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Risk diagnosis of minor muscle injuries in professional football players: when imaging cannot help out biology might

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Muscle strain accounts for ~30% of all football injuries[1] and engages medical staff in a complicated process of diagnostic and management. In 90% of the cases, muscle strains affect hamstrings, rectus femoris, adductors, soleus and gastrocnemius.[2 3] In particular, into the group of the so-called minor muscle injuries, a subgroup defined 'non-structural muscle injuries' (NSI) has been identified.[2] The NSIs do not present any visible muscle fibre lesion at MRI and ultrasound examination and are classified as grade 1a, 1b[2] or zero[3] according to the latest classifications.[2 3] Moreover, they are fare from being rare as they account for 30% – 40% of all muscle lesions recorded by professional football clubs.[1] Despite the fact that NSIs are not detected by imaging, they can have functional sequelae.[2 3]

THE CHALLENGE OF DIAGNOSING SUBTLE MUSCLE STRAINS

The player affected by NSI generally loses between 1 and 7 days of training/match exposure, and the diagnosis of this type of injury can be challenging. Many medical centres are not adequately specialised in sports medicine injuries and the imaging exams may be negative or, at the very most, reveal an ill-defined oedematous area that may be difficult to interpret.[2 3]

In such cases, doctors regularly hear players convey two 'typical complaints' during examination; 'I feel weakness in my muscle' and/or 'I feel stiffness in my muscle'.

But what do these expressive, though relatively vague descriptions mean in biological terms?

The feeling of muscle weakness

We speculate that the statement, 'I feel weakness in my muscle' may reflect an autophagic response of damaged fibres which can cause weakness sensation, triggered by both an enzymatic leakage and a possible metabolic exhaustion.[4] These mechanisms usually occur in muscle fibres that experience a depletion of glycogen storage after strenuous physical activity. Under such conditions, a breakdown of muscle ultrastructure with a loss of sarcomere organisation ensues[5] damage of this entity to the muscle ultrastructure has a metabolic and not a mechanical aetiopathogenesis.

The feeling of muscle stiffness

We speculate that the statement 'I feel stiffness in my muscle' can be related to a muscle injury that is too small to be seen by imaging but which would be visible under an electronic microscope (indeed, NSIs are ultrastructural lesions). This injury would be brought about by a mechanical process, more forceful than that causing the sensation of muscular weakness. Such mechanical damage incurred by the muscle ultrastructure could be further divided into two phases: an initial phase which takes place during physical activity and a later phase associated with a secondary, inflammatory response resulting in a painful condition approximately 24 hours after the triggering event.[4] The biological repercussions of the mechanical damage inflicted on the muscle ultrastructure involve: sarcomere damage, perturbation of the excitation-contraction coupling mechanism and a situation of calcium overload caused by Ca^{2+} spilling out from the damaged sarcoplasmic reticulum into the cytosol.[4]

From a practical point of view, because most clinics will not have MRI scanners handy, the medical staff are in a difficult position when a player or a member of the player's entourage challenges the working diagnosis and suggests shortening the recovery time. Yet, underestimating this clinical condition can lead to the exacerbation of ultrastructural injuries into structural injuries, that is, a higher grade tear (from grade 1 upwards), and potentially force the player to stop for several weeks. Such an exacerbation may jeopardise player availability.[6]

BIOMARKERS ARE NEEDED FOR A RESCUE!

Biological markers for such muscular damages, particularly to the ultrastructure,

could improve the challenges of managing this delicate situation, even if in reality a correct diagnosis does not necessarily lead to a better outcome. Serum creatine kinase (CKMM isoform), aldolase, aspartate aminotransferase and lactate dehydrogenase (LDH-1 and LDH-2) are classically considered the most convenient and sensitive serological biomarkers for muscle damage. However, due to their high interindividual variability and lack of sensitivity and specificity, utilisation of those serological markers is sometimes problematic.[7]

We argue for researchers and funders to ramp up their efforts to identify new biological markers for muscle damage. This could offer a new alternative given the actual failure of classical biomarkers. Currently, the most promising plasma biomarkers seem to be: calpain (n-calpain and μ-calpain), circulating miRNAs (miR208a-3p, miR208b-3p, miR206-3p, miR-133b-3p and miR-434–3 p), skeletal muscle troponin I (type I and type II skeletal troponin), fatty acid-binding protein 3 (cytoplasmic form), plasma myosin heavy chain fragment, interleukins (IL-1, IL-1β, IL-6), tumour necrosis factor and carbonic anhydrase isoenzyme III. Moreover, interesting and promising biomarkers for muscular lesions can be obtained from a simple urinalysis, specifically: urinary myoglobin, 1/3-methylhistidine and metalloproteinase (MMP2, MMP-3, MMP-7, MMP-9, MMP-14) and titin fragment excretion rate.[7 8]

Unfortunately, to date, only a few saliva constituents are associated with musculoskeletal tissue injuries.[8] It is likely that a single marker is not sufficient, and a combination of markers could be necessary. However, current knowledge on the true role and reliability of these biomarkers is not yet clear and needs further studies aimed at identifying blood and urine biomarkers for muscle damage that could be clinically relevant for the sports medicine practitioners.[7]

Therefore, in this editorial we 'call for action' in the field of sports medicine—specifically to increase research in for biomarkers to help clinicians manage muscle injuries when diagnostic imaging lacks sensitivity to detect early muscle injury.

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