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Arthrofibrosis — a myth or true joint disorder?

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Abstract: Authors, mostly specialists on rehabilitation and orthopedic surgery prove that arthrofibrosis is a commonly overlooked phenomenon, which may lead to serious limitation in the range of movement, leading to limitation in patients quality of functioning.
The main goal of this article is to emphasize the importance of understanding a such complex condition. Non typical patomechanism, lack of biomarkers dedicated to this dysfunction and general lack of understanding in this pathology causes that risk factors and the most effective strategies remain vastly unknown. Pathophysiology of the arthrofibrosis in the joints is definitely multifactorial, but intense production of collagen seems to be the main factor. Most modern pharmacological methods concentrate on the regulation of collagen fiber production and reducing the inflammation.
Inflammation from joint contractures stimulates the proliferation of activated cells that results in the production of extracellular matrix macromolecules to form fibrotic tissue that is deposited into the capsule, thereby resulting in fibrosis.
Lack of unified classification scale is caused by relatively high variation of the functions fulfilled by particular joints and each treatment plan should be constructed individually. Quality of surgical treatment and physical therapy play a major role in both prevention and treatment of such complex condition as arthrofibrosis.
Both iatrogenic mistakes and overly aggressive manual therapy are some of main factors increasing the risk of this pathological condition. Introducing properly conducted physical therapy treatment in the early stage is crucial to main the range of movement and preventing this significant problem.

Keywords: arthrofibrosis, knee joint, trauma, rehabilitation, physical therapy, range of motion.

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Introduction

Etymology of the term arthrofibrosis (AF) originates from Greek and it means: arthros (joint), fibros (fibrous), and osis — irregularity or proliferation. In this particular case suffix "osis" means fibrosis, increase of the volume of the fibrous connective tissue in the region of the injury. All this referring to the diarthroses is unequivocal with the limitation of the motion cause mostly by pathologic scar tissue formation [1].

It seems that athrofibrosis is a structural disturbance of the joint fibrous connective tissue, which begins as a trauma-, operation- or infection-initiated inflammation.

Proliferation of the extracellular matrix accompanied by formation of adhaesions causes shrinkage of joint elements, such as synovial bursae, tendons, synovial plicae and villi, adipose bodies, what causes suffers as pain and limitation of motion in the joint.

Inevitable consequence of these conditions is a significant deterioration of patient's life quality. Limitation of the joint motion is an obstacle in the normal functioning, what is manifested in problems with motion and performing the simpliest actions during a day [2, 3].

Arthrofibrosis is a relatively common phenomenon — i.e. in 1997–2000 it concerned up to 15% of patients operated because of necessity of reconstruction of the anterior cruciate ligament (ACL), while 13 years later this percentage dropped to 7% [4, 5]. It is difficult to estimate clearly what may cause over double drop in the frequency of this disease. It is supposed that the main factors which cause reduction are postoperative rehabilitation introduced in the early stage and also application of mini-invasive Techniques (i.e. arthroscopy and arthroplasty; meniscal suturing, ligamentous replacement and transplant). No less important included: properly chosen pharmacological treatment, as well as raising awareness of the medical staff and patients concerning the subject of the arthrofibrosis (properly conducted environmental education).

Arthrofibrosis — definition

In available literature there is a lack of representative, standard definition for arthrofibrosis, and previous scientific reports concentrate mainly around the clinical aspects of the phenomenon. Arthrofibrosis is a lesion that usually occurs as a result of trauma or postoperatively, significantly changing the joint biomechanics. Accompanying inflammation begins and specifically directs the origin and dissemination of the adhesions. It is relatively common incidence, and as its consequence it results in proliferation of the fibrous connective tissue within the joint and in its vicinity, which leads to severe limitation or even loss of motion in the joint. The adhesions depending on their

localization bring restrictions to adhesion of the joint elements and may initiate its degeneration. Pain symptoms usually arise with motion restrictions [2].

