Trauma Case Reports 46 (2023) 100866

Contents lists available at ScienceDirect

Trauma Case Reports

journal homepage: www.elsevier.com/locate/tcr

Case Report

Uncommon, foreign-body induced knee arthrofibrosis in a pediatric patient

Franziska Kocher, Yoann Durand, Ines Raabe, Moritz Tannast, Daniel Petek *

Department of Orthopedic Surgery and Traumatology, HFR-Hospital Cantonal, UNIFR, 1700 Fribourg, Switzerland

ARTICLE INFO

Keywords:
Arthrofibrosis
Foreign body
ACL avulsion

ABSTRACT

Arthrofibrosis is defined as an excessive fibrotic tissue response within a joint leading to a painful loss of motion. This pathological scar formation process with dysregulated, inordinate extracellular matrix formation, especially collagen, may occur in any joints, although is frequently localized in the knee. Different etiologies have been described and most of them are related to trauma, infection or recent surgical procedure. Although arthrofibrosis affects people of all ages, it is unusual in pediatric population. We present a case report of an uncommon, foreign body induced knee arthrofibrosis in a 14-year old boy. We also review the current literature regarding diagnostic procedures and treatment rationale for arthrofibrosis of the knee.

Introduction

Arthrofibrosis is a fibrotic joint disorder with excessive collagen production that promotes adhesions within the joint capsule, tendon and bursa resulting in reduced range of motion (ROM), pain and swelling. Scarred tissue may create a chronic impingement within the joint that sustains a viscous circle of inflammation, leading to constant fibrotic scar formation [1]. Considering the knee, the patient's disability aside from pain is especially due to loss of the knee motion, in extension (which impair walking) and in flexion (which creates difficulties by climbing stairs, getting up or sitting) [2].

Depending on the location within that joint and the extent of fibrosis, there is a great variation of clinical symptoms, ranging from a local disturbance like cyclops lesions after anterior cruciate ligament reconstruction, up to an excessive global response [3]. Frequent affected anatomic structures are Hoffa's fat pad (infrapatellar fat pad), suprapatellar pouch, anterior interval, intercondylar notch, medial and lateral gutter and posterior capsule [3]. Adhesions around Hoffa's fat pad and patellar tendon may produce patellofemoral pain with limited flexion or generate a patella baja that is responsible of a patello femoral overload and a possible osteoarthritis at long-term. The implication of the posterior capsule limits full extension leading to the previously described gait abnormalities.

Etiology

Different etiologies have been described and most of them refer to trauma, infection or surgery [1]. The precise and pathologic pathways are still poorly understood. Among others are mentioned a dysregulated inflammatory and immunological process, a mechanical (trauma, surgery) stimulus and also even physical rehabilitation or immobilization [1]. However, arthrofibrosis is most often seen after surgery [1,4].

* Corresponding author.
E-mail address: daniel.petek@h-fr.ch (D. Petek).

https://doi.org/10.1016/j.tcr.2023.100866
Accepted 3 June 2023
Available online 5 June 2023

Case report

A 14-years old boy consulted our outpatient clinic because of increasing anterior knee pain especially during sports activities, associated with a feeling of progressive loss of motion. The past medical history revealed an anterior tibial spine avulsion type Meyers-McKeever III that has required an open reduction an internal fixation two years ago (Fig. 1). This procedure was followed by hardware removal one year later (Fig. 2).

Physical examination of the knee revealed a swelling of the soft tissues around the patellar ligament, a small amount of effusion, no ligament laxity and a ROM of 95–0-0°. The patello-femoral joint was entrapped with no medial or lateral glide. The American Knee Society Score (AKS) was 73 of 100 points. Additional investigations (standard x-rays, MRI) reported an intra-articulaire fibrous tissue localized behind the patellar ligament and detected a surgical thread running from the Hoffa fat pad through the patellar ligament to the antero-medial soft tissue. Among other noticeable MRI findings was a severe inflammation of the patellar ligament (Fig. 3). Unfortunately, we could not recall the surgical procedure description to give us more information about a possible failure of the material removal.

The foreign body, a Ti-Cron thread, 6 cm long, has been first fully removed from its intraarticular location, followed by an arthroscopic arthrolysis to debride the anterior scar tissue and to recover a proper ROM. Directly after the surgical procedure. Histologic probes have been send for histopathologic and microscopic investigations and showed chronic fibrous scar tissue as well as infiltration by inflammatory cells like macrophages (Fig. 4a–b).

