EARLY ANTERIOR KNEE PAIN IN MALE ADOLESCENT BASKETBALL PLAYERS ARE RELATED WITH BODY HEIGHT AND ABNORMAL KNEE MORPHOLOGY

Mantas Mickevičius¹, Hans Degens¹,², Rolandas Kesminas¹, Saulius Rutkauskas³, Danguolė Satkunskienė¹, Kazys Vadopalaš¹, Kazimieras Pukėnas¹, Jaak Jürimäe⁴, Albertas Skurvydas¹, Sigitas Kamandulis¹

¹ Institute of Sport Science and Innovations, Lithuanian Sports University, Kaunas LT 44221, Lithuania
² School of Healthcare Science, Manchester Metropolitan University, Manchester M15 6BH, United Kingdom
³ Department of Laboratory Medicine, Medical Academy, Lithuanian University of Health Sciences, Kaunas LT 44307, Lithuania
⁴ Institute of Sport Pedagogy and Coaching Sciences, University of Tartu, Tartu 50090, Estonia

Corresponding author: Mantas Mickevičius, Institute of Sport Science and Innovations, Lithuanian Sports University, Sporto 6, LT-44221 Kaunas, Lithuania. Phone: +370 600 73021, email: mantas.mickevicius@lsu.lt
ABSTRACT

Objectives: To compare knee torque, range of motion, quality of movement, and morphology in dominant and nondominant legs of male adolescent basketball players with and without anterior knee pain and untrained peers.

Design: Cross-sectional.

Setting: Sports performance laboratory.

Participants: Male basketball players aged 14–15 years with and without anterior knee pain and healthy untrained subjects (n = 88).

Main outcome measures: Basketball players were allocated to a symptomatic or asymptomatic group based on self-reported anterior knee pain. Associations between pain and body mass, height, passive range of motion, muscle peak torque, coactivation, neuromuscular control, proprioception, and ultrasound observations were investigated.

Results: The prevalence of pain did not differ significantly between sides. Of 176 knees inspected, 44 were painful, and 26 of these exhibited abnormalities in ultrasonography. Symptomatic players were 5.0 and 6.9 cm taller than asymptomatic players and controls, respectively (P < 0.05). In athletes with knee pain, the odds ratios of morphological abnormalities and greater height were increased by 8.6 and 5.0 times (P < 0.001).

Conclusion: Knee pain prevalence in adolescent basketball players was not related to differences between sides but was higher in tall players. Knee pain was accompanied by morphological abnormalities detected with ultrasound.

Keywords Diagnostic ultrasound, Proprioception/Balance, Electromyography, Isokinetic dynamometry
INTRODUCTION

Regular physical activity improves skeletal muscle function and cardiovascular health, and helps to prevent and reverse obesity, reduce anxiety and depression, and improve self-confidence and cognitive function (Haskell et al., 2007). Because of these benefits, regular sports participation is highly encouraged. However, with growing competiveness in sport, children, adolescents, and young adult participants are under increasing psychological pressure to win. For this reason, physical training is often intense, frequent, and highly specialized, which increases the risk of training becoming too intense and/or unbalanced, as well as possible health problems and traumatic injuries (Frisch, Croisier, Urhausen, Seil, & Theisen, 2009). Although healthy tendons and muscles can tolerate large loads, constant overexertion and incorrectly performed exercises and loading while fatigued can change the material properties of the joints, such as the knee joint, which can make them more prone to overuse injuries and associated pain (Helland et al., 2013). Additional risk factors for knee joint pain include muscle weakness, strength imbalance, motor control deficit, knee joint laxity, and changes in the mechanical and morphological properties of tendons and ligaments (Smith et al., 2012; Ladenhauf, Graziano, & Marx, 2013; Gagnier, Morgenstern, & Chess, 2013). These factors can increase the stress on the lower limbs during sports events.

Adolescents are particularly vulnerable to overuse injuries because their musculotendinous system is undergoing significant structural, biomechanical, and, consequently, functional alterations during this phase of rapid growth (Frisch et al., 2009; Adirim & Cheng, 2003). Previous studies have shown that up to 54% of adolescent athletes
experience some form of knee pain annually (Foss, Myer, Magnussen, & Hewett, 2014). Nontraumatic anterior knee pain is one of the most common complaints in young athletes (Adirim & Cheng, 2003).

