
3. Prolonged operative times: longer operative durations, especially for more complex fractures, can contribute to the development of arthrofibrosis, albeit not as an isolated risk factor.[25]
4. Cast immobilization: immobilizing the knee in a cast increases the risk of arthrofibrosis due to locking the patient's knee in a specific position, leading to a loss of extension.[27]
5. Young age: patients under ten face an increased likelihood of developing arthrofibrosis. Achieving a balance between early mobilization and the risk of complete tear is crucial, as is avoiding controllable risk factors like prolonged cast immobilization.[25]

Arthrofibrosis due to delayed surgery for pediatric tibial spine fractures

In pediatric patients, tibial spine fractures are akin to ACL tears in adults and adolescents but occur less frequently.[28,29,30,31] Delayed surgery and extended operative duration are primary factors associated with arthrofibrosis in these patients.[32] Factors contributing to delayed surgery include diagnosis delay, multiple clinical opinions, and insurance issues. The definition of what constitutes a delayed time frame for tibial spine fracture injuries is shorter than that used for ACL injuries. The more severe mechanism and energy requirements for tibial spine fractures might contribute to poorer outcomes and long-term effects in pediatric patients. Operative fixation delayed by over 21 days is associated with a high likelihood of concomitant meniscus pathology. Additionally, operative times exceeding 2.5 hours have a 3.7-fold higher risk of developing arthrofibrosis following surgical procedures.[32] To address the issue of increased rates of arthrofibrosis, more effective treatments should be identified at the initial presentation, where specific treatments should be considered. Physical examination findings, such as effusion, decreased ROM, and weight-bearing challenges, can facilitate early intervention and prevent further treatment delay.[33] Although tibial spine fractures are relatively rare, understanding how to treat them appropriately, with prompt recognition and patient-specific intervention, is vital to reduce treatment delay.

Technical factors in reconstructive surgery of the knee

ACL reconstruction in pediatric patients

Correct graft placement is crucial in ACL reconstruction surgery to minimize motion loss.[34] Graft placement anterior to the native ACL insertion site on the tibia can lead to impingement on the intercondylar notch roof during extension. Lateral tibial graft placement may result in impingement on the lateral notch wall. Grafts placed too far anteromedially can limit flexion.[35] From the femoral side, a common error is placing the graft too far entirely, which can lead to graft strain, limited flexion, and possible graft failure.[36,37]

Other studies have shown that graft impingement can involve the intercondylar notch and adjacent posterior cruciate ligament (PCL). While moderate impingement can lead to pain, effusion, and extension loss, severe impingement can cause graft abrasion and complete failure.[38] Quadriceps contraction during knee extension can exacerbate graft impingement, and a notchplasty can increase the clearance between the graft and the intercondylar roof. Graft impingement on the PCL can limit flexion, often due to a steep tibial tunnel angle (80º).[39]

Graft tension

The relationship between graft tension and motion loss is still debated. Excessive graft tension can be problematic, with a high degree of pretension potentially leading to graft fraying, particularly over the femoral tunnel.[34] However, this does not necessarily result in a complete loss of knee extension.[40] In contrast, inadequate graft tension can lead to anterior-posterior laxity, causing instability, poor graft healing, and failure.

Graft choice in ACL reconstruction

The link between extension loss and graft type remains an ongoing subject of discussion. Some studies found no significant difference in range of motion (ROM) between hamstring and patellar tendon ACL reconstruction. While one study reported that 31% of the patellar tendon group and 19% of the hamstring group experienced extension deficits at five years postoperatively, these results were not statistically significant.[41] These results were not statistically significant. A follow-up study found no

significant difference in ROM in the recent prospective, randomized trial comparing hamstring and patellar tendon ACL reconstruction after a follow-up at year five.[42] This controversy suggests that graft choice is unrelated to the development of motion loss following ACL reconstruction.

Arthrofibrosis of post-ACL reconstruction in pediatrics and adolescents

Arthrofibrosis is a known adverse complication following ACL reconstruction. Its prevalence in the adult population ranges from 4 to 35%.[8,11,12,13,43,44] One study by Fisher and Shelbourne[12] looked at 959 consecutive ACL restrictions and found that 42% experienced a significant loss of extension. This issue is particularly relevant given the increasing frequency of ACL injuries in pediatric and adolescent populations, prompting more ACL reconstruction procedures in these age groups.[45,46,47] A study of 902 patients (933 knees) found an 8.3% overall incidence of arthrofibrosis. Female patients had a significantly higher postoperative arthrofibrosis rate (11.1%) compared to males (4.0%). Notably, increasing patient age was associated with a higher incidence of arthrofibrosis; no cases were reported in patients under 12.[48] Furthermore, patellar tendon autografts were linked to a significantly increased risk of arthrofibrosis.[48]