For example Shelbourne *et al.* [6], who evaluated arthrofibrosis in the knee joint, defined it as 15° loss of extension, and proposed own system of classification based on the loss of flexion and extension which included also the patellar tightness, and also too low set patella [7].

Mayr *et al.* described the arthrofibrosis as a development of the scar fibrous tissue in at least one joint compartment, causing deficit of the range of motion [8]. According to Bosch *et al.* [9] arthrofibrosis can be described as severe complication that occurs in a joint, as a result of its trauma or surgical procedure, which is associated with a complete loss of motion in the joint due to excessive fibrosis in course of repair. He divides it into primary and secondary. Patients with primary arthrofibrosis suffer from a general reaction of fibrosis in the processes of the repair after trauma or surgical procedure, while in patients with the secondary fibrosis the limitation of the range of motion is caused by local fibrosis. In example the inappropriate, inadequate placement of the implant of the cruciate ligament, trauma of the intercondylar fossa or peri-operational complications (i.e. problems with appropriate instruments used during the procedure) may lead to secondary fibrosis [9]. Arthrofibrosis is described also as abnormal proliferation of the fibrous connective tissue inside or outside the joint, that results in the motion limitation, pain, weakness, edema and lesion of the joint functioning [10, 11].

Epidemiology

The authors who carried out the researches in 90s of the XX century estimated that arthrofibrosis develops in 35% of the patients who underwent the reconstruction of the anterior cruciate ligament [12].

However the application of more advanced operation techniques as well as improved rehabilitation protocols caused significant decrease of the arthrofibrosis — till 4% [13].

Arthrofibrosis develops more commonly in patients subjected to the high-energetic and multi-ligamentous trauma than in patients suffering from low-energetic lesion of a single ligament [14] Noyes *et al.* In their studies noticed that arthrofibrosis exists in 23% of patients following unanimous reconstruction of the anterior cruciate ligament and the tibial collateral ligament [15].

During simultaneous reconstruction of the anterior and the posterior cruciate ligaments the loss of full degree of movements is possible up to 57% [14, 16].

Traumatic knee subluxation causing variable patterns of the ligamentous instability results in a highest frequency of movement loss. Sisto and Warren observed problems with motion in 6 out of 20 patients (30%) [17].

Arthroscopic procedures in the shoulder joint may cause arthrofibrosis which is estimated for 1–2.8% [18–20]. The frequency of the arthroscopic repair of the lesion of the rotator cuff ranges from 4 to 9% [21, 22].

The frequency of the arthrofibrosis in other joints has not been the subject of profound studies yet and hasn't been estimated.

Hypotheses on the mechanism of development of the arthrofibrosis

Despite numerous attempts to describe the precise mechanism of the pathogenesis of the arthrofibrosis none of the presented hypotheses was fully accepted [23].

Apoptosis and fibrosis are the most important processes ending the inflammation and facilitating processes of repair. Apoptosis, programmed cellular death, in opposition to the necrosis, is not causing the inflammation. Process of fibrosis is an example of repair reaction and is dependent on the activation of the connective tissue responsible for so called tissue homeostasis [24]. In arthrofibrosis, during fibrosis activated myofibroblasts do not initiate apoptosis after wound healing but continue matrix synthesis, causing formation of pathologic scar tissue. Lack of balance between the synthesis and degradation of the extracellular matrix (ECM) results in overstorage of ECM particles, i.e. collagen and proteoglicans of the intercellular space [25, 26].

