Chapter

Tendon Injury Following Strenuous Activity: (Acute, Repetitive, and Chronic)

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Abstract

Tendon biomechanics are governed by tendon structure. The collagen fibers' "uncrimping effect," which transforms their mutually nonparallel orientation to parallel in response to external force, underlies the range of tendon elasticity. The Golgi tendon organs control tendon proprioception. The mechanosensing proprioception may help to some extent protect the mechanical integrity of the tendon; in degenerative tendons, it could be expressed by pain. The tendon's intrinsic structure may fail when the acute, chronic, or recurrent external load exceeds the tendon's structural and mechanical resistance. The most significant factor leading to tendon rupture is excessive load, either acute or repetitive. When aging or a chronic illness is present, the magnitude of the excessive load is reduced.

Keywords: tendon, stress, strain, golgi tendon organs, proprioception

1. Introduction

Regular exercise is necessary to keep our body parts healthy, and tendons are no exception. Tendinopathy involves damage and inflammation of certain tendons due to chronic overuse or acute injury.

Tendons' structure defines tendon biomechanics. When the environmental and mechanical loads exceed the maximal strain resistance of the tendon, it will structurally fail, and an eventual rupture of the tendon will occur. For this purpose, the damaging mechanical strain may be due to a high acute extensive load or lower repetitive loads that build up the critical extensive stress on the tendon. These two patterns distinguish between acute tendon structural failure and structural failure due to overuse loads, that is, acute tendon rupture vs. rupture following microtrauma to the tendon.

2. Pathophysiology

The tendon elasticity range is based on the "uncrimping effect" of the collagen fibers that change their mutual nonparallel orientation to parallel following external

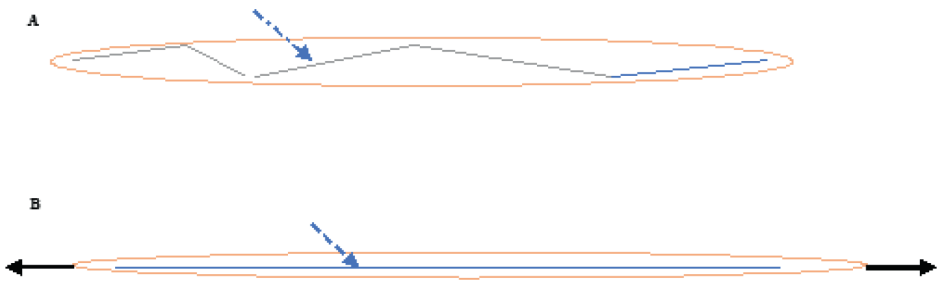

Figure 1.
A simplistic representation of the "uncrimping effect" in the tendon. A: Relaxed tendon with the nonparallel orientation of the collagen fibrils (dashed arrow). B: Stretched tendon by external loads (solid arrows). The collagen fibrils in a parallel orientation allow the elastic tendon elongation.

load (**Figure 1**) [1]. Beyond the elasticity range, with rising stress on the tendon, the integrity of the tendon is based on its collagen fiber content. In contrast, the latter integrity depends on its biochemical characteristics, which depend on age and/or systemic diseases. Therefore, the tendon tangent modulus, which represents the non-elastic range of stress effect on the tendon, depends on age and a specific biochemical environment following systemic illness [2].

The more prevalent systemic conditions that predispose to tendon degeneration are connective tissue diseases (Rheumatoid arthritis, Systemic Lupus Erythematosus, etc.), Sarcoidosis, inherited diseases involving damaged collagen metabolism (Ehlers–Danlos syndrome, Marfan syndrome, and homocystinuria), and more [3].

Still, mechanical excessive load, either acute or repetitive, is the most important cause of tendon rupture. Naturally, the magnitude of the excessive load is lower when exists the factor of age or systemic illness.

The main protective mechanism from the excessive load damaging effect is the proprioception of load buildup on the tendon and pain.

