Mechanisms of Achilles Tendon Rupture in National Basketball Association Players

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A systematic search was performed of online databases for any Achilles tendon (AT) injuries occurring within the National Basketball Association (NBA). Video was obtained of injuries occurring during competition and downloaded for analysis in Dartfish. NBA athletes (n = 27) were identified with AT rupture over a 30-year period (1991–2021). Of the 27 NBA athletes found to have AT ruptures (mean age: 29.3 [3.3] y; average time in the NBA: 8.5 [3.8] y), 15 in-game videos were obtained for analysis. Noncontact rupture was presumed to have occurred in 12/13 cases. Eight of the 13 athletes had possession of the ball during time of injury. The ankle joint of the injured limb for all 13 athletes was in a dorsiflexed position during the time of injury (47.9° [6.5°]). All 13 athletes performed a false-step mechanism at time of injury where they initiated the movement by taking a rearward step posterior to their center of mass with the injured limb before translating forward. NBA basketball players that suffered AT ruptures appeared to present with a distinct sequence of events, including initiating a false step with ankle dorsiflexion of the injured limb at the time of injury.

Keywords: athletic injuries, biomechanics, video analysis

Basketball places high mechanical loads on participants during competition and training,1–3 increasing the risk for musculoskeletal injuries4–7 particularly to the ankle joint.8,9 Achilles tendon (AT) ruptures occur readily in professional basketball evidenced by an increase in the annual occurrence,10 and a greater prevalence compared with other athletic populations in the United States.11 The post-AT rupture rehabilitation is arduous with return to play being a minimum of 6 months, and oftentimes a much longer process.12 Professional basketball athletes that rupture their AT have a high incidence of not returning to competition and have decreased performance after return to sport.13 Specifically, professional basketball players who sustain AT rupture demonstrated compromised career longevity,14 and despite a return to sport and competition, the on-court performance and playing time of National Basketball Association (NBA) players have been shown to decline significantly after AT rupture.13 It is for these reasons that investigating the specific mechanisms involved with AT ruptures is warranted, which is the second step in Van Mechelen injury prevention model after establishing the burden of injury.15

Previous literature has examined the epidemiology13 of AT ruptures in NBA players. These findings showed that only 44% of NBA athletes who sustained AT ruptures were able to return to competition for longer than 1 season,13 and players who returned did not perform as well as their control-matched peers.13 The mechanism of injury has also been studied using 2D video analysis,14 which can be a valuable tool for in situ evaluation during athletic competitions. For example, previous literature has used this method in soccer,16–18 basketball,19 and alpine ski racing20,21 for epidemiological purposes. Lemme et al14 examined contributing factors involved with AT ruptures in the NBA and noted that a large angle of ankle dorsiflexion occurred at the time of AT rupture (see Figure 1). However, greater biomechanical description of AT injury mechanisms, including joint angles and range of motion, is pertinent for practitioners and may contribute to neuromuscular training programs to build athlete resilience against AT rupture. Therefore, the aim of this present study was to analyze game film and determine exact joint positions and angles associated with AT ruptures in the NBA, and to determine the sequence of movements and contributing mechanisms associated with AT ruptures within an NBA population.

Methods

Data Collection

A systematic search was performed of online databases for AT injuries within the NBA. NBA athletes with AT rupture (n = 27) were identified over a 30-year period (1991–2021). Characteristics of NBA athletes with AT ruptures can be seen in Table 1. Of this cohort of 27 athletes (mean age: 29.3 [3.3] y; average time in the NBA: 8.5 [3.8] y), 15 in-game videos were obtained for analysis. Of the 15 videos, 13 were included in the present study. Two of the videos were excluded due to obstructed views from teammates and opponents.

