ACL – deficient knee

Kolano bez więzadła krzyżowego przedniego

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Summary

Anterior cruciate ligament (ACL) acts as a main restraint of anterior translation of the tibia. ACL is intrasynovial but extraarticular, because it reflects synovium from posterior capsule of the knee joint and is covered by a synovial fold that serves as a main source of nutrition and vascularisation for ACL. Part of ACL tear may spontaneously heal inside the synovial coverage. Ligament is highly innervated, more than 1,5% of its volume is constituted by nerve endings, 4 types of receptors are detected inside the ACL, mainly in approximity of bone attachments. ACL serves as a main tract for proprioceptive reactions. In the knee joint ACL is gradually loaded as the knee extends. In the absence of sufficient coactivation from the hamstrings, the posterior pull they apply to the proximal tibia is not available, leaving the ACL as the sole structure to stabilize the joint. Latency of neuromuscular control of deformity in healthy individuals is 53 ms and among the ACL – deficient individuals latency is about 200 ms. In the absence of ACL patients tend to develop a quadriceps-avoidance gait to reduce a quadriceps contraction during walking and finally loose about 10% of quadriceps muscle strength. Untreated ruptures lead to anterior laxity and subsequent meniscal tears in a great majority of cases, but about 1/3 patients in a long-term may be asymptomatic, and 47% may for some time returned to amateur sport. Meniscal tears are observed among 86% patients with an ACL lesion. The clinical investigation is highly unreliable among these patients and arthroscopic assessment is necessary in such cases. Patients with an ACL tear and meniscal destruction develop degenerative joint disease (DJD) visible on X-ray, there's no good evidence of DJD progression among patients with isolated ACL lesions. DJD is visible in 65% of the ACL-deficient knees with meniscal lesions as early as 4,4 years post meniscectomy. [Acta Clinica 2002 2:11-16]

Key words: ACL-deficient knee, ACL tear, knee arthroscopy, sensorimotor control of the knee joint

Streszczenie

Unaczynienie WKP pochodzi w większości od tętnicy środkowej kolana, jak również z gałązek końcowych tętnic dolnych kolana przyśrodkowej i bocznej. Większość ukrwienia dociera z tylno-górnej okolicy przyczepu udowego. Naczynia krwionośne rozgałęziają się w błonie maziowej formując oplatającą więzadło sieć, która tworzy osłonkę odżywczą. Naczynia te komunikują się z siecią naczyń wewnątrzwięzadłowych. Połączenia więzadła z kością nie uczestniczą w zaopatrzeniu WKP w krew. Ponad 1,5% WKP stanowią nerwy. W więzadle stwierdza się 4 typy mechanoreceptorów, pomiędzy nimi są np. wolne zakończenia nerwowe, które inicjują ochronne napięcie mięśni już w 53 msek po urazie, podczas gdy w kolanie pozbawionym WKP ta reakcja jest opóźniona o koło 200 msek. WKP jest więc bardzo ważnym ogniwem w szlakach czucia głębokiego, jego impulsacja pełni podstawową rolę w regulacji odruchowego napięcia mięśni zapewniającego kontrolę stabilności kolana w mechanizmach tzw. kokontrakcji mięśniowej. Większość pacjentów po uszkodzeniu WKP zmienia stereotyp chodu, by uniknąć przedniego podwichnięcia piszczeli, które nasila się przy skurczu mięśnia czworogłowego (przy kącie 15 – 25° podwichnięcie jest największe) i rozwija chód z unikaniem jego aktywności. Nie leczone uszkodzenie WKP prowadzi do przedniej niestabilności stawu kolanowego, a w konsekwencji do uszkodzenia łąkotek, a następnie degeneracji chrząstki stawowej. U młodych i aktywnych pacjentów postęp choroby jest bardzo szybki, w grupie pacjentów starszych, o niewielkich oczekiwaniach ruchowych około 1/3 nie odczuwa dolegliwości z powodu braku więzadła, A 47% może na jakiś czas powrócić do amatorskiego uprawiania sportu. Bardzo niekorzystne rokowniczo są: występowanie objawów niestabilności rotacyjnej, np. testu pivot-shift, uszkodzenie łąkotek, nieprawidłowa oś kończyny, duża aktywność fizyczna chorego – często prowadząca do ponownych urazów. Uszkodzenia łąkotek towarzyszące zerwaniu WKP zaobserwowano u 86% pacjentów. Badanie kliniczne nie jest w pełni wiarygodne u tych pacjentów i wskazana jest artroskopia, celem operacji naprawczych łąkotek. Nie ma dowodów na to, że izolowane uszkodzenie WKP prowadzi do zmian zwyrodnieniowych stawu kolanowego, ale u pacjentów z rozległymi uszkodzeniami łąkotek 64% ma ewidentne cechy gonartrozy w badaniu rentgenowskim już w 4,4 lata po urazie. [Acta Clinica 2002 2:11-16]

Słowa kluczowe: kolano bez więzadła krzyżowego przedniego, uszkodzenie WKP, kontrola sensomotoryczna stawu kolanowego