The Golgi tendon organs govern the tendon proprioception. These are mechanosensing structures encapsulating afferent axons in the tendon–muscle interface, therefore interconnecting between force-generating muscle and neural protective feedback [3]. The Golgi tendon organs are thought to interrelate with muscle spindles in governing muscle-generated force, while limb kinesthesia determines the safe extent of limb movement.

3. Clinical manifestations

Therefore, the physiological limb movement is governed by the feedback of mechanosensors in muscle-tendon "force generators." This type of biofeedback of movement causes, for example, the "paradox effect" of higher mean angular acceleration in the abduction of a nondominant arm during intentional limb rise (**Figure 2**) [4], although the force generation potential of the dominant arm is higher (**Figure 3**) [5].

A more exact physiologic requirement can explain this phenomenon when the dominant limb sacrifices the force demand in favor of precision, and this is orchestrated by the proprioceptive feedback from mechanosensors in tendons and muscles.

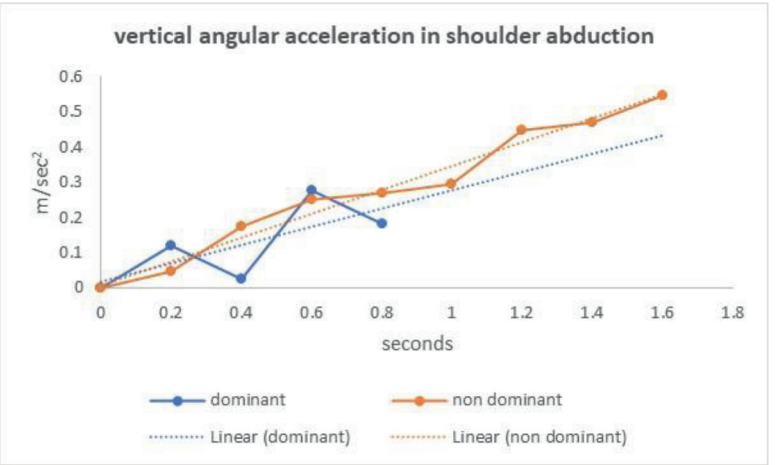

Figure 2.
Example of vertical acceleration during abduction in an adult individual without known shoulder pathology. The linear trendline of movement of the nondominant arm has a higher rate of force buildup in comparison to a dominant limb (linear slope of 0.34 vs. 0.26).

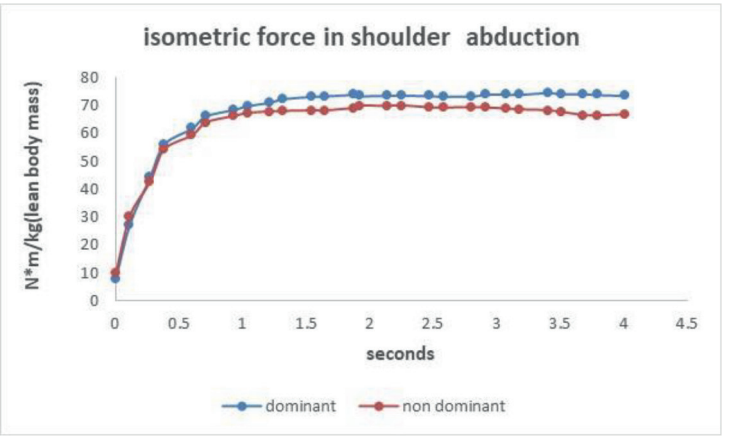

Figure 3.
Example of a force moment buildup during shoulder abduction in an adult individual without known shoulder pathology. The maximal moment in the dominant arm is higher than in a nondominant limb.

Therefore, the tendon's mechanical integrity, determined by its intrinsic properties, may be protected to some extent by the mechanosensing proprioception and might fail when the external load, acute or chronic repetitive, exceeds the latter properties of the tendon.