Immediately after surgery, the ROM reached 125–0-0°. Intensive physiotherapy as well as continuous passive motion started immediately post-operatively. 6 months after surgery, the patient regained free function with a ROM of 120–0-5°and did not report pain at rest, raising to 1–2 points of 10 on visual analog scale (VAS) during sport activities. The AKS improved from 73 to 90.

Discussion

Although arthrofibrosis is a rare complication after knee surgery in pediatric patients, the patient has presented the classical symptoms of this severe and disabling pathology [3]. Even a small loss of ROM creates a high disability of the knee joint and is poorly tolerated, especially during sport activities [3]. For activities of daily life and normal gait, the patients need at least full extension and 90–120° of flexion. Already 5° of extension loss impairs walking [3]. The localization of the fibrotic tissue detected by MRI was typical for the knee joint [3], especially around patella tendon and Hoffa's fat pad, but also around the medial and lateral gutter leading to an entrapped patella.

Werner et al. reported a rate of arthrofibrosis up to 8 % after knee surgeries, excluding total-knee-replacement, with a need of manipulation under anesthesia (MUA) or arthroscopic arthrolysis in all cases [2]. The authors insisted on the fact that even simple diagnostic arthroscopies may lead to arthrofibrosis.

In 2016, an international multidisciplinary expert consensus outlined knee arthrofibrosis as “post-operative fibrosis of the knee defined as a limited ROM in extension and/or flexion” under exclusion of other specific cause. The other causes mentioned are those that are not attributable to an osseous or prosthetic obstruction to physiological movement (malalignment, malposition or incorrectly sized components, presence of hardware, ligament reconstruction, infection (septic arthritis), pain, chronic regional pain syndrome)

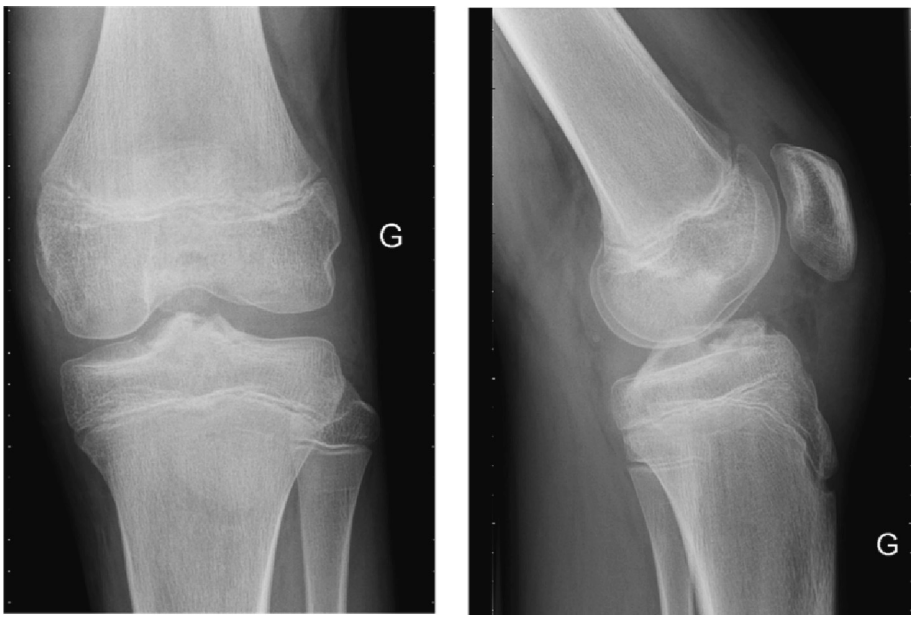

Fig. 1. (a–b): Standard X-Ray AP/lateral of left knee showing an anterior tibial spine avulsion (Meyers-McKeever III).

