

Lower Limb Dominance, Morphology, and Sonographic Abnormalities of the Patellar Tendon in Elite Basketball Players: A Cross-Sectional Study

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Context: Patellar tendinopathy is common in basketball players, and structural ultrasound abnormalities can be found in symptomatic and asymptomatic tendons. Lower limb dominance may also be a critical load factor, potentially leading to overloading of the patellar tendon.

Objective: To describe and compare the prevalence by lower limb dominance of patellar tendons with structural and vascular abnormalities and to describe the morphologic measures of tendons without abnormalities among adult male elite basketball players.

Design: Cross-sectional study.

Setting: Medical center of a professional basketball team in the Spanish league.

Patients or Other Participants: A total of 73 adult male elite basketball players (146 patellar tendons; age = 26.8 ± 4.9 years, height = 198.0 ± 0.1 cm, mass = 95.4 ± 11.4 kg).

Main Outcome Measure(s): We used ultrasound to screen the patellar tendons for the presence of structural and vascular abnormalities. Tendons were categorized as *abnormal* if they demonstrated a focal area of hypoechogenicity, thickening, or neovascularization. We also examined the cross-sectional area and thickness of tendons without abnormalities. Preva-

lence and morphologic measures were compared by limb dominance.

Results: A total of 35 players (48%) had bilateral abnormalities, whereas 21 (28.7%) had unilateral abnormalities. Among the 91 abnormal tendons, 90 (61.6% of 146 tendons) exhibited a focal area of hypoechogenicity, 59 (40.4% of 146 tendons) exhibited thickening, and 14 (9.6% of 146 tendons) exhibited neovascularization. No group differences were detected between the dominant and nondominant limbs. Among the 55 normal patellar tendons, 34 were bilateral (from 17 players) and 21 were unilateral. Approximately 25% ($n = 14$) of all 55 normal tendons had a cross-sectional area that was greater than 182.8 mm^2 and a thickness greater than 7.2 mm. Among the 34 bilateral normal tendons, no group differences were observed between the dominant and nondominant limbs for either cross-sectional area or thickness.

Conclusions: The prevalence of abnormal tendons was high among adult male elite basketball players, and bilateral presentations were more frequent. Structural abnormalities were most common.

Key Words: pain, lower extremity, tendon injury, ultrasonography

Key Points

- Adult male elite basketball players had a high prevalence of abnormal tendons, with bilateral presentation more frequent than unilateral presentation.
- Structural intratendon changes occurred more commonly than vascular abnormalities.
- The distribution of the structural and vascular abnormalities was similar in the dominant and nondominant limbs.
- The cross-sectional area and thickness of patellar tendons without abnormalities were similar in the dominant and nondominant limbs.

Patellar tendinopathy (PT) is a common injury among basketball players.¹ This condition is defined as a clinical presentation of pain that often limits athletes' ability to jump or run, reducing their levels of training or competition.^{2,3} Researchers^{4–8} have shown that PT is correlated with various levels of sonographic abnormalities. However, the "normal" sonographic appearance of these tendons is still a matter of debate,⁹ as abnormalities have been reported¹⁰ to be common even among asymptomatic individuals. In contrast, consistent

evidence^{10,11} has shown that the presence of some abnormal intratendinous changes among basketball players can lead to knee symptoms and tendon-related disabilities. Given the high prevalence of jumper's knee and its negative consequences, the amount of research on the prevalence and determinants of intratendon abnormalities is growing.^{9,12}

Investigators have reported that, among young (age range, 14–18 years) elite male basketball players attending a training camp, 46% had structural abnormalities of the