The pain from the structurally degenerated tendon might have a protective role in further tendon damage, even by lower loads, but this assumption should be further clarified.

Therefore the maximal isometric moment around the joint is significantly diminished, mostly due to pain, when intrinsic tendon damage exists, even without a complete tendon tear (**Figure 4**) [6].

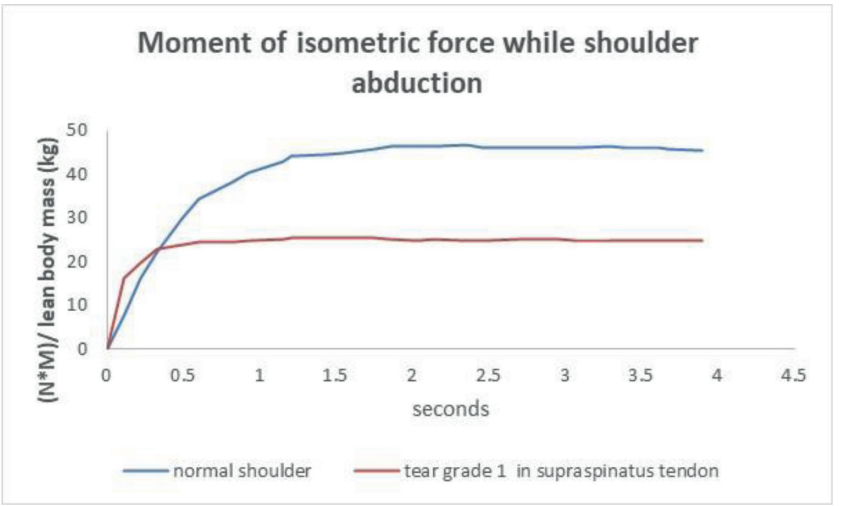

Figure 4.
Example of a force moment buildup during shoulder abduction in adult individuals with and without intrinsic tendon damage. The maximal moment in the normal arm is twice higher than in the case of the rotator cuff with a small degenerative tear.

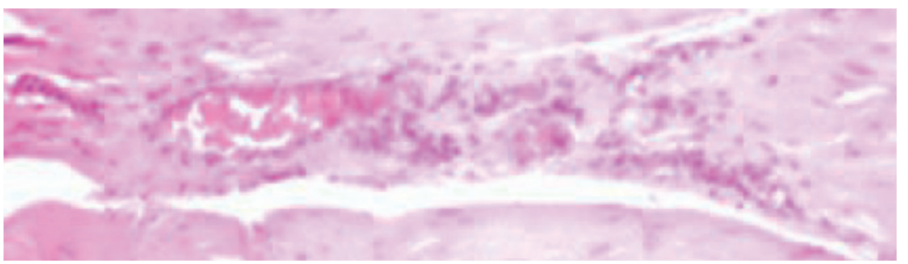

Figure 5.
Degenerative fibrotic tissue in the common extensor origin characteristic of lateral epicondylitis in the elbow. Myxomatous expansion is seen around blood vessels.

A good example of this phenomenon of significant functional impairment is a painful elbow and arm due to lateral epicondylitis, although, In this situation, pain is generated by a relatively small area of tendon degeneration in the common extensor origin at the lateral epicondyle of the distal humerus (**Figure 5**) [7].

Furthermore, the kinematics of the joint, while physiological movement, when a painful tendon is involved, appears to be mechanically inferior to the normal joint, even when the continuity of the degenerative tendon is preserved (**Figure 6**).

Therefore, pain from the shoulder tendinopathy following an acute not physiological load or due to repetitive load, with subsequential microtrauma to the tendon, might be etiologically considered as a protective mechanism from further tendon damage because the pain intensity does not always relate to the extension of the structural impairment in the tendon. Moreover, it is apparent that usually, although the structural damage in the degenerative tendon does not justify mechanical interference with the transfer of force from the contracting muscle to joint movement, still the force generated during joint movement is reduced, and the only causative reason to this impairment is pain originating from the degenerative tendon.