The time between the injury and the reconstruction has been a predictor for arthrofibrosis occurrence. Shelbourne et al. found that patients who underwent index surgery less than 21 days after the injury had a higher rate of arthrofibrosis compared to those who waited more than 21 days after the injury.[2] Adult studies have reported a 12% arthrofibrosis incidence, revealing that reconstructions performed less than three weeks after injury and preoperative extension loss of 10 degrees or more were significant predictors of arthrofibrosis.[49] Others have found that patients who underwent reconstruction after at least four weeks post-injury had even lower rates of arthrofibrosis compared to those who underwent surgery earlier. The pediatric population presents unique challenges, and better treatment guidelines tailored to this age group are warranted to address arthrofibrosis.

Arthrofibrosis after PCL reconstruction in pediatric patients

PCL injuries are less common than ACL injuries and often result from knee trauma with acute hemarthrosis.[50,51] In adults, non-surgical management is favored for isolated PCL injuries. However, in pediatric patients, PCL injuries are less prevalent due to the inherent strength and resilience of the ligaments, especially in those with open physes.[52,53] However, there is concern for long-term outcomes of PCL-deficient knee, which has been shown to lead to progression to osteoarthritis due to continuous laxity.[54,55,56] In pediatrics, surgical treatment options include open reduction and screw fixation, arthroscopically-assisted reconstruction with various autografts (hamstring tendon, quadriceps tendon, bone-patellar tendon-bone),[57,58] and allografts (Achilles tendon with bone block and maternal hamstring tendon).[13] Arthrofibrosis, although infrequent, can occur following pediatric PCL reconstruction. Kocher et al.[59] reported seeing it in three knees (7%), two of which underwent manipulation under anesthesia and one who underwent lysis of adhesions with manipulation. While this is not a large number, it does bring awareness that pediatric PCL reconstruction is not exempt from arthrofibrosis. Increased trauma during open arthrotomy for procedures like PCL reconstruction has been identified as a risk factor for motion loss. These findings underscore the importance of minimizing additional joint trauma during surgical interventions.

Osteoarthritis has also been reported in pediatric patients with PCL injuries.[57] In the same consideration, the treatment recommendations for pediatrics need to be improved as guidance is based on adult literature, indicating the need for practical treatment guidelines tailored to the pediatric population. The rarity of PCL injuries in this age group means that current treatment guidance is based on adult literature, making it crucial to develop specialized protocols.

Preventing and managing arthrofibrosis in pediatric patients: a comprehensive approach

Preventing arthrofibrosis

Arthrofibrosis prevention through early physical therapy intervention and anti-inflammatory medications is paramount to mitigate motion loss risks. Additionally, the incorporation of preoperative oral corticosteroids has proven to be effective in the reduction of MUA rates as well.[60] However, further research is required to know whether these benefits extend to the pediatric population.

One pathway that warrants further research is regarding NSAIDs, specifically when administered intra-articularly via a biologic scaffold. A recent animal study showed sustained anti-fibrotic effects in a rabbit model by administering a specific COX-2 inhibitor (celecoxib) via intra-articular injections using a scaffold method.[61]

Non-operative modalities remain important, and a strong emphasis is placed on prevention and early intervention; however, for refractory cases, surgical interventions remain a consideration.

Surgical errors

Technical errors in reconstructive surgery of the PCL have been associated with stiffness, specifically inappropriate tightening.[62,63] These surgical errors lead to alterations in normal knee kinematics, resulting in microtrauma that triggers the inflammatory process that leads to arthrofibrosis.[64] Emphasis on preoperative planning to avoid the common errors seen in the adult population

has been established for knee arthrofibrosis, highlighting the need for further research and evidence to support the pediatric population in recognizing surgical errors.

Management of arthrofibrosis in children

Clinical management of arthrofibrosis

The clinical management of arthrofibrosis in pediatric patients demands a multifaceted approach due to its varied contexts and etiologies. Some combination techniques include a stepwise approach with physiotherapy, serial casting, epidural therapy combined with inpatient physiotherapy, oral corticosteroids, and some relatively new pharmacologic approaches before any surgical consideration.