Table 1. Participants' Demographic, Anthropometric, and Professional Characteristics

| Factor | Total Sample (N = 73) | Tendon Subgroup | | |
|---|--------------------------|---------------------------|------------------------------------|-----------------------------------|
| | | Normal Tendon (n = 17) | Unilateral Abnormality (n = 21) | Bilateral Abnormality (n = 35) |
| Intrinsic, mean \pm SD | | | | |
| Age, y | 26.8 \pm 4.9 | 24.2 \pm 4.7 | 26.6 \pm 5.0 | 28.2 \pm 4.5 ^a |
| Height, cm | 198.0 \pm 0.1 | 197.0 \pm 0.1 | 197.0 \pm 0.1 | 199.0 \pm 0.1 |
| Mass, kg | 95.4 \pm 11.4 | 93.6 \pm 12.0 | 91.9 \pm 9.1 | 98.2 \pm 12.0 |
| Body mass index, kg/m ² | 24.3 \pm 1.6 | 23.9 \pm 1.6 | 23.6 \pm 1.4 | 24.8 \pm 1.6 |
| Professional | | | | |
| Time practicing as a professional basketball player, mean \pm SD, y | 8.8 \pm 4.8 | 7.2 \pm 4.9 | 8.2 \pm 4.5 | 9.9 \pm 4.9 |
| Player position, No. of players | | | | |
| Point guard | 17.0 | 4.0 | 4.0 | 9.0 |
| Shooting guard | 20.0 | 8.0 | 8.0 | 4.0 |
| Small forward | 11.0 | 2.0 | 2.0 | 7.0 |
| Power forward | 10.0 | 1.0 | 5.0 | 4.0 |
| Center | 15.0 | 2.0 | 2.0 | 11.0 |

^a Different from the normal tendon subgroup ($P = .005$).

tendon (ie, focal hypoechoic area),¹⁰ and the prevalences of unilateral and bilateral abnormal intratendinous changes were similar.¹³ Nevertheless, to our knowledge, data on intratendinous changes in adult elite professionals playing in a higher division are not available. Moreover, we do not know whether quantitative measures of tendon morphology (eg, cross-sectional area [CSA] or thickness) among adult elite basketball players are similar to those of other population groups (eg, amateur athletic or sedentary individuals). Whereas researchers^{14,15} have reported larger CSA and greater thickness in amateur basketball players and other athletes than in sedentary participants, these measures in basketball players are still unclear.

Habitual loading related to sport (years playing and training types) and intrinsic factors, such as age and body mass index, have been identified as factors that can influence the morphologic properties of tendons.^{15,16} In jumping sports, lower limb asymmetries during the typical stop-jump movement have been regarded as an additional and critical load factor, potentially leading to the overloading of the dominant lower limb¹⁷ and contributing to the development of PT.¹⁸ Despite this assumption, the prevalence of abnormalities and quantitative measures of patellar tendons in elite male jumping athletes have not been evaluated according to lower limb dominance.^{5,10} Only recently has this framework been used,^{14,15,19} and the results have been contradictory. Whereas some authors^{15,19} found no differences between the dominant and nondominant sides for structural abnormalities or quantitative measures, others¹⁴ reported the opposite. In basketball, the nondominant limb is more commonly used as the takeoff limb for a layup and the landing limb; therefore, it becomes the dominant leg, and we refer to it as such in this study. Based on our clinical experience and environment, we think that, in clinical environments, the dominant limb may be more affected by microtrauma than the opposite limb. Therefore, the primary purposes of our study were to describe and compare the prevalence by lower limb dominance of patellar tendons with structural and vascular abnormalities among adult male elite basketball players and to determine whether unilateral and bilateral tendon

abnormalities developed because of habitual loading related to sport (years playing and training types) and intrinsic factors, such as age or body mass index. Our secondary purpose was to describe and compare morphologic measures (CSA and thickness) of the tendons without abnormalities by lower limb dominance.

METHODS

Study Design and Participants

We performed this observational study on male professional basketball players in the first division of the Spanish league from November to May during the 2015–2016 basketball season. All 16 teams in the Liga Asociación de Clubes de Baloncesto were invited to participate. Ultimately, 8 teams (73 players, 146 tendons; age = 26.8 \pm 4.8 years, time playing as a professional in a high-level division = 8.8 \pm 4.8 years) participated. The characteristics of participants are displayed in Table 1. Players were prospectively recruited by their physical therapists with their coaches' knowledge and consent. We included athletes who were practicing their sport at the time of assessment, were professional players, and were age 18 years or more. Players were excluded if they had a rheumatic or metabolic illness at the time of enrollment, had a history of knee surgery, had received acupuncture to the tendon in the 3 months before the study, had taken analgesic or anti-inflammatory drugs in the 24 hours before the study, or were unable to communicate in either Spanish or English. All participants provided written informed consent, and the study was approved by the University of Valencia Ethical Committee for Human Research (number H1456389710571).