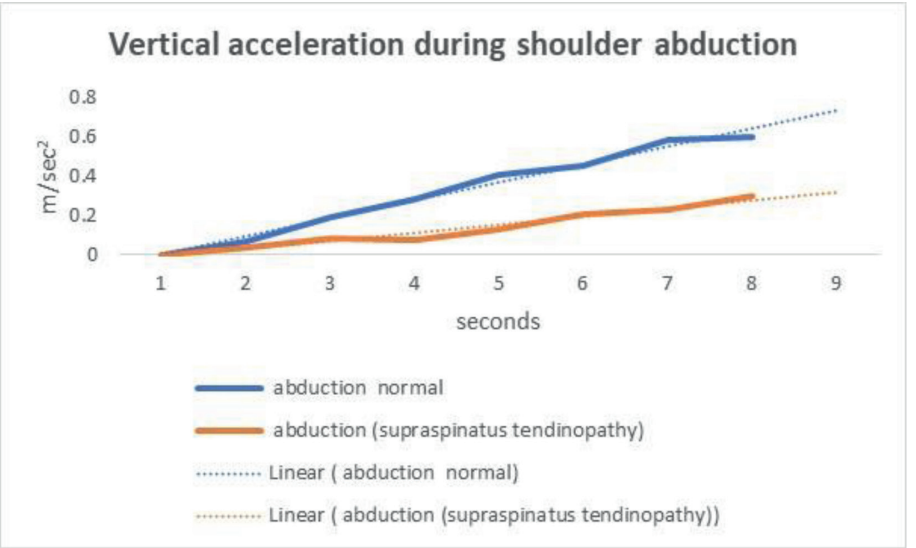

Figure 6.
Example of a vertical force buildup (expressed by acceleration) during shoulder abduction during an unresisted intentional shoulder movement in adult individuals with and without intrinsic tendon damage. The maximal moment in the normal arm is twice higher than in the case of the rotator cuff with a small degenerative tear. The linear trendline of movement in the individual without rotator cuff tendon pathology has a twice higher rate of force buildup compared to the individual with supraspinatus tendinopathy (linear slope of 0.09 vs. 0.04).

This observation should be taken into consideration in the clinical setup when the patients' complaints of pain generated by the tendinopathy are seldom related to the extent of the intrinsic damage of the involved tendon.

4. Prospective

Pain is a subjective complaint, and its quantification is usually obtained by functional self-assessment scores and related to the visual analog scale (VAS). Currently, there is no widespread agreement on the correlation between pain severity and joint impairment due to tendinopathy. Such clinical correlation is imperative for the rational decisions of treatment and the grading of the disability. The main obstacle is the decision on the clinical program related to subjective complaints versus objectively relatively non-extensive tissue damage. Meaning that the standardization of patient disability evaluation in the context of tendinopathy is currently insufficient.

For this type of clinical standardization, the ability to quantify the subjective level of pain is of basic importance [8]. Unfortunately, such an objective method of pain quantization does not exist in clinical use.

Aiming to overcome the difficulty of getting objective measurements of the subjective pain sensation, there is an ongoing research attempt to quantify the pain intensity by the composite algorithms of objective data processing [9]. For this purpose, the main clinical data readily available in the everyday clinical setup may include electrophysiologic and mechanical measurements.

There is experimental evidence that processing such multimodal signals, that is, pulse rate, blood pressure, electroencephalogram, and recording eye movement and facial expression, might provide a basis for a reliable grading of pain level [10].

Thus if the combined data processing methods of pain quantization are available for clinical use in the physician's office setup, it will be possible to evaluate the morbidity caused by tendinopathy, not only on its structural extent but on a more reliable basis of the level of joint impairment due to pain. This study can give an efficient tool and guide for the decision- making, medical and surgical treatment, and disability evaluation related to tendon injury or tendinopathy.

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