Vascularisation

Anterior cruciate ligament (ACL) acts as a main restraint of anterior translation of the tibia. ACL originates from medial aspect of the lateral femoral condyle and passes anteriorly and medially to the posterior cruciate ligament (PCL) to insertion on the anterior aspect of the tibia in front of medial tibial eminence. ACL is intracapsular but extraarticular, because it reflects synovium from posterior capsule of the knee joint and is covered by a synovial fold that serves as a main source of nutrition and vascularisation for ACL. Vessels for ACL originate from middle genicular artery and

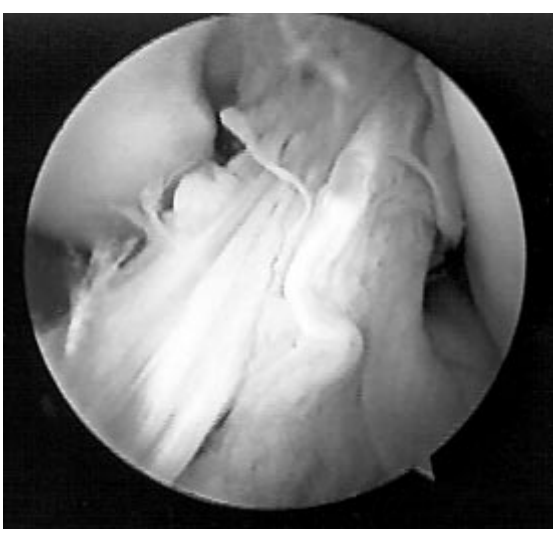

Figure 1. Partial lesion of anterior cruciate ligament – part of fibers outside the synovium, latero-posterior bundle inside the synovial coverage

from terminal branches of medial and lateral inferion genicular circumflex artery. Main part of that nutritional vessels penetrates from upper-posterior aspect of femoral attachment and they form a rich network of intraligamentary vessels. Bone attachments do not participate in nutrition of ACL, that's why part of ACL tear may spontaneously heal inside the synovial coverage (1, 2) (Fig. 1).

Neuromuscular control

5 types of mechanoreceptors appear inside the knee joint (Tab. 1), among them 4 types of receptors are detected inside the ACL (27). So that ligament is highly innervated, more than 1,5% of its volume is constituted by nerve endings, mainly in approximity of bone attachments, and it serves as a main tract for proprioceptive reactions. „Ligamento-muscular protective reflex" was proposed by Payr in 1900 as a part of „kinetic chain" theory according to which ligaments, bones, muscles and receptors acts synergistically to provide safe, stable motion of the joint. For example ACL is subjected by large forces, when tibia is displaced anteriorly beyond the physiologic strain limits and then receptors of ACL release contraction of hamstrings, which will protect against the anterior subluxation by pulling the tibia posteriorly (4, 13, 17). That observation was confirmed and documented in humans by Grüber in 1986 (11). All experiments have confirmed the hypothesis, that there is a direct neuromuscular link in humans in between a ACL and all the muscles surrounding the knee (9, 11, 27). Beard (4) defined a latency of neuromuscular control of deformity in healthy individuals as 53 ms and among the ACL – deficient individuals latency was doubled.

The most primitive neuromuscular reflex is flexion reflex called „withdrawal reflex", because it withdraw of the organism away from noxious stimulus that may harm it – e.g. extreme heat, and invariably is associated with pain. The ligamento-muscular reflex may be considered as an example of „withdrawal reflex" and is directly connected to the ligament receptors. In the knee its action consist of simultaneous hip or knee flexion approximating the limb to the trunk and away from stimulus. Parallel flexion of two joints earned this reflex the name „flexion". This is a spinal reflex,

Tab. 1. Types of mechanoreceptors of the knee joint (acc. to Solomonow M., Krogsgaard M: Sensorimotor control of knee stability. A review. Scand J Med Sci Sports 2001 11:64 – 80)

Receptor	Type of stimulus	Localization	Projection
Bare nerve endings	Extreme deformity, pain, inflammation	Articular surface, ligaments	Spinal cord (reflexes), sensory cortex
Ruffian endings	Low-level deformation (slow adapting)	Ligaments, menisci	Spinal cord (reflexes), sensory cortex
Pacinian corpuscles	High forces and pressure deformation	Ligaments, menisci	Spinal cord (reflexes), sensory cortex
Golgi receptors	Extreme forces	Tendons, ligaments, menisci, capsule	Spinal cord (reflexes), sensory cortex (capsule and menisci Golgi tendons organs) to cerebellum only (Golgi tendons organs)
Muscle spindles	Muscle elongation, velocity and acceleration	Muscles crossing the joint	Spinal cord (monosynaptic reflexes), cerebellum

that's why it is fast, short neural pathway eliminates the cooperation of brain and perceived sensation of pain is delayed 2 sec (4, 9, 22, 26).

Pope (22) identified an intentional voluntary contraction of the musculature for protecting the knee joint from potentially unstabilizing mechanical stimulus. He tested, whether an athlete could learn to contract the muscles across the knee in response to force applied to the joint and prevent subluxation. The patients were contracting their muscles immediately after application of a tap stimulus to the foot. Self-generated acquired contraction required 220 ms and was too long to protect the knee.