A high degree of sport specialization contributes to overuse injuries in young athletes (Bell, Post, Trigsted, Hetzel, McGuine, & Brooks, 2016). The incidence of knee joint injuries in children and adolescents is particularly high in organized team sports, including basketball (Caine, Maffulli, & Caine, 2008; Frisch et al., 2009). This can be attributed to repeated sprinting, stopping, changing direction, and jumping (Cumps, Verhagen, & Meeusen, 2007). These movements place a particularly high amount of stress on the leading knee and associated muscle–tendon unit. Adolescent basketball players therefore provide an ideal population for evaluating the structural and functional changes associated with knee pain and injury.

The purpose of the present study was to compare knee torque, range of motion (ROM), quality of movement, and morphological indicators in the dominant and nondominant legs of male adolescent basketball players with and without anterior knee pain and untrained male adolescents. All basketball players who experienced knee pain started having minor complaints that were not yet intense enough to warrant medical attention. This phase is thus ideal for assessing the associations between pain and functional and morphological adaptations. Understanding these relationships may help to prevent further development of pain or even alleviate existing pain. We hypothesized that adolescent basketball players would have 1) side-to-side imbalances in muscle strength, 2) impaired flexibility and neuromuscular control and 3) show morphological abnormalities that can be detected
noninvasively with ultrasound. We expect that this would help identify the risk factors for, and early indicators of, knee pain and injury.

METHODS

Participants

The present study involved 88 male adolescents, who were divided into the following three groups (Table 1): adolescent basketball players who had participated for ≥5 years in basketball activities and who had experienced anterior knee joint pain during the previous month (symptomatic group, n = 29) or had not experienced pain (asymptomatic group, n = 30). In both groups, players were excluded if they had experienced any acute or contact trauma, including ligament or meniscus injury and/or associated pain in a proximal and/or distal joint. Players who had prior or present pain that required medical attention were also excluded from the study. The basketball players were recruited at the end of the playing season (May–June) from a county basketball school. They played seven to eight times per month for about 7 months per year in the national championships and attended 1.5-h training sessions four to five times per week all year. The third group comprised healthy untrained, non-basketball-playing adolescents without a history of knee pain (control group, n = 29). Control subjects were recruited from a local high school and were matched for age and body mass index (BMI) to the basketball players. The Kaunas Regional Biomedical Research Ethics Committee approved the research protocol, which was consistent with the principles outlined in the Declaration of Helsinki. Written informed consent was obtained from the parent or guardian of each participant.
Pain

For all participants in the control, asymptomatic, and symptomatic groups, the same investigator characterized the severity and frequency of knee pain for each knee according to the Knee Injury and Osteoarthritis Outcome Score (KOOS; Lithuanian version LK1.0) questionnaire using only the nine-item part related to pain. Each question is scored from 0 (no pain) to 4 (extreme pain) (Roos & Lohmander, 2003). Here, we report the average score of the nine items in the test.

Testing procedures

Body mass was measured in kilograms (TBF-300 body composition scale; Tanita, UK Ltd., West Drayton, UK) and height in meters (Anthropometry Martin, GPM SiberHegner, Switzerland) in the laboratory. The ROM of the left and right knee joints was determined with a goniometer. This was followed by a warm-up of 8 min of cycling at a power of 50–70 W at a cadence of 70 rpm on a cycle ergometer. Next, knee extensor and flexor muscle proprioception, peak torque, and coactivation were measured in each leg on an isokinetic dynamometer (System 3; Biodex Medical Systems, Shirley, NY, USA). The next day, clinical observation of the left and right knee joints was performed, and patella tendon dimensions were measured by ultrasound. This was followed by a neuromuscular control test for visual inspection of movement stability. The leg used to kick a ball was considered the dominant leg. The above methods are detailed in the following sections.
Figure 1. Graphical overview of the testing protocol.

Note. Slump – Slump Test; SLR – Straight Leg Rise Test; KE – Knee Extension Test (90/90); PNB – Prone Knee Bend Test; IEHR – Internal and External Hip Rotation; NC – Neuromuscular control; SLS – Single Leg Squat Test; SLH – Single Leg Hoop Test; FRS – Foam Roller Supine Test; SLR – Straight Leg Rise Test; MVC – maximal voluntary contraction force; Con – concentric muscle contraction; Ecc – eccentric muscle contraction; US – ultrasonography; CSAPT – cross-sectional area of the patellar tendon; TPT – thickness of the patellar tendon.

