

Mechanism of Non-contact Anterior Cruciate Ligament Injury

What Does Delayed Onset Muscle Soreness Have to Do With Non-contact Anterior Cruciate Ligament Injuries?

Abstract of PhD Thesis

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1. INTRODUCTION

Anterior cruciate ligament (ACL) injury is when the ACL ligament is stretched, partially- or completely torn, while delayed onset muscle soreness (DOMS) is defined as delayed onset soreness, muscle stiffness, swelling, loss of force-generating capacity, reduced joint range of motion, and decreased proprioceptive function.

Vast majority of ACL injury is on a non-contact basis, comprising 70-84%. The exact mechanism of non-contact ACL (NC-ACL) injury is not completely known. The running theories of NC-ACL injuries are as follows: impingement on the intercondylar notch, quadriceps contraction, quadriceps-hamstring force balance, increased knee valgus or abduction moments, generalized joint laxity, knee recurvatum, ACL size and hormonal effects of estrogen on the ACL and the latest is the axial compressive forces on the lateral aspect of the joint. Impaired neuromuscular control also identified as a risk factor, like decreased neurocognitive function, increased trunk displacement after sudden force release and the weakened neuromuscular control of core and hip muscles.

I theorized that DOMS is an acute compression axonopathy at the proprioceptive sensory terminals in the muscle spindle caused by superposition of compression forces under an acute stress response. I proposed the same novel injury mechanism in NC-ACL injury where the initial cause could be an analog acute microinjury of the proprioceptive sensory nerve terminals in the periosteum of the proximal medial tibia in the form of a nerve compression or crush injury. Even the axial impulsive force theory attributed two compression forces as the cause of the damage in NC-ACL injury, namingly the compressing valgus force due to leg buckling and the compressive anterior force due to quadriceps contraction. I rather suggest that the superposition of compression forces cause the initial damage of a bi-phasic non-contact injury mechanism within the proximal medial tibia and in its periosteum, and an acute nerve compression or crush injury could prevail, like in DOMS.

I emphasize the similarity of the innervation of periosteal bone compartments and the muscle spindles, as both compartments are innervated by non-nociceptive and nociceptive sensory neurons. Accordingly, I suggest that damaging superposition of compression forces (including shear force) are microdamaging the proprioceptive sensory terminals of the muscle spindle in DOMS, while in NC- ACL injury the initial damaging superposition of compression forces (including shear force) are microdamaging the axon

terminals of the encapsulated sensory neurons in the periosteum of the proximal medial tibia in the form of a compression or crush nerve injury.

2. OBJECTIVES

The objective of my investigation is to examine the theory whether proprioceptive terminal microinjury could lead to non-contact injuries, like it is suspected in NC-ACL injury and DOMS. Furthermore, I found it important to test the hypothesis whether the infrapatellar nerve innervated area at the medial proximal tibia could have a potential role in the injury mechanism as is presumed by my theory.

It is known that repetitive eccentric contractions damage proprioception. Accordingly, I hypothesize that these repetitive unaccustomed or strenuous eccentric contractions could microdamage the proprioceptive axon terminals in the muscle spindles and even in the periosteum of the proximal medial tibia. This terminal microinjury is proposed to be a terminal arbor degeneration (TAD) like mechano-energetic lesion on the peripheral ends of these pseudounipolar proprioceptive somatosensory fibers. Paclitaxel-based chemotherapy also exhibits this type of terminal sensory lesion and evolves after a dose limiting, cumulative manner and not associated with Wallerian axonal degeneration.

I theorize that DOMS alters the static encoding of the mostly unaffected stretch reflex of the preprogrammed postural control. This suggested minor alteration is presumably due to the exchange of monosynaptic static encoding of the stretch reflex derived from impaired Type Ia afferents to Type II polysynaptic afferent ones. I propose that the encoding of the dynamic changes component of the sensory encoding is barely affected, like it could be observed in Paclitaxel-based chemotherapy.