In 1909 Sherrington (7, quoted after 3) published his classical work where he noted that joint motion is always accompanied by co-contraction, or co-activation of the joint agonistic or antagonists muscles. For the knee extension agonistic quadriceps applies large forces and antagonistic hamstrings and biceps are antagonists and apply low-level forces. Sherrington thought it was a central mechanisms, but Feneys (9) demonstrated that it may also be a peripheral reflex. Antagonistic activity compensates the effect of gravity on the limb mass, opposes torque of an acting joint and maintains it despite changes in muscle length and moment arm around the center of rotation.

Sport, acquired skill has a significant impact on coactivation pattern of the muscles around the joint. Person as early as in 1957 was the first to show that as an athlete acquires a skill by practicing the repetitive joint motion, the antagonists coactivation level is markedly decreased, increasing a joint efficiency (4, 23).

In the knee joint ACL is gradually loaded as the knee extends (23). In the absence of sufficient coactivation from the hamstrings, the posterior pull they apply to the proximal tibia is not available, leaving the ACL as the sole structure to stabilize the joint and it puts the ACL of highly skilled athlete at a high risk of injury. Louie and Mote measured (17) that contraction of the

hamstring reduces rotary laxity of the joint by 76%, while contraction of quadriceps reduces laxity by 23%, the stiffness of the knee joint increased by two- to threefold when the muscles were active.

In the situation of absence of richly innervated ACL, other structures – capsule, menisci may activate antagonists muscles with significant delay (27).

Hirokawa (13, 27) showed, that isolated loading of quadriceps in ACL – deficient patient at 15° of flexion leads to 4 mm anterior displacement of tibia under load of only 12 kg. At 15° of flexion a 33% reduction is available by hamstrings, at 30° of flexion a 70% reduction is evident. The excessive anterior displacement of the tibia associated with isolated quadriceps contraction exhibits, why this muscle exhibits partial atrophy post ACL rupture. Quadriceps muscle atrophy was about 10% of its mass, mainly of vastus medialis and hamstrings about 4% (10). Patients tend to develop a quadriceps-avoidance gait to reduce a quadriceps contraction during walking.

Natural history of the ACL-deficient knee

In majority of cases with an evident trauma and with a post-traumatic haemarthrosis 72% to 84% presents lesion of ACL, approximately 70% total rupture, 30% partial (3, 8, 16, 19). In 86% ACL lesion was accompanied by major meniscal tears.

There are many factors that should be taken under the consideration, while planning the treatment of such a patients:

– categories of activity level – passive, active, sportsmen,

– type of athletic activity (jumping, twisting activity versus light, recreational pursuits),

– age – adolescents, middle aged, old,

– level of instability – in particular the presence of pivot shift sign,

– meniscal damage,

– other ligamentous lesions e.g. PCL,

– lower limb alignment,

– tibiofemoral crepitus,

– patellofemoral problems,

– patient compliance and expectations (20).

Untreated ruptures lead to anterior laxity and subsequent meniscal tears (15, 18) in a great majority of cases, but about 1/3 patients in a long-term may be asymptomatic, and 47% may for some time returned to amateur sport (6, 15, 18, 20). In the group of middle-aged, low expectation patients (6) good results may be expected after a conservative treatment, while the young, active athletes will not do well. On the other hand, the re-injury ratio is very high among the other group and may occur in 50% of young patients over one year (20). This observation is not confirmed by our own data.

Partial ACL-tear

Among patients with an ACL tear in about 30% of cases (in our material 21%) – (3) a partial tear is detected (21, 24). 38% of these patients progressed to complete deficiency – one half of fibers in 50%, 3/4th tears in 86%. So there's a chance to treat it conservatively in 2/3 of cases. Some of them may develop a Wittek mechanism of adhesions in between ACL and PCL thus somehow decreasing a degree of instability (16, 21, 24).

Meniscal tears are observed among 86% patients with an ACL tear (15). The clinical investigation is highly unreliable among these patients and arthroscopic assessment is necessary in such cases. Patients with an ACL tear and meniscal destruction develop degenerative joint disease (DJD) visible on X-ray, there's no good evidence of DJD progression among patients with isolated ACL lesions (12, 25). DJD is visible in 65% of the ACL-deficient knees as early as 4,4 years post meniscectomy. Secondary lesions

of menisci may develop in ACL deficient knee (28).

In the conservatively treated patients only 50% of results were graded as excellent or good, in surgical group 94% (7).

The ACL deficient patients often develop an unicompartmental arthritis of the knee, because the biomechanical effects of ACL deficiency predispose the knee to DJD, in particular among the patients with often „giving-way" episodes. Probably also o metabolic effect of inflammation caused by ACL stump resorbtion may have a negative effect on viscoelastical properties of joint fluid. Cameron reported elevated levels of cytokines: Interleukin 1 and 6, TNF-α; and keratan sulphate (5) in an ACL-deficient patient, that might be responsible for a quick progression of DJD.

Our own clinical observations considering a natural course of degenerative knee disease in ACL-deficient knee patients have been already published in Acta Clinica No 2 (3).

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