Proteoglycans consist of a core-protein covalently linked to glycosaminoglycan chains. Glycosaminoglycans regulate important cellular functions including proliferation, sequestration, and release of growth factors and maintain cartilage hydration [27, 28]. Glycosaminoglycan biosynthesis is initiated by xylosyltransferase-I and -II (XT-I/-II, EC 2.4.2.26), both Golgi-resident isoenzymes catalyzing the rate-limiting step in proteoglycan glycosylation. Since XT are secreted into the ECM with the xylosylated acceptor proteoglycan, quantification of serum XT activity provides a powerful technique for monitoring disregulated tissue remodeling processes [29, 30]. Upregulation of XT activity in serum as well as increased cellular XYLT1 expression are correlated with disorders of proteoglycan accumulation, e.g. scleroderma and liver fibrosis [31, 32]. Furthermore, it could be demonstrated that XYLT1 expression is induced by TGF-β 1 in the early onset of OA cartilage repair, while XYLT1 expression is reduced by IL-1β in late stage OA. Therefore, XT is a central regulator of cartilage destruction, cartilage repair, and glycosaminoglycan homeostasis in fibrosis and degenerative joint diseases [33, 34].

Despite much more frequent application of preventives after the Surgical procedurę it seems that arthrofibrosis indicators remain rather constant. Lack of understanding the role of the inflammation may lead to „over-agressive" physiotherapeutic programs. Aggressive exercises may initiate or worsen fibrosis of the joints, because they may evoke the inflammation reactions, increase of the pro-inflammatory cyto-

kines, collagen and TGF-β production, which are disregulated during fibrosis. Some patients inform that their signs started (or became significantly worse) after they were instructed that they should exercise despite the pain during the rehabilitation or after they performed more exhaustive exercises [3].

Risk factors

Factors of the risk for arthrofibrosis have been best described for the knee joint where it is rather common [3]. Many factors may have influence of the final degree of movements in the knee — they are associated mostly with the type of trauma, operation time (how fast after lesions is it performed), preoperative treatment, technical aspects of the procedure, and post-operative treatment and physical therapy [35].

The risk factors of arthrofibrosis in the knee joint can be divided into pre-operative, intra-operative, and post-operative.

Pre-operative risk factors

- Degree of injury

Patients with knee sprain have higher risk of problems with the degree of movements. Lesions that arise as a result of high-energetic trauma and cause extreme edema of the soft tissues, hematomas, muscle lesion etc. must be treated before considering the soft tissues reconstruction [35].

- Pre-operative problems

The delay of the procedure until the moment of restoring the full degree of movements, edema disappearance, and strengthening the muscles (i.e. in ACL lesion — the quadriceps femoris muscle) is beneficial for the decrease of the post-operative risk of arthrofibrosis [35].

Shelbourne *et al.* Noticed in their studies that arthrofibrosis affects more the patients who underwent ACL reconstruction within a week after the lesion than patients who were operated later, at least 21 days from the moment of the lesion [6].

Intra-operative risk factors

- Technical errors during the operation

The proper placement of the graft during ACL reconstruction is a key factor in avoiding the limitation in the degree of movements. Positioning the graft of the ligament anterior to its attachment before tearing results in impingement on the roof of the intercondylar notch in extension. Overly lateral position of the ligament on the tibia may cause the impingement on the lateral wall of the intercondylar

notch, while overly anterior and medial placement may limit the knee flexion. When it comes to the femoral part the most frequent error is too far anteriorly placed graft, which may cause its overload leading to limitation of the flexion and potential lack of success of the transplant [14]. It is recommended to preserve a 3 mm distance between the anterior portion of the intercondylar notch and the graft to prevent impingement [35].

- Inappropriate tension of the transplant during the ACL reconstruction

  Too much tension on the ACL graft may lead to disturbances in the kinetics of the knee joint.

Postoperative factors

- Course of postoperative rehabilitation

  Contemporary rehabilitation programs assume the quick mobilization of the joint and muscle strengthening within the time of postoperative treatment. During the postoperative physical therapy a patellar mobilization should be performed to avoid subpatellar contracture.
- Immobilization

  Early mobilization of the knee joint decreases the pain and postoperative exudation, helps to prevent scar tissue formation and muscular atrophy. It participates in nourishing of the articular cartilage [35].
- Infection

  Infection may cause loss of particular range of movement after knee joint ligaments reconstruction. Intraarticular mediators of the inflammation responsible for infection cause synovitis and degeneration of the articular surface. Deficit in range of movements is caused directly by fibrous scar formation initiated by local cytokine activation and indirectly by the pain caused by the tissue swelling and joint irritation [14].