I propose that the actual microdamage that could lead to this Type Ia impairment is an energy depleted mitochondria induced dysfunctional glutamate vesicular release and Piezo2 channelopathy. Noteworthy, that Piezo2 ion channels are the principal mechanotransduction channels for proprioception. The evoked secondary compensatory pathway on the spinal dorsal horn, as a consequence of this microdamage, is suggested to be preprogrammed. I recommend that this exchange of static monosynaptic Type Ia afferent connection to Type II polysynaptic afferent ones is represented in the delayed latency of the medium latency response (MLR) of the affected stretch reflex. Noticeable, that the conduction velocity of Type II sensory fibers is slower than Type Ia fibers and polysynaptic signaling is slower than monosynaptic signaling. Not to mention, that this

exchange to polysynaptic signaling is highly energy consuming neuro-energetically within the proprioceptive system, that is suggested to have resource limitations. Furthermore, numerous studies are pointing toward that MLR is dominantly Type II afferent mediated. Finally, it is important to know that both Types Ia and Type II sensory fibers transduce static encoding, but dynamic encoding is conveyed only by Type Ia fibers. This is the reason why I hypothesize that this switch of static encoding delays the medium latency response of the stretch reflex, leading to impaired proprioception of DOMS or in other non-contact injuries, like in NC-ACL injury.

My NC-ACL injury mechanism hypothesis suspects the above TAD like primary lesion of the proprioceptive terminal is at the periosteum of the medial proximal tibia. This hypothesis is based on the suggested to be analogous innervation of the medial ankle. Correspondingly, I propose that the infrapatellar branch of the saphenous nerve innervates not just the skin, but the periosteum and joint capsule as well, as it was demonstrated at the medial ankle. Accordingly, the second objective of my dissertation was to develop a novel measurement method and to demonstrate its applicability in order to approximate the point of attack of the resultant force (hereinafter knee point) in the knee in a geometric way. I hypothesize that in the most provocative position of the NC-ACL injury, the involvement of the point of attack of the resultant force will be at the medial proximal tibia due to the repetitive superimposed compression forces.

3. METHODS

3.1. Test measurement of the knee point at the NC-ACL injury provocative position

3.1.1. Participant

The tested individual has been a competitive handball player for more than 15 years. She suffered an ACL injury on her right knee three years ago on a handball match during the game, correspondingly she underwent an ACL reconstruction (ACLR). Before the test examination she was informed about the protocol, and signed a written consent. The Ethical Committee of the Hungarian University of Sports Science (TE-KEB/26/2021) approved the experimental study.

3.1.2. Procedure

The measurement was taken in the laboratory of the Department of Mechatronics, Optics and Mechanical Engineering Informatics, Budapest University of Technology and Economics. OPTITRACK (NaturalPoint, Corvallis, Oregon, USA) was used which is an optical motion analysis system of the laboratory. Markers were applied to the adequate anatomical points of the tested individual. After a brief warm-up session, three jump tasks were executed during the analysis by the participant. The first task was a vertical jump with both legs and landing on both feet. The second task was a vertical jump with both legs again and landing on the right (injured) foot. The third task was a vertical jump with both legs and landing on the left (healthy) foot. The optical motion detector system recorded all the three tasks.

3.1.3. Exercise protocol

The motion detection system recorded the exercise task with the Motive:Body software. We examined the intersection point of the tibia axis and sagittal knee plane from frontal view, because we wanted to show whether the point of attack of the resultant force skew to medial or lateral side after vertical jump. We approximated the axis of the tibia in a way that the midpoint of the connecting axis of the applied markers on the ankle and the midpoint of the connecting axis of the applied markers on the proximal tibia were connected with a line from a frontal direction. The approximation of the knee plane was carried out in a way that the midpoint of the connecting line of the applied markers on the distal femur and the midpoint of the connecting line of the applied markers on the proximal tibia were connected with a line from a frontal direction. We approximated the point of maximum force transfer with the point of intersection of the above lines and that is the knee point. The relative angle offset of the tibia was calculated with the assistance of the tibia axes. The relative angle offset of the tibia was continuously compared to the axis of the tibia at first frame of record.

We analyzed the cycles of the three separate vertical jump tasks independently. Furthermore, we investigated the biomechanical parameters of the cycles of each task, namely the knee joint flexion/extension angle, the intersection points of the tibia axis and the plane of the knee, and the relative angle offset of the tibia from frontal direction.

3.2. MLR study

3.2.1. Participants

Nine female professional handball players participated in the experiment. Before the experiment they were informed about the protocol, and signed a written consent prior to the electrophysiological measurements. The Ethical Committee of the Hungarian University of Sports Science (TE-KEB/26/2021) approved the experimental study.