Measures

All data were collected by 2 experienced sport physical therapists (J.C.B., P.M.) using a standardized procedure during a session at the end of the week between 12 and 24 hours before the regular match. One professional (J.C.B.) collected demographic data (age), anthropometric data

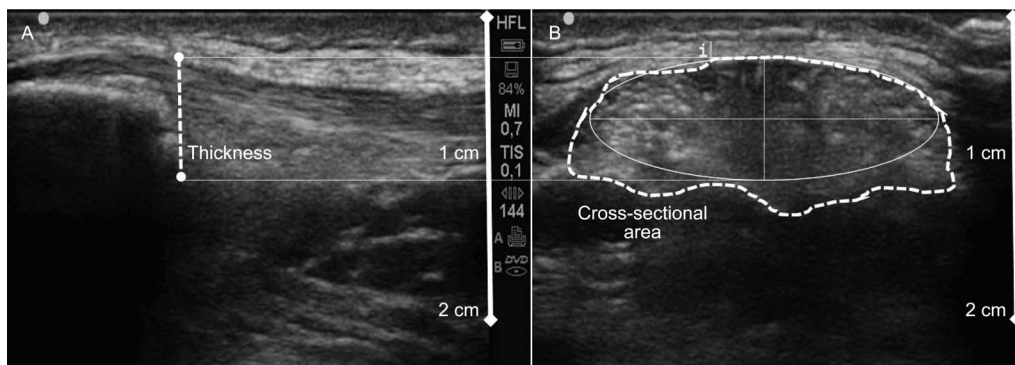


Figure 1. Morphologic measures in an ultrasonographic image of a tendon without abnormalities. **A**, Longitudinal view to measure thickness. **B**, Cross-sectional view and cross-sectional area delimited by semiautomatic adaptive contours.

(height, mass), and loading related to sport characteristics (dominant limb, years playing as a professional in a high division, playing position, and training type). The second professional (P.M.), who was blinded to other measures, carried out the ultrasound protocol and tendon assessment in a separate room.

Ultrasound Protocol. The goals of the ultrasound protocol were twofold: (1) to examine the presence or absence of neovascularization (NV) and to screen for 2 sonographic structural abnormalities (focal area of hypoechogenicity [FAH] and thickening) and (2) to establish the CSA and thickness of tendons without abnormalities (Figure 1). The patellar tendons of both knees were assessed with gray scale and Doppler color using a Sonosite M-Turbo (Sonosite, Inc, Bothell, WA) portable ultrasound device equipped with a 10- to 5-MHz transducer head. The same settings were standardized for all participants, and the gain was adapted to show the tendon structure clearly.

The examination protocol for the patellar tendon was based on the technical guidelines for ultrasound examination defined by the European Society of Musculoskeletal Radiology.²⁰ Players lay supine with the knee flexed to approximately 30°, which was achieved by placing a small pillow beneath the popliteal space. The tendons imaged in both the longitudinal and transverse scans were recorded at the widest thickness in the sagittal plane where abnormalities were observed. The axial image was taken approximately 5 mm distal to the inferior pole of the patella. After the knee was extended and the quadriceps muscles were relaxed to prevent physical constriction of the blood vessels, the longitudinal view was performed over the proximal patellar tendon. The power Doppler settings were standardized with an automatic gain, a sensitivity of 4 to -4 cm/s, and a pulse repetition frequency of 608 Hz, which was determined by the sonographer's experience and the manufacturer's recommendations. Power Doppler sonography was used to study blood flow at the patellar tendon. Given that only high flows can be registered, the technique did not allow us to register normal circulation in the tendon because of the relatively low flow rate.