Others

- Complex Regional Pain Syndrome (syndrome of Sudeck / algodystrophic syndrome)

  Angodystrophic syndrome inhibits patients effective participation in the postoperative rehabilitation. Increased sensitivity to pain, chronic edema, muscle atrophies and movement avoidance create vicious circle leading to development of the intrarticular adhesions and arthrofibrosis [14, 35].
- Sex

  According to Csintalan *et al.* Arthrofibrosis is more frequent in females [36].

Types of arthrofibrosis

In the available literature unanimous classification of the discussed sickness does not exist. However the arthrofibrosis of the knee joint was most frequently described.

With reference to the most commonly cited four-level stiffness of the knee joint classification proposed by Shelbourne *et al.* [6] one can distinguish the following types of movement limitation:

- I° — lack of limitation of the flexion and maximally 10° of the extension deficit,
- II° — lack of limitation of the flexion and at least 10° of the extension deficit,
- III° — decreased patellar mobility, limitation of the flexion above 25°, at at least 10° extension deficit,
- IV° — more than 30° limitation of the flexion, associated with the extension deficit above 10° and shortening of the patellar ligament.

Some authors postulate that when the degree of the knee movement is equal to 70°, one can diagnose the arthrofibrosis. According to Cosgarea the factor determining this pathology is the limitation of the extension and the flexion of at least 10°, while the other authors suggest that 10° of the extension deficit is enough and/or 95° of flexion [8, 15, 37–39].

According to the elbow joint it is worth to use the classification primarily proposed by Kay [40].

I° — soft tissue contracture,
II° — soft tissue contracture associated with calcification,
III° — intraarticular fracture without dislocation, with soft tissue contracture,
IV° — intraarticular fracture with dislocation and soft tissue contracture,
V° — presence of posttraumatic bony bridges.

Morrey [41] proposed the classification of the posttraumatic elbow stiffness into extrinsic, intrinsic and mixed contracture.

Intrinsic stiffness concerns the articular surfaces (intraarticular adhesion, deficits of the articular cartilage, poor adherence of the articular surfaces), while it is opposite when it comes to the extrinsic (capsule and ligamentous contracture, heterotopic ossification, exogenous lesions of articular surface adherence, soft tissue contracture caused by inflammation. Most of the cases of posttraumatic elbow stiffnes shows both extrinsic and intrinsic features [42].

Classification of arthrofibrosis in different body regions

- The knee joint

There are dozens of systems that classify the knee joint arthrofibrosis — part of them is based on the certain limitation of the movements, another on the localization of the scar tissue, and next on the range of movements in the joint involved (Table 1) [35].

Table 1. Systems of classification of knee joint arthrofibrosis according to various researchers.

Author	System of classification
Sprague *et al.* (1982)	I: Discreet bands or a single sheet of adhesions traversing the suprapatellar pouch II: Near-complete obliteration of suprapetallar pouch and peripatellar gutters with masses of adhesions III: Multiple bands of adhesions or complete obliteration of suprapatellar pouch with extracapsular involvement
Del Pizzo *et al.* (1985)	Classification based on the deviation from the full extension and the current range of flexion mild: <5 extension, >110 flexion moderate: 5–10 extension, 90–100 flexion severe: >10 extension, <90 flexion
Paulos *et al.* (1987)	Three stages of infrapatellar contracture syndrome : I: Precursor stage (2–8 week after operation): hardening of the synovial capsule, fat pad, retinaculum marked by painful motion, quadriceps muscle lag II: Active stage (6–20 week after operation): peripatellar swelling, significantly limited patellar mobility, hardening of the anterior tissues, step-off between patellar ligament and tibial tuberosity III: Residual stage (>8 months after operation): atrophy of fat pad, patellofemoral crepitus and arthrosis, depressed patella, quadriceps femoris atrophy
Blauth and Jaeger (1990)	Classification based on complete reflex arch: I (mild): >120° II (moderate): 80°–120° III (severe): 40°–80° IV (extreme): <40°
Shelbourne *et al.* (1996)	Classification based on the deviation from the full flexion and extension of the knee on the opposite side to the arthrofibrosis: I: <10° extension, range of flexion correct II: >10° extension, range of flexion correct III: >10° extension, >25° loss of flexion IV: >10° extension, >30° loss of flexion, depressed patella