Arthrofibrosis after ACL reconstruction is best treated in a stepwise approach with early recognition and intervention. In ACL reconstruction, the optimal timing for surgery is typically 3-4 weeks post-injury.[65] This strategy capitalizes on reduced inflammatory mediators and profibrotic signals, leading to a more favorable surgical outcome.[66] In cases of periarticular knee trauma associated with quadriceps muscle injury (e.g., from femoral external fixator pin placement or extensive quadriceps elevation during surgical fixation).[67] Initial manipulation under anesthesia (MUA) and more severe cases may require open release and quadricepsplasty.[68,69] The treatment objective is the mechanical disruption of adhesions and contractures via aggressive physiotherapy, physical joint manipulation, and surgical release and debridement.[66] While these techniques have offered a basic need in treating arthrofibrosis and or treatment, the progress in identifying biochemical mediators and pharmacologic options is beginning to catch up.[70]

There have been improvements in pharmacological treatments, such as intra-articular injections of collagenase clostridium histolyticum, which has been used successfully in Dupuytren's disease of the hand.[71,72] Understanding the mechanism of action and underlying pathophysiology being a somewhat similar disease to arthrofibrosis may be the key to more pharmaceutical management in treating arthrofibrosis.

The use of antifibrotic agents has been explored; one of these is the use of Relaxin-2, a hormone secreted by the placenta that promotes tissue laxity by inhibiting fibrinogen and collagen expression.[73,74] Relaxin-2 has been shown to reduce concentrations of type-1 collagen and can inhibit the activity of myofibroblasts at a biochemical level. While this has only been demonstrated in a murine model, if it can be reproduced in human subjects, it may offer a strong alternative or combination treatment in arthrofibrosis. Another option being explored is intra-articular injection of anakinra, an interleukin-1 (IL-1) receptor antagonist.[75]

Current pharmacological interventions show promise but necessitate further research. Understanding the underlying pathophysiology and the role of biochemical mediators may offer new avenues for pharmaceutical management. As the field advances, these developments promise better outcomes for arthrofibrosis prevention and treatment.

Nonsurgical management

Non-operative treatment has shown varying levels of success. A recent study revealed that high-intensity home mechanical stretch therapy effectively restored knee flexion within two months or less.[76] This approach was shown to prevent additional surgery and minimize emotional distress, irrespective of sex, age, or worker's compensation status.[76] The authors recommended a high-intensity stretching program for any patient at risk of secondary motion loss after surgery. The 6-10 week program provided an average of > 25º of flexion gain.[76]

Dynamic splinting in children and adolescents with stiffness after knee surgery

Dynamic splinting offers another alternative to surgical intervention.[77,78,79,80] It uses a bracing approach that gradually lengthens scar tissue via constant force and a spring-loaded coil. This method has been effective in preventing the need for surgical intervention in up to 58% of patients, according to one study.[81] That study also showed that higher gains in flexion significantly reduced the risk of subsequent need for manipulation under anesthesia (MUA)—all of the patients who did receive MUA after dynamic splinting resulted in improvements in ROM. In the case of those who failed dynamic splinting and required a surgical intervention, the procedure was simplified, and the authors found improved motion in postoperative recovery.[81] Dynamic splinting has been recommended for pediatric patients with postoperative knee arthrofibrosis within six weeks to 30 months post-surgery, especially those who have undergone combined procedures. Individuals who have also undergone combined procedures such as ACLR with meniscal repair or other ligamentous reconstruction, as well as advanced chondral repair,[82] can warrant earlier consideration for dynamic splinting.

Surgical management

The surgical management typically involves arthroscopic lysis of adhesions (LOA). The primary indication for LOA is restricted knee range of motion, with no specific agreement on defined measures and a wide variability amongst surgeons based on preference. An objective measure that can be used as an indication is a failure to achieve a 90º arc of range of motion (ROM) within six weeks following ligamentous knee reconstruction.

A recent review assessed the effectiveness and safety of the procedure done in conjunction with manipulation under anesthesia (MUA). Eight studies involving 240 patients were included in the analysis, and the results indicated

that arthroscopic LOA and MUA significantly improved the range of motion (41.6º). Moreover, clinically relevant improvements in various outcome measures were observed. Regarding safety, only one complication (a synovial fistula) was resolved without further intervention. These findings suggest that arthroscopic LOA and MUA are safe and effective treatment for postoperative knee arthrofibrosis.[83] Specifically looking at the pediatric population, a recent study revealed that LOA/MUA significantly improved ROM in 90% of patients, with only 10% requiring revision surgery. Preoperative dynamic splinting enhanced preoperative flexion but did not considerably influence postoperative ROM or the likelihood of treatment failure.[82]

Many have advocated for a short course of aggressive inpatient physical therapy, often with the use of continuous epidural anesthetic.

Conclusion

Arthrofibrosis in pediatric patients has similarities to the adult population but also exhibits distinctions requiring a nuanced approach. A deep understanding of these differences is essential for effective arthrofibrosis management in the pediatric population, from injury prevention to surgical procedures to restore knee function. Emphasizing prevention through early mobilization and timely surgical interventions, when necessary, is a crucial aspect of managing arthrofibrosis and its debilitating impact. Additionally, exploring non-surgical or minimally invasive treatment modalities, such as physiotherapy and pharmacological therapies, is vital for the future of arthrofibrosis management. As research in this field expands, focusing on the pediatric population is essential, as it will require unique perspectives and customized interventions to achieve optimal patient outcomes.