Qualitative Analysis of Tendon. The images were analyzed by 2 experienced musculoskeletal sonographers (F.V., P.M.) independently and then by consensus in the event of disagreement. The presence of FAH was identified when zones with focal changes in echogenicity were observed. We determined thickening to be present when a thickened tendon at one site altered the tendon symmetry in

a transverse section. Neovascularity was established when we observed a vessel in the sagittal plane of the tendon that was greater than 1 mm long, as assessed by Doppler sonography.^{21,22} The initial κ coefficient ranged from 0.88 to 0.99 for the 3 sonographic abnormalities: FAH ($\kappa = 0.97$; 95% confidence interval [CI] = 0.93, 1.00), thickening ($\kappa = 0.88$; 95% CI = 0.80, 0.96), and NV ($\kappa = 0.99$; 95% CI = 0.98, 1.00). A tendon was considered *abnormal* if it demonstrated FAH, thickening, or NV (Figure 2).

Tendons without abnormalities were measured by another experienced musculoskeletal sonographer (J.R.).²³ We used ImageJ software (version 1.51g; National Institutes of Health, Bethesda, MD) to perform the quantitative analysis. We selected the contour of the tendon in cross-sectional images using semiautomatic segmentation based on the adaptive active contours (snakes)²⁴ plugin for ImageJ that reduces operator variability and is less time consuming.²⁵ The operator carried out a fine adjustment of the region of interest, and all images were calibrated in millimeters to determine the CSA and thickness and analyzed 3 times. If the coefficient of these 3 measures exceeded 5%, the measurements for that image were repeated.

High reliability of the image-analysis process has been reported using intraclass correlation coefficients (ICCs) for CSA (ICC = 1.0; 95% CI = 0.9, 1.0) and thickness (ICC = 0.99; 95% CI = 0.98, 1.0). Small differences in pairs of measures were present for CSA (0.11 mm²; 95% limits of agreement = -0.01 mm², 0.3 mm²) and thickness (0.04 mm; 95% limits of agreement = 0.02 mm, 0.11 mm).²³

Statistical Analysis

The frequency and number of sonographic abnormalities (FAH, thickening, NV) per tendon were calculated. Descriptive statistics were calculated for quantitative measures of tendons without abnormalities (CSA, thickness), including the mean, standard deviation, range, and 25th and 75th percentiles. Players were categorized as having *normal tendons*, *unilateral abnormality*, or *bilateral abnormality*, depending on the number of abnormal patellar tendons. A 1-way between-groups analysis of variance was conducted to assess differences among these 3 participant groups for the intrinsic and professional factors. We used post hoc Bonferroni and Tukey tests to analyze differences between pairs of groups.