The most popular classification of the arthrofibrosis for the knee joint is given by Shelbourne [3, 7]. In the past it has been widely used but it refers mainly to arthrofibrosis after ACL reconstruction. Using these criteria the diagnosis of the arthrofibrosis requires patient who suffer from loss of the extension, excluding the individuals who suffer from the pain and loss of flexion.

Mild, moderate, and severe arthrofibrosis was described by different ranges of flexion — 90°–100°, 70°–89°, less than 70°, and/or loss of extension: 5°–10°, 11°–20° and more than 20° respectively [7].

In knees the suprapatellar pouch, anterior interval, intercondylar notch, medial and lateral gutters, posterior capsule and infrapatellar fat pad (IFP or Hoffa's fat pad), may all be affected, with symptoms varying depending on the location and extent of adhesions, but typically involving loss of flexion and/or extension.

- Shoulder complex

Arthrofibrosis of the shoulder complex can be classified as idiopathic or acquired. "Frozen shoulder" is another commonly used term for idopathic artrofibrosis. Despite the fact that this term is widely spread, there is no agreement regarding to the diagnostic criteria of the frozen shoulder. Its characteristic features are lack of preceding trauma or surgical intervention within the shoulder complex, significantly limited passive and active range of motion in comparison to the opposite shoulder joint, lack of visible pathologies in the imaging. Acquired arthrofibrosis of the shoulder complex develops secondary to the intrinsic or extrinsic process. These processes are trauma, i.e. humerus fractures [43].

- The wrist

Lee *et al.* (2006) proposed classification of the wrist arthrofibrosis based on its location and functional limitations [44].

Patomechanism of development of arthrofibrosis

Current reports published in medical journals postulate that arthrofibrosis touches all age groups (although it is definitely rare in children). There is little known about the patomechanism of the disease. There is a lack of biomarkers dedicated to this dysfunction, and general lack of understanding of patomechanism in this pathology causes that risk factors and the most effective strategies are not known very well. Some of the present researches help to understand this phenomenon [3, 45]. Pathophysiology of the arthrofibrosis in the joints is multifactorial, and the main effect of it is the intense production of collagen. Most modern pharmacological methods concentrate on the regulation of collagen fiber production and reduction of the inflammation [46].

Changes in the connective tissue begin at the moment of influence of the stress factor (trauma, operation, infection) on the cells of the immune system, which stimulate cytokin cascade and a number of other mediators causing transformation of fibroblast into myofibroblasts. The latter secrete collagen fibers and transform growth factor β (TGF-β). Previously mentioned reactions result in disregulation of the physiological healing processes (positive feedback) [45].

It is stated that two types of arthrofibrosis exist: so called active and rudimentary. In the latter form, despite the fibrotic processes have been finished, the joint remans stiff. Depending on the type of arthrofibrosis the treatment significantly differs — surgery is a standard treatment of choice, but in the future it might be the pharmacological therapy that corrects the signal path for the immune cells.

Myofibroblasts are able to revert their differentiation, so understanding of the mechanisms of the pathogenesis might be the key for cell therapy development (i.e. inhibition of the signal path for TGF-β) [45].