References

1. Freeman MA, Pinskerova V. The movement of the normal tibio-femoral joint. *J Biomech*. 2005; 38(2): 197-208. doi: 10.1016/j.jbiomech.2004.02.006.
2. Shelbourne KD, Wilckens JH, Mollabashy A, DeCarlo M. Arthrofibrosis in acute anterior cruciate ligament reconstruction. The effect of timing of reconstruction and rehabilitation. *Am J Sports Med*. 1991; 19(4): 332-6. doi: 10.1177/036354659101900402.
3. Magit D, Wolff A, Sutton K, Medvecky MJ. Arthrofibrosis of the knee. *J Am Acad Orthop Surg*. 2007; 15(11): 682-94. doi: 10.5435/00124635-200711000-00007.
4. Perry J, Antonelli D, Ford W. Analysis of knee-joint forces during flexed-knee stance. *J Bone Joint Surg Am*. 1975; 57(7): 961-7.
5. Gogia PP, Braatz JH, Rose SJ, Norton BJ. Reliability and validity of goniometric measurements at the knee. *Phys Ther*. 1987; 67(2): 192-5. doi: 10.1093/ptj/67.2.192.
6. Schlegel TF, Boublik M, Hawkins RJ, Steadman JR. Reliability of heel-height measurement for documenting knee extension deficits. *Am J Sports Med*. 2002; 30(4): 479-82. doi: 10.1177/03635465020300040501.
7. Shelbourne KD, Patel DV, Martini DJ. Classification and management of arthrofibrosis of the knee after anterior cruciate ligament reconstruction. *Am J Sports Med*. 1996; 24(6): 857-62. doi: 10.1177/036354659602400625.
8. Strum GM, Friedman MJ, Fox JM, Ferkel RD, Dorey FH, Del Pizzo W, et al. Acute anterior cruciate ligament reconstruction. Analysis of complications. *Clin Orthop Relat Res*. 1990; (253): 184-9.
9. Harner CD, Irrgang JJ, Paul J, Dearwater S, Fu FH. Loss of motion after anterior cruciate ligament reconstruction. *Am J Sports Med*. 1992; 20(5): 499-506. doi: 10.1177/036354659202000503.
10. Dodds JA, Keene JS, Graf BK, Lange RH. Results of knee manipulations after anterior cruciate ligament reconstructions. *Am J Sports Med*. 1991; 19(3): 283-7. doi: 10.1177/036354659101900313.
11. Shelbourne KD, Nitz P. Accelerated rehabilitation after anterior cruciate ligament reconstruction. *Am J Sports Med*. 1990; 18(3): 292-9. doi: 10.1177/036354659001800313.
12. Fisher SE, Shelbourne KD. Arthroscopic treatment of symptomatic extension block complicating anterior cruciate ligament reconstruction. *Am J Sports Med*. 1993; 21(4): 558-64. doi: 10.1177/036354659302100413.
13. Noyes FR, Mangine RE, Barber SD. The early treatment of motion complications after reconstruction of the anterior cruciate ligament. *Clin Orthop Relat Res*. 1992; (277): 217-28.
14. Watson RS, Gouze E, Levings PP, Bush ML, Kay JD, Jorgensen MS, et al. Gene delivery of TGF-β1 induces arthrofibrosis and chondrometaplasia of synovium *in vivo*. *Lab Invest*. 2010; 90(11): 1615-27. doi: 10.1038/labinvest.2010.145.