ROM

Passive ROM was determined in both legs with a standard goniometer and demarcated by the positions at which resistance to movement was felt (Sauer, Potter, Weisshaar, Ploeg, & Thelen, 2007). Participants did not report neurogenic pain in prone knee-bend, straight-leg raise, or seated slump tests. We first assessed resistance to movement in these neurodynamic tests (Boyd, Wanek, Gray, & Topp, 2009). To minimize subjective bias, all ROMs were determined by the same researcher. High intraclass correlation coefficients (ICCs) of 0.88–0.97 have been reported for the ROM tests used in the present study (Cibulka, Sinacore, Cromer, & Delitto, 1998; Gabbe, Bennell, Wajswelner, & Finch, 2004; Russell & Bandy,
The 90/90 test was performed by the participant in a supine position with the hip and knee flexed to 90° and gently stretched until resistance was felt. The lateral epicondyle of the femur was palpated, and the goniometer placed over the epicondyle. The lateral malleolus of the tibia and the greater trochanter of the femur were marked. The arms of the goniometer were aligned with these proximal and distal landmarks (Russell & Bandy, 2004).

Next, the prone knee-bend test for femoral nerve tension was performed on each leg. The examiner passively flexed one knee slowly as far as possible until unilateral neurogenic pain was felt along the lumbar area, buttock, and/or anterior thigh. Knee ROM was recorded as the angle at which subjective symptoms were reported (Dutton, 2004).

For the internal and external hip rotation test, the participant lay in the supine position with the hip and knee flexed 90°. The hip was then maximally rotated internally and externally (Byrd, 2005).

The straight-leg raise test was performed in the supine position. The tested limb was raised by the examiner with the knee fully extended and the foot in a relaxed position. The contralateral limb was kept fully extended and in neutral rotation by a second examiner. The goniometer was placed over the greater trochanter, with one arm aligned with the lateral femoral condyle and the other aligned parallel to the table in the direction of the midaxillary line (Neto et al., 2014).

For the seated slump test, the participant sat in a slouched position and actively flexed the cervical spine as far as comfortably possible. The ankle was then passively dorsiflexed to slight resistance, and the knee was slowly passively extended. The knee was extended until
full extension was achieved or until the participant reported the onset of neural-mediated symptoms. The goniometer was aligned in a standard fashion with the axis over the lateral femoral epicondyle, the stationary arm was aligned with the femoral trochanter, and the moveable arm was aligned with the lateral malleolus (Davis, Anderson, Carson, Elkins, & Stuckey, 2008).

**Proprioception**

A familiarization session for measurement of proprioception and peak torque was performed 2 days before data collection. The participant sat in the isokinetic dynamometer chair and performed a shorter version of the testing protocol described below. Proprioception was assessed using an isokinetic dynamometer during both knee flexion and extension at a knee angle of 90° (0° = leg fully extended). During the testing, repeated isometric knee flexion and extension (10 trials at 1-s intervals) were performed in an attempt to match the required 20% maximal voluntary isometric contraction (MVC). The participant could see the force generated on the instrument’s screen. To provide a measure of accuracy of isometric contractions, we calculated the absolute error size (Magill, 2007) using the following formula:

$$\text{Absolute error} = \frac{\sum |x_i - T|}{n}$$

where $x_i$ is the torque generated during trial $i$, $T$ is the target torque, and $n$ is the number of trials. The vertical brackets $|$ $|$ indicate that the difference was always given a positive value. This gives an absolute error value in N·m. Not every individual produces the same maximal
torque, so to compare the data between different subjects, we also calculated the absolute error (AE) size as a percentage of MVC as follows:

\[ AE(\%) = \frac{AE}{20\%\ MVC} \times 100 \]

Moderate ICCs of 0.73–0.81 for the proprioception test have been reported (Zavieh et al., 2016).

**Peak torque**

After completion of the proprioception evaluation, concentric and eccentric isokinetic and isometric peak torque of the knee extensor and flexor muscles were measured using an isokinetic dynamometer. First, an MVC was performed twice, each separated by a 2-min rest interval with the knee joint at 90° (full extension = 0°). The participant then performed three maximal contractions at angular velocities of 60°/s and 240°/s in both the concentric and eccentric modes, and the best of three trials was used for further analysis. Each of the angular velocities and the concentric and eccentric measurements were separated by ≥2-min rest to prevent the development of fatigue. Two warm-up trials at submaximal intensity were performed before measurement of maximal peak torque at each velocity. The ICC for peak torque varies from 0.81 to 0.95 depending on exercise mode and velocity (Janusevicius et al., 2017). The ratios of peak torque for concentric hamstring:concentric quadriceps and eccentric hamstring:concentric quadriceps were calculated.