Each diarthrodial joint has its mechanical parameters and the norms of the range of the movements. Crossing those ranges is conditional for recognition of the pathology, i.e. arthrofibrosis.

Arthrofibrosis of the big diarthrodial joints, a fibrosis which occurs in the elbow, wrist, shoulder, hip, knee and the ankle joints result in loss of function and increasing immobilization. The degree of the stiffness and fibrosis depends on the specification of joint in which it occurs.

In example the contracture of the elbow joint has been defined as a loss of extension of more than 30° and loss of flexion less than 120° [47].

In comparison arthrofibrosis of the shoulder known as frozen shoulder or adhesive shoulder capsulitis causes more than 25% loss of movements at least in two planes, usually in external rotation and the abduction.

On the other hand the definitions of the knee stiffness vary. They are described frequently as flexion range of motion less than 75° and 15° (or more) limitation of extension [48].

Lack of unified classification scale is caused by relatively high variation of the functions fulfilled by particular joints thus each case should be considered individually.

Biology of joint contracture formation

All large synovial joints (i.e. shoulder, elbow, wrist, hip, knee and ankle) are restrained by capsules, which are sleeves of fibrous connective tissue composed mainly of collagen fibers. Collagen molecules are composed of three α-chains that form a procollagen triple helical structure in the endoplasmic reticulum; they have short telopeptides and N-terminal/C-terminal propeptides on the ends. Procollagen propeptides flank the N and C termini. Proline and lysine residues of immature nascent collagen α-chains are hydroxylated after translation, with subsequent selective glycosylation of certain hydroxylysine residues. Premature aggregation of procollagen molecules is prevented by protein chaperones, such as protein disulfide isomerase (PDI), and these same protein chaperones help form the disulfide bonds in procollagen. These mature procollagen chains are then folded into triple-helical structures with the assistance of chaperones, such as heat-shock 70-kDa related luminal binding protein, heat-shock protein 47 (HSP47), and PDI [49]. When these chains enter the extracellular space, N and C-terminal propeptides are enzymatically cleaved, thereby forming collagen fibrils. Collagen fibril assembly is driven by site-specific interaction of collagen molecules and is stabilized by intra and intermolecular cross-links with oxidation of specific lysine residues catalyzed by lysyl oxidase [50]. This is followed by the sponta-

neous condensation of the resultant aldehydes with lysyl or hydroxylysyl residues on another alpha-chain either within the same triple helix or between triple helices. Inhibition of chaperones, such as HSP47, can interfere with procollagen triple helix production and contribute to extracellular matrix (ECM) degradation. Aberrations in collagen synthesis and orientation of collagen fibrils can occur, resulting in thickening of the posterior capsule and leading to joint contracture [51].

Joint contractures can be noninflammatory or inflammatory in nature. Noninflammatory joint contractures often result from congenital conditions, such as arthrogryposis, where there are abnormalities in the genes responsible for connective tissue development [52]. Inflammatory joint contractures are more common after posttraumatic conditions followed by prolonged joint immobilization and chronic disease. Inflammation from joint contractures stimulates the proliferation of activated cells that results in the production of ECM macromolecules to form fibrotic tissue that is deposited into the capsule, thereby resulting in fibrosis [53].

Histopathology of arthrofibrosis

Previous studies considering the structural changes of the course of arthrofibrosis did not reveal uniformity of modifications of specific elements of the connective tissue in a disease. According to current opinions no specific elements of the connective tissue were distinguished but they were described only as disseminated fibrosis [54, 55]. Similar changes were observed in the animal model [56].