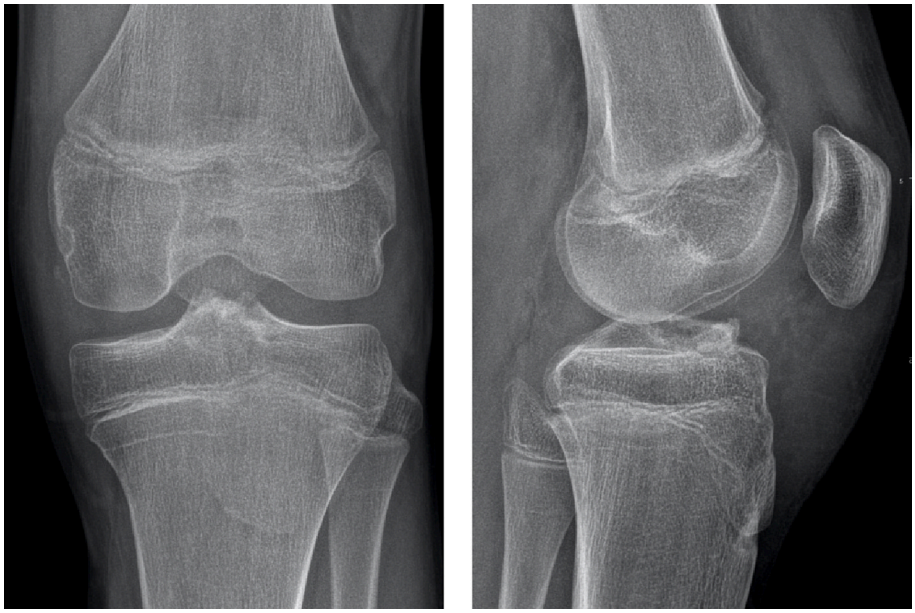

Fig. 2. (a–b): Standard X-Ray AP/lateral of left knee 3 months after open reduction and internal fixation with a Ti-Cron Thread.

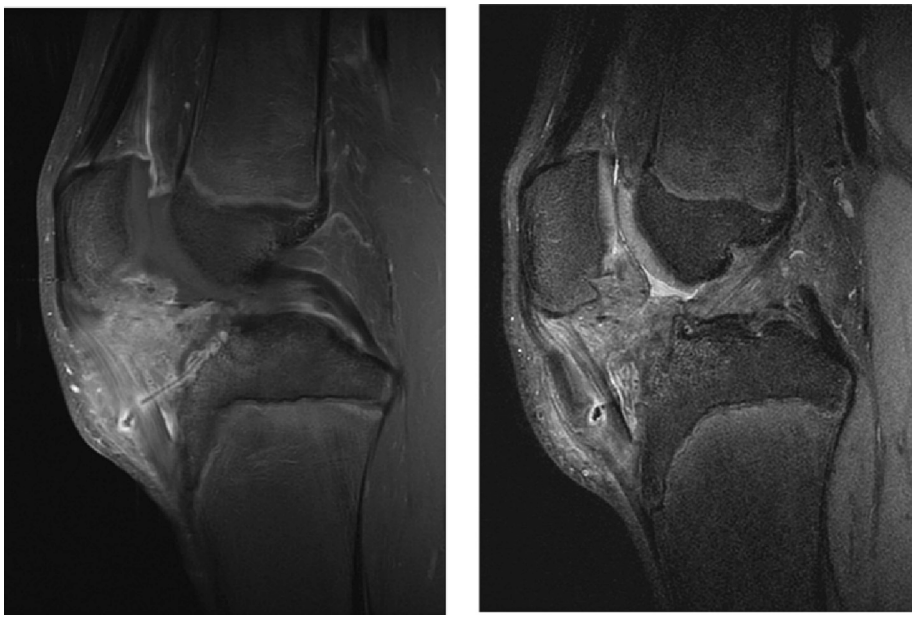

Fig. 3. (a–b): MRT T1 FS with Gadolinium sagittal views showing a foreign body running from the anterior tibial spine through the fat pad into the patellar tendon. Severe inflammatory response is visualized.

[5]. The authors noted that pain was considered as an important aspect of this disease. The degree of severity is depending on the limitation of flexion and extension; from mild impairment with a flexion up to 90–100° and a loss of extension of 5–10° to severe with flexion less than 70° and loss of extension of more than 20° [5].

A classification was proposed by Shelbourne to describe arthrofibrosis after ACL reconstruction. The limitation of this classification is the mandatory extension loss, which is not always present and which therefore excludes many patients who report only pain and flexion limitation [6].