**Electromyography**

Electromyographic activity was assessed for the rectus femoris (RF) and the long head of the biceps femoris (BF) muscles as described previously (Hermens, Freriks, Desselhorst-Klug, & Rau, 2000). An MP150 instrument (Biopac Systems, Inc., CA, USA) was used to record the electromyogram (EMG). Two self-adhesive disposable Ag–AgCl electrodes (10-mm diameter, Ceracorta, Forli (FC), Italy) were placed on the hamstring and quadriceps muscles with a 20-mm interelectrode distance, and the ground electrode was positioned on the knee. A raw EMG was acquired with a sampling frequency of 1000 Hz and was filtered using analogue high-pass (10 Hz) and low-pass (500 Hz) filters. The EMG signals were analyzed using Acknowledge software (Biopac Systems). Coactivation of the RF and BF was assessed using the root mean square (RMS) of the EMG signal. The analyzed EMG signal was taken at 200 ms around the peak for isometric and 60°/s for concentric contractions, at 100 ms for 240°/s concentric and 60°/s eccentric contractions, and at 20 ms for eccentric contractions at 240°/s. Coactivation of the BF was assessed as the EMG activity during knee extension as a percentage of the EMG activity of the BF during maximal knee flexion either at a constant length or during a constant-velocity contraction (Kellis & Baltzopoulos, 1996). The ICCs for the RMS of the EMG signal are 0.80–0.88 (Larsson, Karlsson, Eriksson, & Gerdle, 2003).

Ultrasonographic assessment

A radiologist with nearly 10 years clinical work experience assessed both knees in all participants in the control, asymptomatic, and symptomatic groups using an ultrasound device (Esaote MyLab 50 XVision, Italy) with a 7–12-MHz linear probe. The radiologist
was blinded to the group. The participant was diagnosed as having morphological abnormalities if ultrasound inspection exposed pathological signs such as oedema, increased connective tissue deposition, or fragmentation of the tibial tuberosity ossification centre, which is linked to Osgood–Schlatter syndrome. Other characteristic signs were recorded as morphological abnormalities; these included semimembranosus bursitis, observed as an enlarged semimembranosus bursa, bipartite patella, as a patella with an unfused accessory ossification centre, or thickening of the patella, which indicates patellar tendonitis. If the participant had noticed any of these abnormalities, he was advised to consult an orthopaedic surgeon, but we did not request the examination results.

The morphology of the knee joint is represented here as the patellar tendon (PT) cross-sectional area (CSA) and thickness. The CSA and thickness in both the longitudinal and transverse planes were measured in both knees while the participant was in a supine position with his knees supported and flexed (~30°). The PT was assessed in two areas: 1 cm from the apex of the patella, and 1 cm above the tibia attachment. The data from these two PT measurement areas was averaged for further analysis. High ICCs of 0.82–0.93 for PT dimension measurements have been reported (Gellhorn & Carlson, 2013).

Neuromuscular control

The following day, neuromuscular control was assessed by a physical therapist with >10 years’ experience. High to moderate ICCs of 0.77–0.92 for the tests used in the present study have been reported (Mens, Vleeming, Snijders, Koes, & Stam, 2001; Stensrud, Myklebust, Kristianslund, Bahr, & Krosshaug, 2011).
During the single-leg squat, the participant squatted five times on each leg and then performed five single-leg hops on each leg (Bailey, Selfe, & Richards, 2011). In these two tests, position and movement were assessed and graded as follows: 1, the ankle, knee and hip were maintained in line, the pelvis was horizontal, and the trunk was vertical in the sagittal plane; 2, the participant displayed minor disturbances in the lower extremity and trunk alignment in the sagittal plane and/or a slight pelvic drop; 3, the participant displayed moderate disturbances in the lower extremity and trunk alignment in the sagittal plane and/or moderate pelvic drop; and 4, the participant displayed severe disturbances in the lower extremity and trunk alignment in the sagittal plane and/or significant pelvic drop.