Video Analysis

Each video was examined by 3 reviewers with biomechanical expertise in high-performance sport and basketball (Petway, Epsley, and Anloague). Each video was downloaded to a personal computer prior to analysis. Upon download, each video was uploaded to MyDartFish 360 Software (DartFish; dartfish.com 5.0.1.0), and still, shots were obtained to the hundredth of a second to identify the index frame (ie, the frame before the injury event). Index frames were used to determine the lower limb joint angles. Measurements were taken at the moment of end-range ankle dorsiflexion of the trail leg (ie, the injured limb) during
injury (Figure 2). For each situation, distinct time points were analyzed for the sequence of movements leading up to the AT rupture. Video reviewers were asked to judge whether or not the athlete had possession of the ball at the time of injury and whether there was contact with an opponent before or during injury. Player action was categorized by the range of motion of the ankle, knee, and hip of the injured leg, the hip and knee of the noninjured limb, and the hip–trunk angle. Subsequently, 3 joint angles were obtained for analysis, including ankle dorsiflexion angle of the injured leg, knee flexion angle of the noninjured leg, and trunk angle relative to the hip. The 3 joint measurements were taken because involvement of trunk flexion, injured limb ankle dorsiflexion, and noninjured limb knee flexion were present in all 13 instances at the time of injury. These outcome measures were inputted into a spreadsheet (Microsoft Excel 2016) for examination. All data are presented as the mean angle (SD) unless noted otherwise.

Results

Results of the video analysis are presented in Table 2. Only a single video (1/13) was classified as a contact injury, and 12/13 were classified as noncontact mechanisms. During the film review, it was observed that 62% (8/13) of AT ruptures occurred during possession of the ball. All athletes (100%) had a dorsiflexed ankle position on the injured limb at the time of injury, and 69% (9/13) had an outwardly turned foot position on the injured limb.

The average dorsiflexion angle of the injured ankle was 47.9° (6.5°), the average trunk flexion angle relative to the hip axis was 54.2° (6.8°), and the average knee flexion angle of the lead leg (noninjured limb) was 67.4° (17.4°). The kinematics were averaged, and a representative example of the injury mechanism is shown in Figure 2.

Also of note was a consistent sequence of movements leading to the injury occurrence. In all videos analyzed, the athlete took a backward step with the injured limb well outside of the base of support without any displacement of the body center of mass (BCM) (ie, posterior and outside of the BCM). This motion was followed by rapid ankle dorsiflexion of the injured limb and lowering of the trunk. The sequencing of these movements can be seen in Figure 2.

Discussion

The most important finding of this study is that AT rupture was frequently associated with an extreme and rapid increase in ankle dorsiflexion of the injured limb during tactical maneuvers while athletes had possession of the ball. Findings from the present study support existing epidemiological data on AT rupture in professional basketball players and address step 2 in Van Meekelen injury prevention model. Also of note were the maneuvers prior to the AT injuries. Specifically, the sequencing of movements leading to this position was proceeded by a false-step mechanism of the injured limb where the athlete’s trail leg (ie, injured limb) would strike posterior to the BCM without the athlete translating forward.

A potential interpretation of these findings is that the stretch load placed on the tendon during this false-step action exceeded the

Figure 1 — Proposed mechanism of Achilles tendon rupture in professional basketball players.

Figure 2 — Average angle of ankle dorsiflexion (injured limb), average angle of knee flexion (noninjured limb), and average trunk flexion angle during Achilles tendon rupture.
structural tolerance capacity of the tendon in terminal ranges. Peak strain in the AT has been demonstrated in vivo to be between 6.2 and 10.3%, and ex vivo testing has shown failure within similar strain thresholds, while in vitro modeling shows failure to be estimated at 45%\textsuperscript{22} of peak strain. Another study modeled peak strains during sprinting in the free portion of the tendon to be as high as 16.2%.\textsuperscript{23} Such discrepancies of in vivo strains compared with ex vivo failure points are likely due to the measurement of the whole tendon unit compared with an isolated portion of the tendon.\textsuperscript{22,23} Consequently in vivo modeling would suggest that failure strain may not be approached during intense exercise at physiologic ranges, but this has not been examined in the extreme end ranges of motion. Ankle dorsiflexion range of motion has also been shown to increase immediately postgame in basketball players,\textsuperscript{24} suggesting that the operating range of the AT during basketball match play may encroach upon the 47.9 degrees that was observed as the average ankle dorsiflexion at rupture. The potential for large range of motion demands at the ankle in basketball is important because theoretically, if all series elastic elements were pretensioned, the free portion of the AT, which has lower strain capacity, may have demonstrated increased susceptibility for rupture. However, this notion is purely speculative.