3.2.2. Procedure

In my second study, I was to find out how an acute fatiguing task changes the time characteristics of the MLR based on the short latency response (SLR). The protocol consisted of two surface electromyography (sEMG) measurement recordings, one before and one after the fatiguing 20 minutes multistage fitness test. EMG activity of the vastus medialis (VMO) and the rectus femoris (REC) were recorded during the two measurements. The dominant leg of the handball players was measured.

3.2.3. Exercise protocol

The 20 minutes multistage fitness test is a test commonly used to measure aerobic fitness. This test consists of continuous running between two lines 20 meters apart in time to recorded beeps. The speed at the start of the test is slow. After one minute, an increase in speed happens, and the time between beeps decreases. Each minute time between beeps decreases so a level consists of 1 minute. If the line is reached before the beep sounds, the subject must wait until the beep sounds before continuing. If the line is not reached before the beep sounds, the subject is given a warning and after the second warning subject is eliminated.

3.2.4. Electromyography

For processing sEMG data Noraxon MyoResearch Master Edition 1.08.27 software was used. Frequency range under 5 Hz, over 350 Hz were cut, 50–60 Hz frequency domains were filtered and the whole signal was smoothened.

3.2.5. Statistics

Due to the limited sample size and for the purpose of selecting the adequate statistical procedure Shapiro Wilk's W test was calculated for checking the normal distribution of

the data. Only the VMO before the 20 m multistage test is not normally distributed, so for comparing the VMO data Mann-Whitney-U test, while comparing the REC data independent T-test is used. To further support the results of this study Effect size (ES-Cohen's d) value is calculated and included. Statistical Power is also calculated for the significant differences with significance level set for $p < 0.05$.

3.2.6. Muscle soreness questionnaire

Electronic statement form was sent out one day after the 20 minutes multistage fitness test to the professional handball players electronically to declare whether they experienced DOMS after the fitness test. All players sent back the statement form with their declaration within 48 hours.

4. RESULTS

4.1. Applicability of the novel measurement method for the knee point at the NC-ACL injury provocative position

The results of our measurement method of the knee point could suggest that they are in accordance with my NC-ACL injury hypothesis, namely the knee point of the tibia is suspected at the medial part of the proximal tibia in the provocative position. The knee point of the healthy lower limb skewed toward the medial head of the tibia during landing. Furthermore, the limited stress attenuation capacity of the injured leg correlates with other studies, since proprioceptive nerve injury is likely due to the NC-ACL injury itself and to reconstructive surgical intervention.

4.2. EMG activity of the observed muscles

After the evaluation of the obtained data determined from the measured sEMG signal, statistically significant increase is only observed in the latency of MLR compared with the SLR of rectus femoris muscle after the fatiguing protocol ($p < 0.05$, $ES = 1.14$, $power = 0.59$). Furthermore, increase in the latency of MLR was observed in all participants who experienced DOMS in our study. In no other comparisons between the before-after latency values significant difference could be observed.

Important to note the limitation that our sample size was low, as the calculated moderate power value suggests. In spite of that, the very high effect-size value shows that

the intervention in the fatiguing exercise protocol indeed had an effect on the neuromuscular system, which substantially supports my hypothesis.

5. CONCLUSION

The results of the present studies substantiate my hypothesis that DOMS inducing exercise delays the latency of the MLR. Moreover, the delayed latency of MLR supports the theory of muscle spindle involvement in DOMS. Furthermore, this muscle spindle involved impairment can be measured by EMG right after DOMS inducing exercise and hours before the onset of mechanical hyperalgesia. The study also indicates that SLR and LLR are unaffected by DOMS, which correlates with earlier findings and it is in accordance with my hypothesis.

This finding is a step forward, because it further substantiates the proprioceptive sensory involvement in non-contact injuries, like NC-ACL. I hypothesized that prior to NC-ACL injury the microdamaged proprioceptive fibers in the proximal medial tibia could alter the stretch reflex in the form of a delayed latency of MLR. Correspondingly, I theorized that the fatigued proprioceptors in the proximal tibia could become oedematous and entrapped in the constrained bony canals due to superimposed burst compression and shear forces. As a result, they could be more susceptible to compression or crush injury even at the proprioceptive sensory terminals. Our novel test measurements substantiated my hypothesis that the point of attack of the resultant force is in the medial part of the proximal tibia in the NC-ACL injury provocative position (that is the almost fully extended knee), which is the innervation area of the infrapatellar branch of the saphenous nerve.

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