For the foam roller supine test, the participant lifted each leg five times from the supine position lying on a foam roller with the hands not touching the floor. Finally, for the active straight-leg raise test, the participant was asked to lift each leg 20 cm straight five times while lying supine on the floor (Mens et al., 2001). The last two tests were assessed as follows: 1, stable pelvis; 2, minor sway of pelvis; 3, moderate sway of pelvis; 4, the participant falls off the roller or strong rotation or sway of the pelvis from the horizontal position. Raising one leg with a little sway and the other leg with a larger sway was considered a minor disturbance, and moderate pelvic sway during the active straight-leg raise test was considered a moderate disturbance.

**Statistical analysis**

Data are presented as mean and standard deviation (SD). The Kolmogorov–Smirnov test was used to check whether the data were normally distributed. If not normally distributed,
the data were log-transformed or the inverse was taken to ensure a normal distribution, which
was then used for statistical analysis. In some cases, no normal distribution developed even
after transformation of the data. In these cases, a nonparametric exact Wilcoxon test was used
to evaluate the differences, such as for the neuromuscular control tests. For normally
distributed data, a repeated-measures ANOVA was used with a within-factor (side: dominant
vs. nondominant) and a between-factor (group: symptomatic vs. nonsymptomatic vs. control)
analysis. When a significant group effect but no side × group interaction was found, Tukey’s
post hoc test was performed to locate the differences. A significant interaction between side
and group indicated that the side difference differed between the three groups.

To identify any associations between knee pain and parametric variables (peak torque,
ROM, AE, RMS), these variables were divided into two percentiles using the median value
to calculate the odds ratios for a 2 × 2 contingency table and 95% confidence intervals (CIs).
The participants were allocated to a low (3 or 4 points) or high (1 or 2 points) group for each
neuromuscular control test or for the number of morphological abnormalities in positive and
negative groups, and the odds ratios and CIs were calculated for nonparametric variables.
The sample size required to detect a change of 15% in peak torque, which is considered to
be clinically relevant, was 25 at a one-tailed $\alpha = 0.05$ and statistical power $\beta = 0.80$. For all
statistical tests, differences were regarded as significant at $P < 0.05$.  

RESULTS

Participant characteristics
Thirteen players had bilateral pain, while 16 players had unilateral (8 in the dominant, 8 in the non-dominant leg) pain. The pain intensity and frequency in the symptomatic athletes were $1.5 \pm 0.7$ points and $10.7 \pm 5.7$ days per month. The pain intensity and frequency did not differ between legs ($1.4$ points and $10.9$ days in the dominant leg; $1.5$ points and $10.4$ days in the nondominant leg, $P > 0.05$). The symptomatic players were taller than the asymptomatic players and controls ($P < 0.05$), but body mass and BMI did not differ significantly between groups (Table 1). The odds ratio of having knee pain was increased $5.0$ times in tall players compared with asymptomatic players and controls ($95\%$ CI $2.2$–$11.2$; $P < 0.001$).

### Table 1. Participants’ characteristics.

<table>
<thead>
<tr>
<th>Group</th>
<th>Number</th>
<th>Age (years)</th>
<th>Height (cm)</th>
<th>Body mass (kg)</th>
<th>BMI</th>
<th>Training experience (years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptomatic</td>
<td>29</td>
<td>$14.5 \pm 0.6$</td>
<td>$179^{a,b} \pm 8$</td>
<td>$65.4 \pm 10.6$</td>
<td>$20.3 \pm 2.1$</td>
<td>$6.3 \pm 1.6$</td>
</tr>
<tr>
<td>Asymptomatic</td>
<td>30</td>
<td>$14.0 \pm 0.6$</td>
<td>$174 \pm 11$</td>
<td>$61.4 \pm 13.2$</td>
<td>$20.0 \pm 2.5$</td>
<td>$6.3 \pm 1.4$</td>
</tr>
<tr>
<td>Control</td>
<td>29</td>
<td>$14.2 \pm 0.7$</td>
<td>$172 \pm 9$</td>
<td>$63.4 \pm 15.1$</td>
<td>$21.2$</td>
<td>$–$</td>
</tr>
</tbody>
</table>

$^a$: significantly different from control at $P < 0.05$.

$^b$: significantly different from asymptomatic at $P < 0.05$. Data are expressed as mean ± standard deviation.

ROM

There were no significant side differences, side × group interactions, or significant associations with pain for ROM in any joint. Figure 2 shows that symptomatic players had a
larger ROM than controls in knee extension during sitting (slump test, P = 0.020), and a smaller ROM in hip external rotation (P < 0.001) and knee bend in the prone position (P < 0.001). These variables did not differ significantly between the symptomatic and asymptomatic groups.