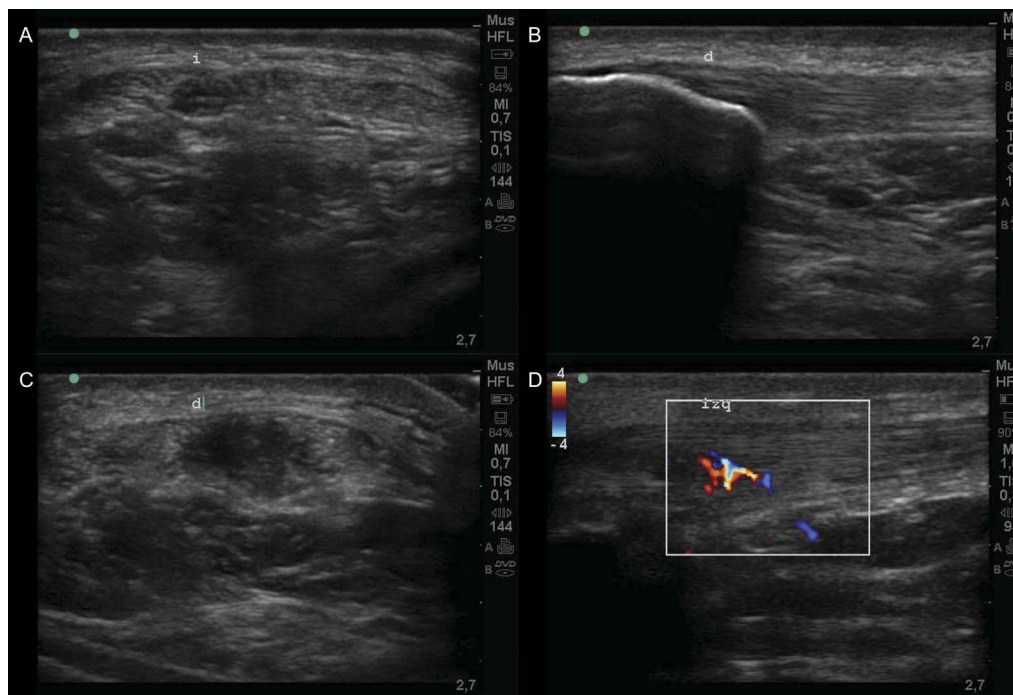


Figure 2. Sonographic images of tendon assessments. **A**, Transverse scan shows tendon with focal area of hypoechogenicity (FAH). **B**, Longitudinal scan shows tendon with FAH and thickening. **C**, Transverse scan shows tendon with FAH and thickening. **D**, Longitudinal view shows tendon with FAH, thickening, and neovascularization.

The χ^2 analysis was used to examine the frequency of each abnormality (FAH, thickening, NV) in players with 1 or more abnormalities in a given tendon collapsed between the dominant and nondominant sides. Similarly, we used a *t* test to compare the means of CSA and thickness by lower limb dominance. The α level was set at .05. We used SPSS (version 21; IBM Corp, Armonk, NY) for all analyses.

RESULTS

Of the 146 tendons, 55 had no detectable tendon changes, and 91 had abnormalities. A total of 21 (28.8%) of the 73 players had unilateral tendon abnormalities, 35 (48.0%) had bilateral abnormalities, and only 17 (23.3%) had no abnormalities. The 1-way analysis of variance revealed a difference in age among the 3 groups ($F_{2,70} = 4.16$, $P = .02$). Post hoc comparisons indicated that only players with bilateral abnormalities were older than players without abnormal tendons ($P = .005$). No group differences were

detected among the 3 player groups for the other intrinsic and professional factors (Table 1).

Among the 91 abnormal tendons, 90 (61.6% of 146 tendons) exhibited FAH, 59 (40.4% of 146 tendons) exhibited thickening, and 14 (9.6% of 146 tendons) exhibited NV (Table 2). Thirty-two tendons had 1 of 3 sonographic abnormalities, 46 had 2, and 13 had all 3. No group differences were observed between the dominant and nondominant limbs for the type and number of sonographic abnormalities.

Of the 55 patellar tendons without abnormalities, 21 were unilateral and 34 were bilateral. A sonographic quantitative analysis of all unilateral and bilateral patellar tendons without abnormalities is provided in Table 3. Approximately 25% ($n = 14$) of the 55 tendons without abnormalities showed a CSA greater than 182.8 mm² and a thickness greater than 7.2 mm. The mean values of the CSA and thickness were similar between the unilateral and bilateral tendons without abnormalities (167.8 versus 164.6

Table 2. Structural and Vascular Tendon Abnormalities

| Tendon Abnormality(ies) | All Abnormalities (N = 91), No. (%) | Abnormalities by Limb, No. (%) | |
|--------------------------------|--|--------------------------------|----------------------|
| | | Dominant (n = 49) | Nondominant (n = 42) |
| Sonographic | | | |
| Focal area of hypoechogenicity | 90 (98.9) | 48 (98.0) | 42 (100) |
| Thickening | 59 (64.8) | 32 (65.3) | 27 (64.3) |
| Neovascularization | 14 (15.4) | 8 (16.3) | 6 (14.3) |
| Some abnormality | 91 (100) ^a | 49 (53.8) | 42 (46.2) |
| Number | | | |
| 1 | 32 (35.2) | 17 (34.7) | 15 (35.7) |
| 2 | 46 (50.5) | 25 (51.0) | 21 (50.0) |
| 3 | 13 (14.3) | 7 (14.3) | 6 (14.3) |

^a Only 1 tendon demonstrated neovascularization and no structural abnormality.