Histologically, arthrofibrosis is marked by a dysregulated and enhanced myofibroblastic proliferation due to an inflammation process with reduced cellular apoptosis leading to an excessive formation of extracellular matrix (ECM) primarily composed of collagen. This results in fibrosis and contraction of the joint pouches. Regularly, heterotrophic ossifications are concomitantly found

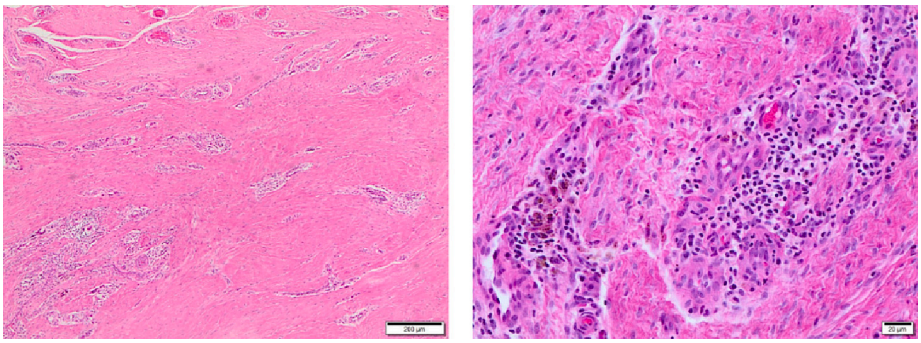

Fig. 4. a; HE stain magnification 100× with areas of fibrous scar tissue including collagen
b: HE stain magnification 200× with infiltration by acute and chronic inflammatory cells, predominantly macrophages.

[7]. Upregulation and maintenance of inflammation is carried out by inflammatory cytokines. The transforming growth factor-β (TGF-β) as inflammatory cytokine, plays an important role in the collagen synthesis and therefore also in the dysregulated fibrosis.

The histopathologic findings focus on tissue composition, structure and cellular activity in order to confirm the presence of fibrotic tissue and the excessive amount of ECM. Standard histological stains are sufficient to visualize fibrosis, although the results depend on the type of biopsy and the donor side [1].

Several predisposing have been described. These factors are associated with a dysregulation of the inflammation process or autoimmune diseases like diabetes type II or rheumatoid arthritis.

Sanders et al. expressed the rates of arthrofibrosis between females and males. They published that there is a 2.5–2.8 times higher rate of arthrofibrosis in women than men [8,9]. Among the possible explanations are the different immunological responses with higher prevalence of 80 % of autoimmune disease in women and higher prevalence of osteoarthritis with subsequent inflammatory process.

Treatment options

Current treatment concepts are focused to regain full ROM through a stepwise approach including different strategies like pharmaceutical treatments, physical rehabilitation therapy, mobilization under anesthesia (MUA), arthroscopic and open articular release.

Because fibrosis is due to a dysregulated inflammatory response, pharmacological treatments are focused on inhibition of inflammatory pathways and cytokines. In a clinical setting, NSAIDS are used, as well as corticosteroids in oral or injected form.

The physical rehabilitation therapy aims to increase strength and ROM around the affected joint. The use of continuous passive motion is controversial in terms of economic costs and forced over-bending, although patients with arthrofibrosis will benefit from a slightly better ROM [10]. Too aggressive physical therapy may worsen or even initiate arthrofibrosis by triggering an inflammatory response as described by Cheuy and Millett et al. [5,11].

The preferred treatment for arthrofibrosis is arthroscopic arthrolysis [12] associated with mobilization under anesthesia (MUA), which can also be performed alone if the sufficient amount of mobility is achieved. These procedures are nevertheless associated with a risk of hemarthros, wound complications, heterotopic ossification, tendon rupture, cartilage damage and even fractures [3,12].

The postulated advantage of surgical treatment with a debridement of fibrotic tissue is the interruption of the feedback loops between myofibroblast activation and fibrosis expression by profibrotic and inflammatory mediators. On the other hand, MUA and surgery lead to tissue damage that may increase the inflammatory process, thus increasing fibrosis and worsen symptoms. The success of surgical release is difficult to assess, as it depends on the severity of the fibrosis and the inflammation, making comparison of the different patients difficult [3].

Newer therapeutic approaches focus on the inflammation process. The use of pharmacological molecules such as TGF-β antibodies to interrupt the feedback loop between inflammatory cytokine and myofibroblasts proliferation have shown promising preliminary results and are expected to enhance the success of standard treatments.

Conclusion

An intra-articulaire foreign body may induce a chronic inflammatory response that may lead to a secondary arthrofibrosis. Complete surgical removal of the foreign body combined with arthroscopic arthrolysis is mandatory to allow the recovery of knee function. In this particular condition, manipulation under anesthesia alone would not be sufficient to treat the arthrofibrosis. Therefore, and before any type of treatment, a proper diagnosis and consideration of primary or secondary causes of arthrofibrosis are mandatory to obtain a successful result.

Patient consent

Written consent to publish the case report was obtained from the patient and father.

Funding

No funding or grant support.
All authors attest that they meet the current ICMJE criteria for Authorship.

Declaration of competing interest

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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