15. Unterhauser FN, Bosch U, Zeichen J, Weiler A. Alpha-smooth muscle actin containing contractile fibroblastic cells in human knee arthrofibrosis tissue. Winner of the AGA-DonJoy Award 2003. *Arch Orthop Trauma Surg*. 2004; 124(9): 585-91. doi: 10.1007/s00402-004-0742-x.
16. Younai S, Venters G, Vu S, Nichter L, Nimni ME, Tuan TL. Role of growth factors in scar contraction: an *in vitro* analysis. Ann Plast Surg. 1996; 36(5): 495-501. doi: 10.1097/00000637-199605000-00011.
17. Rockey DC, Bell PD, Hill JA. Fibrosis--A common pathway to organ injury and failure. *N Engl J Med*. 2015; 373(1): 96. doi: 10.1056/NEJMc1504848.
18. Iredale JP, Thompson A, Henderson NC. Extracellular matrix degradation in liver fibrosis: Biochemistry and regulation. *Biochim Biophys Acta*. 2013; 1832(7): 876-83. doi: 10.1016/j.bbadis.2012.11.002.
19. Usher KM, Zhu S, Mavropalias G, Carrino JA, Zhao J, Xu J. Pathological mechanisms and therapeutic outlooks for arthrofibrosis. *Bone Res*. 2019; 7: 9. doi: 10.1038/s41413-019-0047-x.
20. Lee DR, Therrien E, Song BM, Camp CL, Krych AJ, Stuart MJ, et al. Arthrofibrosis nightmares: prevention and management strategies. *Sports Med Arthrosc Rev*. 2022; 30(1): 29-41. doi: 10.1097/JSA.0000000000000324.
21. Dagneaux L, Owen AR, Bettencourt JW, Barlow JD, Amadio PC, Kocher JP, et al. Human fibrosis: is there evidence for a genetic predisposition in musculoskeletal tissues? *J Arthroplasty*. 2020; 35(11): 3343-52. doi: 10.1016/j.arth.2020.05.070.
22. Vander Have KL, Ganley TJ, Kocher MS, Price CT, Herrera-Soto JA. Arthrofibrosis after surgical fixation of tibial eminence fractures in children and adolescents. *Am J Sports Med*. 2010; 38(2): 298-301. doi: 10.1177/0363546509348001.
23. McLennan JG. The role of arthroscopic surgery in the treatment of fractures of the intercondylar eminence of the tibia. *J Bone Joint Surg Br*. 1982; 64(4): 477-80. doi: 10.1302/0301-620X.64B4.6896515.
24. Mah JY, Adili A, Otsuka NY, Ogilvie R. Follow-up study of arthroscopic reduction and fixation of type III tibial-eminence fractures. *J Pediatr Orthop*. 1998; 18(4): 475-7.
25. Bram JT, Aoyama JT, Mistovich RJ, Ellis HB Jr, Schmale GA, Yen YM, et al. Four risk factors for arthrofibrosis in tibial spine fractures: a national 10-site multicenter study. *Am J Sports Med*. 2020; 48(12): 2986-93. doi: 10.1177/0363546520951192.
26. Edmonds EW, Fornari ED, Dashe J, Roocroft JH, King MM, Pennock AT. Results of displaced pediatric tibial spine fractures: a comparison between open, arthroscopic, and closed management. *J Pediatr Orthop*. 2015; 35(7): 651-6. doi: 10.1097/BPO.0000000000000356.
27. Patel NM, Park MJ, Sampson NR, Ganley TJ. Tibial eminence fractures in children: earlier posttreatment mobilization results in