Table 1 Characteristics of NBA Athletes With AT Ruptures

<table>
<thead>
<tr>
<th>Mean (SD)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>29.3 (3.3)</td>
</tr>
<tr>
<td>Years in NBA</td>
<td>8.5 (3.8)</td>
</tr>
<tr>
<td>Game # of injury</td>
<td>40 (21.7)</td>
</tr>
<tr>
<td>Minutes per game (career)</td>
<td>28 (8)</td>
</tr>
<tr>
<td>Minutes per game (season)</td>
<td>27 (9)</td>
</tr>
<tr>
<td>Minutes per game (last 10)</td>
<td>30 (8)</td>
</tr>
</tbody>
</table>

Abbreviations: AT, Achilles tendon; NBA, National Basketball Association. Note: Game # of Injury is the number of games played during the season of AT injury.

Table 2 Descriptive of Film Review of AT Rupture in the NBA

<table>
<thead>
<tr>
<th>Injured with ball</th>
<th>Sagittal plane (trail leg)</th>
<th>Shank angle (trail leg)</th>
<th>Trunk angle</th>
<th>Knee angle (lead leg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Athlete 1</td>
<td>No</td>
<td>HF, KF, and DF</td>
<td>56.1</td>
<td>Obstructed</td>
</tr>
<tr>
<td>Athlete 2</td>
<td>Yes</td>
<td>HE, KE, and DF</td>
<td>45</td>
<td>73</td>
</tr>
<tr>
<td>Athlete 3</td>
<td>Yes</td>
<td>HE, KE, and DF</td>
<td>49.3</td>
<td>57</td>
</tr>
<tr>
<td>Athlete 4</td>
<td>No</td>
<td>HE, KF, and DF</td>
<td>41.2</td>
<td>54.2</td>
</tr>
<tr>
<td>Athlete 5</td>
<td>Yes</td>
<td>HE, KE, and DF</td>
<td>49.3</td>
<td>50.5</td>
</tr>
<tr>
<td>Athlete 6</td>
<td>No</td>
<td>HE, KE, and DF</td>
<td>60.9</td>
<td>57.6</td>
</tr>
<tr>
<td>Athlete 7</td>
<td>Yes</td>
<td>HF, KF, and DF</td>
<td>40.8</td>
<td>54.9</td>
</tr>
<tr>
<td>Athlete 8</td>
<td>No</td>
<td>HF, KF, and DF</td>
<td>42.3</td>
<td>43.7</td>
</tr>
<tr>
<td>Athlete 9</td>
<td>Yes</td>
<td>HE, KE, and DF</td>
<td>45.5</td>
<td>55.3</td>
</tr>
<tr>
<td>Athlete 10</td>
<td>Yes</td>
<td>Obstructed</td>
<td>53.4</td>
<td>Obstructed</td>
</tr>
<tr>
<td>Athlete 11</td>
<td>Yes</td>
<td>Obstructed</td>
<td>40.7</td>
<td>Obstructed</td>
</tr>
<tr>
<td>Athlete 12</td>
<td>Yes</td>
<td>HF, KF, and DF</td>
<td>50.4</td>
<td>67.6</td>
</tr>
<tr>
<td>Athlete 13</td>
<td>Yes</td>
<td>Obstructed</td>
<td>Obstructed</td>
<td>Obstructed</td>
</tr>
<tr>
<td>Athlete 14</td>
<td>Yes</td>
<td>Obstructed</td>
<td>Obstructed</td>
<td>Obstructed</td>
</tr>
<tr>
<td>Athlete 15</td>
<td>No</td>
<td>Contact</td>
<td>Contact</td>
<td>Contact</td>
</tr>
</tbody>
</table>

Abbreviations: AT, Achilles tendon; DF, dorsiflexion; HE, hip extension; HF, hip flexion; KE, knee extension; KF, knee flexion; NBA, National Basketball Association. Note: Injured with ball, had possession of ball during AT rupture.

Furthermore, the AT has been noted to have a twisted structure, with composite fibers from each component of the triceps surae rotating counterclockwise from proximal to distal on the right, and clockwise on the left.\textsuperscript{25} MacMahon et al\textsuperscript{26} also found that the AT was more externally rotated in ruptured than in nonruptured tendons when retrospectively examining patients with ruptured and healthy AT. Findings showed that the lateral edge of the AT was positioned closer to the fibula and more lateral to the calcaneus in those who suffered AT rupture. When considered together, there
may be more overall torsion of the AT in those who sustain rupture as the tendon fibers rotate about the tendon axis, and the tendon moves laterally as a whole. Whether there is any association between torsion of the AT and our finding of an outwardly turned foot as part of the rupture mechanism is beyond the scope of this paper but may warrant further investigation.

