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Research Article

Clinical Management of Tendon Injuries in Basketball: Part 1

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Abstract

Tendon-Ligament injuries are the most common injuries in Basketball. From an epidemiological standpoint, these injuries affect the lower extremity (knee, ankle, foot) due to seasonal variation in volume. Clinicians and coaches can see the effects of detrimental tendon injuries via games lost, decreased performance, and movement impairments. Tendons are interesting structures because their primary function is to store and transfer load from muscle to joints and produce movements such as jumping, top-end speed, and change of direction. When tendons become injured such as in the tendinopathic model, type 1 collagen is replaced by type 3 collagen which is weaker due its entropic structure. Furthermore, there are numerous inflammatory factors (depending on stage of injury) present in the extracellular matrix which may lead to central pain sensitization. As this injurious process progresses, performance decrements can debilitate a player's career longevity and organizational success. To date, there have been numerous studies regarding the histology, pathophysiology, and mechanopathology in injured tendons along with treatment strategies. However, the research looking at the integration of injury, treatment, and longevity is very scarce. Thus, the purpose of this 2-part paper is to encompass the various pathological models of tendon pathology through the lens of the basketball player followed by practical management strategies for the clinician and coach.

Introduction

The demands of Basketball have been described primarily as a fast-paced game that is aerobic with multiple anaerobic bouts and quick changes of directions [1]. Within the physiologic assessment, each player is subjected to large volumes of accelerations and jump's while the coaching staff seeks to outmaneuver the other team to win [2]. The professional basketball season can last from 7-10 months and have 72-90 games with the NBA having 82 regular season games [3]. The professional basketball season is also made more complex by factors such as travel, extended hotel stays, and game frequency [3]. These factors can predispose athletes to reduced quality of sleep, reduced opportunity for sound nutrition/hydration strategies, and a higher risk for in-game fatigue [4,5]. Although acute ligamentous injuries are common within basketball Torres, et al. recently found that tendon-ligament injuries are the most common type of injuries in basketball [6]. The primary role of the tendon is to transmit force from muscles to bones [7]. Overuse injuries occur due to repetitive loading and de-loading cycles. These types of injuries are more prevalent when poor movement, inadequate recovery, and/or unfiltered volume is involved [8] Although the common term for these injuries is tendinitis, we now know the more accurate term is tendinosis [9]. Tendinosis is an encompassing term that can include the paratendon and epitendon which seem to show more injury than the tendon proper [10]. This type of injury results in the dominance of Type 3 collagen which is weaker due its entropic structure; an increase in inflammatory mediators; and neural ingrowth which leads to an increase in pain sensitization. Interestingly, while the tendon proper has been previously considered as the pain generating tissue, more recent literature suggests the epitendon and para tendon are primary contributors to pain and dysfunction [11]. The epitendon and paratendon (making the peritendon) are the two structures surrounding the tendon proper. The peritendon functions to unload and facilitate tendinous

movement. Over time, the peritendon becomes more viscous due to increased mucoid substance in its extracellular matrix. This makes tendon gliding more difficult from a histological perspective. Clinically, the athlete may complain of a dull ache within the tendon only at the beginning of activity that improves with warming-up, to a constant ache no matter the level of activity in advanced stages of injury [12]. This correlates with the four stages of tendinopathy proposed by Cook et al.: reactive, disrepair, degenerative, and reactive on degenerative. The first line of defense after tendinopathic diagnosis is usually activity modification, exercise therapy, and manual therapy [12]. Of these defensive strategies, managing player overload should be addressed immediately as it creates the cornerstone for the success of adjunctive manual and exercise therapy. Activity modification may limit the amount of jumping and high-intensity activities on the court and in the weight room. Exercise therapy has been investigated by Alfredson which involves eccentric training. More recently Heavy Slow Training (HST) has gained popularity [13]. Isometric training has also been proven to provide immediate pain modulation and may provide a good tool for extending the ability of function during in-season management. Lastly, manual therapy in the sense of Active Release Technique (ART), Fascial techniques, and dry needling have all shown to have positive effects on pain modulation [14]. The purpose of this paper will be to explore tendinopathy from a holistic model while interpreting it for the sport of basketball.

The Normal Tendon

The normal tendon is primarily composed of Type 1 collagen, the tendon proper, extracellular matrix (ECM), and tendon sheath [15]. Type 1 collagen is produced by tenocytes and is well-organized, allowing it to handle high loads [16]. The ECM is ground substance that makes up the internal milieu of the tendon, which is composed primarily of collagen and elastin [17]. The remainder of the ECM consists of water, various proteins, and proteoglycans (PG's). The tendon receives blood supply from the musculotendinous and osteotendinous junctions, along with vessels in the surrounding connective tissue [18]. The tendon sheath is made of the paritendon which is comprised of the inner paratendon and outer epitendon. Within the tendon, all 4 types of afferent receptors are present: Type 1 Ruffini corpuscle, Type 2 Vater-Paccinian corpuscles, Type 3 Golgi Tendon Receptors (GTO's), and Type 4 free nerve endings [19]. Since the tendon proper is rich in mechanoreceptors and Golgi tendon organs, this means it can store high amounts potential energy and release it as kinetic injury. In the context of Basketball, it is the healthy tendon which allows the athlete to jump explosively and play a part in quick change of direction movements. However, things can go awry when factors such as repeated movements, lack of adequate recovery, and extreme load is placed onto the tendon. These factors will be explored in following sections [20].