Table 3. Quantitative Sonographic Analysis of Tendons Without Abnormalities

| Measure | Total Tendons (N = 55) | Unilateral Tendons (n = 21) | Bilateral Tendons | | |
|---------------------------------------|---------------------------|--------------------------------|------------------------|--|---|
| | | | Both Limbs (n = 34) | Dominant Limb (n = 17) ^a | Nondominant Limb (n = 17) ^a |
| Cross-sectional area, mm ² | | | | | |
| Mean ± SD | 165.9 ± 26.6 | 167.8 ± 27.4 | 164.6 ± 26.5 | 168.7 ± 31.2 | 160.6 ± 20.8 |
| Minimum–maximum | 112.6–223.4 | 112.6–223.4 | 113.9–214.2 | | |
| 25th percentile | 147.5 | 112.6 | 142.8 | | |
| 75th percentile | 182.8 | 187.0 | 182.5 | | |
| Thickness, mm | | | | | |
| Mean ± SD | 6.6 ± 1.0 | 6.7 ± 3.3 | 6.5 ± 0.9 | 6.6 ± 1.1 | 6.2 ± 0.7 |
| Minimum–maximum | 4.5–8.8 | 4.5–8.8 | 4.5–8.8 | | |
| 25th percentile | 5.8 | 6.2 | 5.7 | | |
| 75th percentile | 7.2 | 7.4 | 7.1 | | |

^a The minimum–maximum and the 25th and 75th percentiles were not calculated for the dominant and nondominant limbs.

mm² and 6.7 versus 6.5 mm, respectively). Among the 34 bilateral tendons without abnormalities, we observed no group differences between the dominant and nondominant limbs for CSA or thickness.

DISCUSSION

Our study of active adult male elite basketball players demonstrated that only a small number of players had no abnormalities affecting either tendon and that structural intratendinous changes, especially FAH, were common. The prevalence of structural and vascular tendon abnormalities and the quantitative measures (CSA, thickness) in tendons without abnormalities were similar between the dominant and nondominant limbs.

Among the abnormal tendons, we found that sonographic structural abnormalities (FAH and thickening) were much more frequent than vascular changes (NV). The different prevalences of structural and vascular abnormalities has also been reported⁵ in the patellar tendons of adult volleyball athletes who played in elite and national competitions. In our study, the prevalence of NV was congruent with findings in adult volleyball players⁵ and female elite dancers.⁹ In contrast, the structural intratendinous changes in our study were more frequent than those in junior populations²⁶ and young elite basketball players.⁷ This increase may be explained by the greater impacts sustained at the competition level and by the older age or more years of training of players. The effect of older age or more years of training is supported by research data^{11,15} on basketball and volleyball players that showed a higher frequency of structural changes among the older age groups. Our findings suggested that the competition level should be regarded as a possible factor in future studies.

Whereas Warden et al²⁷ described the prevalence of PT symptoms of athletes by lower limb dominance, we are the first, to our knowledge, to compare the prevalence of abnormal patellar tendon changes by limb dominance in relation to PT. The distributions of the structural and vascular abnormalities were similar in the dominant and nondominant limbs. These results are consistent with another study²⁸ of adult jumping athletes. However, they contradicted the findings of Toprak et al,¹⁴ who noted more abnormalities in the dominant limbs of junior female elite volleyball players. Discordance between this study and ours may be explained by differences in participants' demo-

graphic (ie, age, sex) characteristics and training, technical skills, or years of training.