The pathological reports and the descriptions in the literature were extremely variable and inconsistent. Jackson and Schaefer [54] in 1990 described a cyclops syndrome as a soft-tissue mass at the base of the ACL stump that caused isolated loss of extension. They described the arthroscopic appearance of the cyclops lesion as a nodular and vascular soft-tissue mass located anterolateral to the surgical site of the tibial tunnel. Microscopically the cyclops lesion showed peripheral fibrous tissue surrounding a central core of granulation tissue; several specimens were noted to contain bony and cartilaginous tissue. The cyclops lesion is felt to be a localized form of arthrofibrosis. Although some are convinced it is a mild form of the spectrum of arthrofibrosis, the others think it is a stretch to say it is universally accepted. Some orthopedists have felt it should be separated from global cases because clinically it behaves in a different way. Studies documenting the histopathology of arthrofibrosis opened the door to link the variously described clinical presentations into a spectrum of the same process.

In patients with patella infera syndrome, Noyes *et al.* described an extensive replacement of the fat pad with fibroconnective tissue in various stages of maturation [55–57]. They noted fibroblastic and endothelial proliferation among the dense collagen fiber formation.

Why the mode of physical therapy is so important?
— very often overly aggressive manual manipulation or operation may stimulate proliferation of the connective tissue and worsen the patient's condition,
— studies on the arthrofibrosis report that 4–35% of the patients suffer from loss of range of motion after surgery,
— loss of range of movements is diagnosed when the range of extension has a value higher than –10°, and range of flexion is less than 125°,
— localization of the adhesions holds great importance for the limitation of the range of movements -for example in the knee joint the adhesions localized in the vicinity of the anterior cruciate ligament or tibio-femoral joint may influent loss of the extension, while the same in the region of the patellofemoral joint may cause loss of flexion,
— usually pain accompanies the limitation of the movements.
Changes in the biomechanics of the knee joint:
— fibrosis "lowers" patella and causes so called "patella baja" or "infera",
— the further effect of the proces is shortening of the patellar ligament,
— the sliding force for tibia increases,
— pressure between femur and patella increases,
— may cause patellofemoral pain syndrome (PFPS),
— final result — athgrosis of the patellofemoral joint,
— 89% of the patients operated for arthrofibrosis revealed degeneration, besides it is a firm connection between the persistent reduction of the extension and the PFPS that results from weakening of the quadriceps femoris and the patellar pain.

Etiology of arthrofibrosis

— development of the arthrofibrosis of the knee joint is multifactorial, in the first place it is mentioned the immobilization in the plaster dressing or stabilizator, even lack of activity after contusion,
— extent of the trauma influences the arthrofibrosis (it predisposes to the arthrofibrosis),
— multi-ligamentous contusion with injury to ACL and tibial collateral ligament (TCL),
— it is postulated that also operation carried out within acute inflammation may cause arthrofibrosis, that is why it is suggested to delay the operation even for few weeks,
— intraoperative errors may also cause arthrofibrosis — i.e. incorrectly placed transplant, too high tension of the graft,
— delaying even for 1 day (24 hrs) the ROM exercises (range of movement) may increase the risk of the arthrofibrosis,

Important: it isn't enough to measure if the extension is 0°, but it is recommended always to compare it to the healthy limb because 96% of individuals are characterized by over-extension.

Prevention of arthrofibrosis

— early resurrection of the range of movements after trauma or operation,
— delay in operation treatment until the inflammation is gone,
— patient mobilization,
— most of the physicians and physical therapists use nowadays so called aggressive protocols (i.e. from Shelbourne's clinic),
— application of nonsteroidal anti-inflammatory drugs,
— cryotherapy.

Factors that increase risk of arthrofibrosis — treatment:
— hematoma of the joint,
— massive exsudate,
— immobilization,
— complicated contusions — fractures within the joint,
— biopsy or arthroscopy — aspiration,
— arthroscopic "cleaning" — application of CPM — stabilization and CPM,
— avoiding long lasting traction.

The key to the success after operation seems to be an immediate initiation of the exercises maintaining range of motion, while CPM is successful only within 2–3 following days after surgical procedure.

Conflict of interest

None declared.

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