The present findings suggest the importance of generating increased AT stiffness during the false-step movement. This movement strategy ensures that terminal ranges are not encroached upon concurrently with high mechanical load, and energy storage of the tendon can be optimized leading to potentially decreasing the risk of rupture and improving movement efficiency, respectively. Theoretically, this stiffened spring system would also allow improved force transfer from muscle to tendon, an important determinant of rapid force capacity.27 Furthermore, the technical aspects within the sporting activity should be addressed, such as training effective movement strategies for the false-step maneuver in basketball. Skill development coaches should have contingencies to limit the potential for injury in this biomechanical position. When assessing active dorsiflexion from a biomechanical standpoint, practitioners should be cautioned if an athlete has extreme laxity upon ground contact of a false-step maneuver and repeatedly demonstrates this pattern when the foot is posterior to the center of mass. Excessive passive mobilization or the overemphasis on dorsiflexion range of motion should also be questioned.25 Improving stiffness mechanisms within a controlled environment is supported in previous literature.28,29 For example, Werkhausen et al30 found that after a 10-week resistance training program consisting of single-leg isometric plantarflexion AT stiffness and plantarflexion strength improved by 18% and 15%, respectively. Plyometric training with short ground contact times has also been shown to significantly increase extensibility of tendon structures during ballistic contractions and active muscle stiffness during fast stretching.31,32

The AT is an elastic energy storage tendon designed to improve high-speed locomotion and movement efficiency.33 In this sense, it is analogous to the superficial digital flexor tendon in racehorses, which is also prone to rupture.25 Research in the equine tendon suggests that movement between tendon fascicles contributes significantly to the elastic component and promotes cyclic fatigue resistance in energy storage tendons over positional tendons.33,34 Interfascicular gliding has been shown to decrease with increasing age, alongside an accumulation of partially degraded collagen.34,35 The possibility that age-related AT changes may have contributed to the occurrence of AT rupture in this cohort of basketball players in addition to the biomechanics should be emphasized.10,13

Cumulative AT load may be another potential confounder to AT ruptures observed here as previous research has shown evidence that tissues from tendinopathic and ruptured AT have chronic (nonresolving) inflammation.36 Microdamage that has not been adequately repaired by local tenocytes is thought to accumulate and predispose to tendon rupture. In race horses the adult superficial digital flexor tendon demonstrated decreased levels of type 1 collagen with age and increased levels of weaker collagen. Gap junction expression which enables communication between tenocytes and is important in the reparative response, is also decreased.35 Notably, catabolic remodeling has been estimated to occur at >9% strain in horses, although this value is more uncertain in humans.23 As previously discussed, in vivo strain during sprinting and hopping is estimated to be well above this threshold. When these factors are considered together, the AT appears to become less adaptable to high loads with increasing biological age such as those occurring in the present cohort of basketball players who were relatively old with respect to elite athletes (approaching their 30). To this end, it has been posited that the time course change of tendinopathy is a progressive continuum whereby repetitive loading and tissue maladaptation eventually lead to a tendon in disrepair, a state when tendon rupture may occur readily.37–39 Thus, the proper distribution of mechanical load and proper biomechanical positioning during technical development seem crucial in decreasing potential risk of AT rupture.

There were limitations of the present study. First, only 13 AT ruptures from in-game situations in the NBA could be obtained, which is a small sample size relative to the total number of ACL tears within the same population.40–42 Nevertheless, the results were strengthened by the high degree of commonality between the injury events and low variance in the observed joint angles. However, previous research supports our findings that extreme dorsiflexion and positive shank angles are contributing factors for AT ruptures.28,29 Second, the 2-dimensional video analysis technique was obstructed at certain time points preventing a complete analysis of the change in hip and knee angles during the injury event. While this method has its limitations, it is an accepted practice for in situ competition analysis.16–19

The AT ruptures can be deleterious to professional basketball athletes. Extreme ankle dorsiflexion was identified with outward turning of the foot following a “false step” as the predominant mechanism of AT injury in NBA players. Medical, performance, and technical skill practitioners should consider a model to help athletes avoid high-risk positions during offensive maneuvers, as this may be important in the prevention of AT rupture, particularly in the older NBA athlete.

**References**


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