Cook et al²⁶ and Gaida et al¹³ found an equal number of patellar tendons with bilateral and unilateral abnormalities in young elite basketball players. However, we demonstrated that bilateral abnormalities were much more prevalent than unilateral abnormalities among adult elite basketball players. Age differences between participants in the earlier studies and our study may explain our higher percentage of tendons with bilateral sonographic abnormalities. This possibility is consistent with the observation that the oldest basketball players in our study had bilateral abnormalities. The relationship between age and the number of lower limbs with sonographic abnormalities may indicate changes in the mechanical properties of the tendon²⁹ or its material properties associated with a decreased water content.³⁰ Older adult male basketball players also may have experienced greater stress associated with repeated jumping and landing over years of training.¹⁵

The CSA and thickness values of the patellar tendons without abnormalities were similar in the dominant and nondominant limbs. Thus, lower limb dominance during the stop-jump movement may not provide enough differential stimulus to elicit variations in the tendon's morphologic properties. It is likely that the cumulative stress associated with repeated jumping during years of training and matches induced similar amounts of stimulus to both limbs. Nevertheless, the CSA and thickness values of the normal patellar tendon in our cohort were not negligible, and some researchers^{15,26} have reported lower measures in adult amateur basketball players and sedentary participants. Specifically, our mean values of CSA and thickness were approximately 1.3 and 1.6 times higher, respectively, than those of amateur players and sedentary individuals.

A valuable aspect of our study was the selection of adult elite basketball players from 1 or more leagues according to the European National Basketball League rankings.³¹ However, most of the studies we reviewed were focused on young athletes or university-level competitions, meaning that the participants generally had fewer years of training and, therefore, less continuous loading of their patellar tendons. Another strength of our study was that we used lower limb dominance to describe both the prevalence of sonographic abnormalities and morphologic measures of

normal tendons. Furthermore, the strength of our results, using both limbs, reinforced our findings. In future studies with larger samples, investigators could compare the thickening of a tendon without abnormalities in adult elite professionals playing in high-level divisions with tendons of those in other populations, such as individuals with sedentary lifestyles, young players, or adult recreational players. Such information would be useful to potentially characterize the size of the patellar tendon in different populations and to gain insight into the structural adaptations of the tendon in high-level players.

Although tendon abnormalities are a matter of debate because they have also been reported in asymptomatic individuals,³² our findings have clinical implications for developing a prevention or treatment or rehabilitation program to be implemented in the early stages of basketball practice. Given that age and years of training are intrinsic factors that seem to lead to tendon abnormalities, screening and preventive measures should be focused on older players and those with more years playing. Researchers should consider professional factors, such as playing position, focusing on centers, in whom bilateral PT is more common. Clinically, ultrasonography should be used to screen the patellar tendon during the preseason to detect abnormalities and should be included in the eccentric strengthening program for PT and training-load control. Similarly, years of training and player position should be addressed as possible risk factors for other PT abnormalities.

Our study had several limitations. First, given that all participants were men, these results should be generalized to women with caution. Second, only 8 basketball teams participated in the study. Nevertheless, we believe that the participants' characteristics were comparable with those of nonparticipants because all played in the same league. Third, inactive players with PT symptoms may have further abnormal sonographic tendon findings; therefore, their exclusion from our study may have biased our results concerning prevalence. We excluded these players because our research question was based on active players. Fourth, the results and concerns raised in the "Discussion" suggested that age may influence abnormal tendons. This notion is speculative because of the cross-sectional study design; therefore, our data need to be confirmed in prospective studies. Fifth, our values for CSA and thickness were limited to normal tendons. We did this because it was difficult to delimit the measurement points of abnormal tendons as a result of the tissue degeneration, so bias may have been introduced.

CONCLUSIONS

In adult male elite basketball players, structural intra-tendon changes were more common than vascular abnormalities; however, all changes were distributed equally in the lower limbs. The CSA and thickness of tendons without abnormalities were similar between the dominant and nondominant limbs. The prevalence of abnormal tendons was high among male elite basketball players, and bilateral changes were more frequent than unilateral changes on ultrasound imaging. Strategies to prevent PT should involve both limbs and start in the early stages of basketball practice. Longitudinal studies need to be conducted from an early stage of the injury to investigate when, over time,

lower limb dominance is a greater determinant of the morphologic properties of the tendon and sonographic abnormalities.

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