Mechanopathology of Tendinopathy

Knowing that tendons are made to store and release energy, a goal of strength and conditioning is to increase the cross-sectional area of the skeletal muscle and enhance the stiffness of the tendon itself [21]. This will result in increased ability to generate more force via physical (myofibrillar, sarcoplasmic reticulum) and neural adaptations (Henneman's size principle, Rate coding) which in basketball correlates positively with performance within the sport of basketball [22]. From a needs analysis view, the ability to produce force quickly in jumping, acceleration, and change of direction determines a large portion of objective success. However, over the course of an 82-game season, the repetitive motions, primarily flexion and extension in the sagittal plane can lead to overuse tendon injuries. These tendon injuries can be likened to lumbar spine disc injuries, where the joint has a threshold [23]. Once this threshold has been violated, the structure begins to break down to the detriment of the athlete [24]. In the tendon, repetitive cycles of plantarflexion/ dorsiflexion or knee flexion/extension without adequate rest or when workload spikes are inconsistent will lead to tendinosis. The tendon homeostasis becomes disrupted leading to collagen disorganization, loss of cytoskeleton tension, and an altered cellular matrix (such as increased matrix metalloproteinases) [25]. Although the argument remains nebulous of whether tendinosis results from overuse or under recovery, it is the repetitive cycles of movement that result in tendon injury and violate its natural healing properties within the extracellular matrix [25].

Histopathology of Tendinopathy

The tendon is a complex structure spanning more than just the tendon proper. The tendon proper is enclosed by a paratenon and epitendon which together form the peritendon [26]. The peritendon serves to protect and lubricate the tendon proper to ensure smooth gliding throughout motion [27]. However, this process can break down leading to dysfunction and pain. For example, research has shown an increase of inflammatory mediators such as IL-1β (Interleukin 1-Beta), TNF-α (Tumor Necrosis Factor-Alpha), and IL-6 (Interleukin six) in the injured extracellular matrix [28]. From a fascial perspective, there is increased viscosity in hyaluronan which can decrease smooth gliding of the tendon proper in its sheath [29]. The peritendon also has a proclivity for neurovascular ingrowth, this can lead to nociception and central sensitization [30]. This is associated with the pain reported by athletes most commonly in the patellar, quadriceps, or Achilles' tendon.

Etiology of Tendinopathy

The work of Stuart McGill has been seminal in understanding how lumbar spine injuries occur[31]. His research reveals the spine has a threshold for which it can handle flexion-extension cycles [31]. Once this threshold has been violated, injury occurs or at least there is a much higher risk of occurring. Similarly, the work by Jill Cook and Keith Barr correlate that tendon of the knee and ankle are most affected and have a physiological limit of flexion-extension cycles [32]. However, when combining frequent competition alongside team practice, individual skill work, and strength and

conditioning sessions, an increases likelihood of tendon injuries can be seen within this population of athletes. The tendon will be exposed to the 3 different vectors of tension, compression, and friction during court play [33]. Thus, it is likely that a combination of these vectors along with increased load and under recovery contribute to the ever-increasing prevalence of tendinopathy among basketball players.

Prevalence of Tendinopathy

The prevalence of tendoninopathy within Basketball has been found to be about 20% with that number increasing to 30% in the Achilles and patellar tendon [34]. However, this number is likely much higher due to multiple factors. For example, there is a weak correlation between pain and pathology, the different stages of tendon injury, and fear of being placed on the injury list [25]. Prevalence describes the total number of individuals in a population who have an injury at a specific period, usually expressed as a percentage[35]. Incidence on the other hand refers to the number of new cases in a population [35] These two epidemiologic terms become useful for the clinician and coach in being able to track tendon injuries within their own organization such as existing vs new condition. Using these basic tracking methods can also help trace the origin of injury such as: increase game frequency or spikes in playing minutes [36].

Conclusion

The purpose of this paper has been to inform the clinician and coach in the field of professional basketball the basic science background of tendon injuries. This paper highlights the physiological and histochemical changes that occur when tendinopathy occurs. It is often an athlete will report to the performance staff with general pain over their tendon. However, it is difficult to quantify what the athlete is going through because there will rarely be swelling, ecchymosis, or an obvious deformity present. The history taking process will be important to identify what stage of tendon injury the athlete is in and how it is affecting their style of play. The clinician is armed with manual palpation and pain provocation tests which can be supplemented with diagnostic ultrasound or MRI to further investigate the tendon in question. The second part of this article will address topics that once tendinopathy is diagnosed, how do the medical and performance staff come together to best manage it throughout during the season using a multi-modal approach.

Practical Applications

Tendon injuries range from acute ruptures to tendinopathy. This paper covers tendinopathy from the perspective of basic science as this is largely overlooked in the current graduate educational model. Tendinopathy is linked to overuse and under recovery, especially in professional basketball. The demands of an 82-game season are significant, compounded with the additional demands of being a professional athlete. Tendinopathy is complicated by the dull pain usually felt by athletes that can't be quantified or "seen" by clinicians. Besides manual palpation and location of perceived pain, we have little in the way of clinical testing for seeing stage of injury or irritability of the tendon itself. From a physiological standpoint, the area between the peritendon and the tendon proper (extracellular matrix) shows increases in inflammatory and destructive mediators depending on stage of injury [27]. As these negative changes within the peritendon progress, there is evidence of neurovascular ingrowth which may be responsible for pain generation within the area [37]. As the tendon proper and peritendon continue to experience cycles of stress, pain begins inhibiting physical activity which can lead to increased time on the injured list. The second part of this paper will strive to provide integrated information for the medical and performance staff on the treatment and management of tendinopathy for the in-season basketball player.

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