improved outcomes. *J Pediatr Orthop*. 2012; 32(2): 139-44. doi: 10.1097/BPO.0b013e318242310a.
28. Adams AJ, O'Hara NN, Abzug JM, Aoyama JT; Tibial Spine Research Group; Ganley TJ, et al. Pediatric type II tibial spine fractures: addressing the treatment controversy with a mixed-effects model. *Orthop J Sports Med*. 2019; 7(8): 2325967119866162. doi: 10.1177/2325967119866162.
29. Aoyama JT, LaValva SM, Bram JT, Reese T, Ganley TJ. Comparing rates of tibial spine fractures to ACL tears: a 7-year trend. *Orthop J Sports Med*. 2020; 8(4 suppl 3): 2325967120S00173. doi: 10.1177/2325967120S00173.
30. Lafrance RM, Giordano B, Goldblatt J, Voloshin I, Maloney M. Pediatric tibial eminence fractures: evaluation and management. *J Am Acad Orthop Surg*. 2010; 18(7): 395-405. doi: 10.5435/00124635-201007000-00002.
31. Shin YW, Uppstrom TJ, Haskel JD, Green DW. The tibial eminence fracture in skeletally immature patients. *Curr Opin Pediatr*. 2015; 27(1): 50-7. doi: 10.1097/MOP.0000000000000176.
32. Smith HE, Cruz AI Jr, Mistovich RJ, Leska TM, Ganley TJ, Aoyama JT, et al. What are the causes and consequences of delayed surgery for pediatric tibial spine fractures? A multicenter study. *Orthop J Sports Med*. 2022; 10(3): 23259671221078333. doi: 10.1177/23259671221078333.
33. Anderson CN, Anderson AF. Tibial eminence fractures. *Clin Sports Med*. 2011; 30(4): 727-42. doi: 10.1016/j.csm.2011.06.007.
34. Yaru NC, Daniel DM, Penner D. The effect of tibial attachment site on graft impingement in an anterior cruciate ligament reconstruction. *Am J Sports Med*. 1992; 20(2): 217-20. doi: 10.1177/036354659202000222.
35. Romano VM, Graf BK, Keene JS, Lange RH. Anterior cruciate ligament reconstruction. The effect of tibial tunnel placement on range of motion. *Am J Sports Med*. 1993; 21(3): 415-8. doi: 10.1177/036354659302100315.
36. Shelbourne KD, Patel DV. Treatment of limited motion after anterior cruciate ligament reconstruction. *Knee Surg Sports Traumatol Arthrosc*. 1999; 7(2): 85-92. doi: 10.1007/s001670050127.
37. Markolf KL, Hame S, Hunter DM, Oakes DA, Zoric B, Gause P, et al. Effects of femoral tunnel placement on knee laxity and forces in an anterior cruciate ligament graft. *J Orthop Res*. 2002; 20(5): 1016-24. doi: 10.1016/S0736-0266(02)00035-9.
38. Howell SM, Taylor MA. Failure of reconstruction of the anterior cruciate ligament due to impingement by the intercondylar roof. *J Bone Joint Surg Am*. 1993; 75(7): 1044-55. doi: 10.2106/00004623-199307000-00011.
39. Simmons R, Howell SM, Hull ML. Effect of the angle of the femoral and tibial tunnels in the coronal plane and incremental excision of the posterior cruciate ligament on tension of an anterior cruciate ligament graft: an *in vitro* study. *J Bone Joint Surg Am*. 2003; 85(6): 1018-29. doi: 10.2106/00004623-200306000-00006.
40. Markolf KL, Burchfield DM, Shapiro MM, Davis BR, Finerman GA, Slauterbeck JL. Biomechanical consequences of replacement of the anterior cruciate ligament with a patellar ligament allograft. Part I: insertion of the graft and anterior-posterior testing. *J Bone Joint Surg Am*. 1996; 78(11): 1720-7. doi: 10.2106/00004623-199611000-00013.
41. Pinczewski LA, Deehan DJ, Salmon LJ, Russell VJ, Clingeleffer A. A five-year comparison of patellar tendon versus four-strand hamstring tendon autograft for arthroscopic reconstruction of the anterior cruciate ligament. *Am J Sports Med*. 2002; 30(4): 523-36. doi: 10.1177/03635465020300041201.
42. Sajovic M, Vengust V, Komadina R, Tavcar R, Skaza K. A prospective, randomized comparison of semitendinosus and gracilis tendon versus patellar tendon autografts for anterior cruciate ligament reconstruction: five-year follow-up. *Am J Sports Med*. 2006; 34(12): 1933-40. doi: 10.1177/0363546506290726.
43. Mayr HO, Weig TG, Plitz W. Arthrofibrosis following ACL reconstruction--reasons and outcome. *Arch Orthop Trauma Surg*. 2004; 124(8): 518-22. doi: 10.1007/s00402-004-0718-x.
44. Cosgarea AJ, Sebastianelli WJ, DeHaven KE. Prevention of arthrofibrosis after anterior cruciate ligament reconstruction using the central third patellar tendon autograft. *Am J Sports Med*. 1995; 23(1): 87-92. doi: 10.1177/036354659502300115.
45. Schachter AK, Rokito AS. ACL injuries in the skeletally immature patient. *Orthopedics*. 2007; 30(5): 365-70; quiz 371-2. doi: 10.3928/01477447-20070501-01.
46. McIntosh AL, Dahm DL, Stuart MJ. Anterior cruciate ligament reconstruction in the skeletally immature patient. *Arthroscopy*. 2006; 22(12): 1325-30. doi: 10.1016/j.arthro.2006.07.014.
47. Pavlovich R, Goldberg SH, Bach BR. Adolescent ACL injury: treatment considerations. *J Knee Surg*. 2004; 17(2): 79-93. doi: 10.1055/s-0030-1248203.
48. Nwachukwu BU, McFeely ED, Nasreddine A, Udall JH, Finlayson C, Shearer DW, et al. Arthrofibrosis after anterior cruciate ligament reconstruction in children and adolescents. *J Pediatr Orthop*. 2011; 31(8): 811-7. doi: 10.1097/BPO.0b013e31822e0291.
49. Kocher MS, Garg S, Micheli LJ. Physeal sparing reconstruction of the anterior cruciate ligament in skeletally immature prepubescent children and adolescents. Surgical technique. *J Bone Joint Surg Am*. 2006; 88 Suppl 1 Pt 2: 283-93. doi: 10.2106/JBJS.F.00441.
50. Fanelli GC. Posterior cruciate ligament injuries in trauma patients. *Arthroscopy*. 1993; 9(3): 291-4. doi: 10.1016/s0749-8063(05)80424-4.
51. Fanelli GC, Edson CJ. Posterior cruciate ligament injuries in trauma patients: Part II. *Arthroscopy*. 1995; 11(5): 526-9. doi: 10.1016/0749-8063(95)90127-2.
52. Al-Ahaideb A. Posterior cruciate ligament avulsion fracture in children: a case report with long-term follow-up and comprehensive literature review. *Eur J Orthop Surg Traumatol*. 2013; 23 Suppl 2: S257-260. doi: 10.1007/s00590-012-1146-1
53. Pandya NK, Janik L, Chan G, Wells L. Case reports: pediatric PCL insufficiency from tibial insertion osteochondral avulsions. *Clin Orthop Relat Res*. 2008; 466(11): 2878-83. doi: 10.1007/s11999-008-0373-6.
54. Sanders TL, Pareek A, Barrett IJ, Kremers HM, Bryan AJ, Stuart MJ, et al. Incidence and long-term follow-up of isolated posterior cruciate ligament tears. *Knee Surg Sports Traumatol Arthrosc*. 2017; 25(10): 3017-23. doi: 10.1007/s00167-016-4052-y.
55. Shelbourne KD, Clark M, Gray T. Minimum 10-year follow-up of patients after an acute, isolated posterior cruciate ligament injury treated nonoperatively. *Am J Sports Med*. 2013; 41(7): 1526-33. doi: 10.1177/0363546513486771.
56. Shelbourne KD, Muthukaruppan Y. Subjective results of nonoperatively treated, acute, isolated posterior cruciate ligament injuries. *Arthroscopy*. 2005; 21(4): 457-61. doi: 10.1016/j.arthro.2004.11.013.
57. Wegmann H, Janout S, Novak M, et al. Surgical treatment of posterior cruciate ligament lesions does not cause growth disturbances in pediatric patients. *Knee Surg Sports Traumatol Arthrosc*. 2019; 27(8): 2704-9. doi: 10.1007/s00167-018-5308-5.
58. Sorensen OG, Fauno P, Christiansen SE, Lind M. Posterior cruciate ligament reconstruction in skeletal immature children. *Knee Surg Sports Traumatol Arthrosc*. 2017; 25(12): 3901-5. doi: 10.1007/s00167-016-4416-3.
59. Kocher MS, Shore B, Nasreddine AY, Heyworth BE. Treatment of posterior cruciate ligament injuries in pediatric and adolescent patients. *J Pediatr Orthop*. 2012; 32(6): 553-60. doi: 10.1097/BPO.0b013e318263a154.
60. Salmons HI, Payne AN, Taunton MJ, Owen AR, Fruth KM, Berry DJ, et l. Nonsteroidal anti-inflammatory drugs and oral corticosteroids mitigated the risk of arthrofibrosis after total knee arthroplasty. J *Arthroplasty*. 2023; 38(6S): S350-4. doi: 10.1016/j.arth.2023.03.076.
61. Trousdale WH, Salib CG, Reina N, Lewallen EA, Viste A, Berry DJ, et al. A drug eluting scaffold for the treatment of arthrofibrosis. *Tissue Eng Part C Methods*. 2018; 24(9): 514-23. doi: 10.1089/ten.TEC.2018.0136.
62. Insall JN, Hood RW, Flawn LB, Sullivan DJ. The total condylar knee prosthesis in gonarthrosis. A five to nine-year follow-up of the first one hundred consecutive replacements. *J Bone Joint Surg Am*. 1983; 65(5): 619-28.

63. Rand JA, Ilstrup DM. Survivorship analysis of total knee arthroplasty. Cumulative rates of survival of 9200 total knee arthroplasties. *J Bone Joint Surg Am*. 1991; 73(3): 397-409.
64. Schiavone Panni A, Cerciello S, Vasso M, Tartarone M. Stiffness in total knee arthroplasty. *J Orthop Traumatol*. 2009; 10(3): 111-8. doi: 10.1007/s10195-009-0054-6.
65. Kim YM, Joo YB. Prognostic factors of arthroscopic adhesiolysis for arthrofibrosis of the knee. *Knee Surg Relat Res*. 2013; 25(4): 202-6. doi: 10.5792/ksrr.2013.25.4.202.
66. Ibrahim IO, Nazarian A, Rodriguez EK. Clinical management of arthrofibrosis: state of the art and therapeutic outlook. *JBJS Rev*. 2020; 8(7): e1900223. doi: 10.2106/JBJS.RVW.19.00223.
67. McAlister I, Sems SA. Arthrofibrosis after periarticular fracture fixation. *Orthop Clin North Am*. 2016; 47(2): 345-55. doi: 10.1016/j.ocl.2015.09.003.
68. Moore TJ, Harwin C, Green SA, Garland DE, Chandler RW. The results of quadricepsplasty on knee motion following femoral fractures. *J Trauma*. 1987; 27(1): 49-51. doi: 10.1097/00005373-198701000-00009.
69. Oliveira VG, D'Elia LF, Tirico LE, Gobbi RG, Pecora JR, Camanho GL, et al. Judet quadricepsplasty in the treatment of posttraumatic knee rigidity: long-term outcomes of 45 cases. *J Trauma Acute Care Surg*. 2012; 72(2): E77-80. doi: 10.1097/ta.0b013e3182159e0a.
70. Zhang D, Nazarian A, Rodriguez EK. Post-traumatic elbow stiffness: Pathogenesis and current treatments. *Shoulder Elb*. 2020; 12(1): 38-45. doi: 10.1177/1758573218793903.
71. Smith SP, Devaraj VS, Bunker TD. The association between frozen shoulder and Dupuytren's disease. *J Shoulder Elbow Surg*. 2001; 10(2): 149-51. doi: 10.1067/mse.2001.112883.
72. Bunker TD, Anthony PP. The pathology of frozen shoulder. A Dupuytren-like disease. *J Bone Joint Surg Br*. 1995; 77(5): 677-83.
73. Blessing WA, Okajima SM, Cubria MB, Villa-Camacho JC, Perez-Viloria M, Williamson PM, et al. Intraarticular injection of relaxin-2 alleviates shoulder arthrofibrosis. *Proc Natl Acad Sci U S A*. 2019; 116(25): 12183-92. doi: 10.1073/pnas.1900355116.
74. Dehghan F, Soori R, Dehghan P, Gholami K, Muniandy S, Azarbayjani MA, et al. Changes in knee laxity and Relaxin receptor isoforms expression (RXFP1/RXFP2) in the knee throughout estrous cycle phases in rodents. *PLoS One*. 2016; 11(8): e0160984. doi: 10.1371/journal.pone.0160984.
75. Ekhtiari S, Horner NS, de Sa D, Simunovic N, Hirschmann MT, Ogilvie R, et al. Arthrofibrosis after ACL reconstruction is best treated in a step-wise approach with early recognition and intervention: a systematic review. *Knee Surg Sports Traumatol Arthrosc*. 2017; 25(12): 3929-37. doi: 10.1007/s00167-017-4482-1.
76. Stinton SK, Beckley SJ, Branch TP. Efficacy of non-operative treatment of patients with knee arthrofibrosis using high-intensity home mechanical therapy: a retrospective review of 11,000+ patients. *J Orthop Surg*. 2022; 17(1): 337. doi: 10.1186/s13018-022-03227-w.
77. Biggs A, Shelbourne KD. Use of knee extension device during rehabilitation of a patient with type 3 arthrofibrosis after ACL reconstruction. *N Am J Sports Phys Ther*. 2006; 1(3): 124-31.
78. Finger E, Willis FB. Dynamic splinting for knee flexion contracture following total knee arthroplasty: a case report. *Cases J*. 2008; 1(1): 421. doi: 10.1186/1757-1626-1-421.
79. Bonutti PM, McGrath MS, Ulrich SD, McKenzie SA, Seyler TM, Mont MA. Static progressive stretch for the treatment of knee stiffness. *Knee*. 2008; 15(4): 272-6. doi: 10.1016/j.knee.2008.04.002.
80. Bonutti PM, Marulanda GA, McGrath MS, Mont MA, Zywiel MG. Static progressive stretch improves range of motion in arthrofibrosis following total knee arthroplasty. *Knee Surg Sports Traumatol Arthrosc*. 2010; 18(2): 194-9. doi: 10.1007/s00167-009-0947-1.
81. Pace JL, Nasreddine AY, Simoni M, Zurakowski D, Kocher MS. Dynamic splinting in children and adolescents with stiffness after knee surgery. *J Pediatr Orthop*. 2018; 38(1): 38-43. doi: 10.1097/BPO.0000000000000730.
82. Werner BC, Cancienne JM, Miller MD, Gwathmey FW. Incidence of Manipulation Under Anesthesia or Lysis of Adhesions After Arthroscopic Knee Surgery. *Am J Sports Med*. 2015; 43(7): 1656-61. doi: 10.1177/0363546515578660.
83. Fackler N, Chin G, Karasavvidis T, Bohlen H, Smith E, Amirhekmat A, et al. Outcomes of arthroscopic lysis of adhesions for the treatment of postoperative knee arthrofibrosis: a systematic review. *Orthop J Sports Med*. 2022; 10(9): 23259671221124911. doi: 10.1177/23259671221124911.

Conflict of interests: [Faust and Castañeda] or any member of their immediate family, has no funding or commercial associations (e.g., consultancies, stock ownership, equity interest, patent/licensing arrangements, etc.) that might pose a conflict of interest in connection with the submitted article.