Figure 2. Comparison of ROM in controls and symptomatic and asymptomatic basketball players.
*: significantly different from control at P < 0.05. The data are presented as mean ± standard deviation.

Peak torque

There were no significant group × side interactions or associations between torque and knee pain. The isometric torque, concentric torque at 60°/s, and eccentric torque at 60°/s were higher in the dominant than in the nondominant leg (Table 2; P < 0.05). Both symptomatic and asymptomatic players developed higher torques than controls during concentric 60°/s extension and flexion and 60°/s eccentric flexion (Table 2; P < 0.05). In addition, symptomatic players developed higher torque than controls for isometric flexion, concentric
240°/s extension and flexion, and eccentric 60°/s and 240°/s flexion and extension (Table 2; 
P < 0.05). The symptomatic players developed higher torques than asymptomatic players for 
isometric extension and 60°/s eccentric flexion (Table 2; P = 0.044).
Table 2. Comparison of peak torque, ratio, and coactivation (CoAc) for the dominant and nondominant leg of controls (C), and symptomatic (S), and asymptomatic (A) basketball players.

<table>
<thead>
<tr>
<th></th>
<th>Symptomatic</th>
<th></th>
<th></th>
<th></th>
<th>Side effect</th>
<th>Group effect</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Dominant</td>
<td>Nondominant</td>
<td>Dominant</td>
<td>Nondominant</td>
<td>Control</td>
<td></td>
</tr>
<tr>
<td><strong>Isometric</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MVC extension</td>
<td>175±29</td>
<td>162±34</td>
<td>159±48</td>
<td>144±38</td>
<td>160±42</td>
<td>149±35</td>
</tr>
<tr>
<td>CoAc</td>
<td>(11.7)</td>
<td>(15.7)</td>
<td>(15.4)</td>
<td>(14.9)</td>
<td>(17.5)</td>
<td>(18.0)</td>
</tr>
<tr>
<td>MVC flexion</td>
<td>88±21</td>
<td>78±18</td>
<td>79±26</td>
<td>71±23</td>
<td>71±17</td>
<td>64±21</td>
</tr>
<tr>
<td>CoAc</td>
<td>(8.0)</td>
<td>(7.3)</td>
<td>(8.1)</td>
<td>(7.8)</td>
<td>(7.9)</td>
<td>(7.7)</td>
</tr>
<tr>
<td><strong>Concentric</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>60°/s extension</td>
<td>154±29</td>
<td>147±35</td>
<td>145±42</td>
<td>135±33</td>
<td>130±34</td>
<td>128±34</td>
</tr>
<tr>
<td>CoAc</td>
<td>(14.0)</td>
<td>(14.9)</td>
<td>(17.0)</td>
<td>(16.6)</td>
<td>(16.9)</td>
<td>(18.3)</td>
</tr>
<tr>
<td>60°/s flexion</td>
<td>108±26</td>
<td>102±23</td>
<td>103±29</td>
<td>93±25</td>
<td>91±26</td>
<td>83±19</td>
</tr>
<tr>
<td>CoAc</td>
<td>(7.9)</td>
<td>(8.2)</td>
<td>(6.6)</td>
<td>(6.2)</td>
<td>(9.9)</td>
<td>(8.9)</td>
</tr>
<tr>
<td>240°/s extension</td>
<td>96±16</td>
<td>92±20</td>
<td>87±23</td>
<td>86±21</td>
<td>84±23</td>
<td>83±21</td>
</tr>
<tr>
<td>CoAc</td>
<td>(18.3)</td>
<td>(17.0)</td>
<td>(17.4)</td>
<td>(19.3)</td>
<td>(16.2)</td>
<td>(18.6)</td>
</tr>
<tr>
<td>240°/s flexion</td>
<td>79±19</td>
<td>76±15</td>
<td>75±21</td>
<td>71±18</td>
<td>71±22</td>
<td>67±17</td>
</tr>
<tr>
<td>CoAc</td>
<td>(10.0)</td>
<td>(10.3)</td>
<td>(9.0)</td>
<td>(8.0)</td>
<td>(11.4)</td>
<td>(9.7)</td>
</tr>
<tr>
<td><strong>Eccentric</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>60°/s extension</td>
<td>199±39</td>
<td>198±49</td>
<td>190±60</td>
<td>179±50</td>
<td>173±43</td>
<td>172±43</td>
</tr>
<tr>
<td>CoAc</td>
<td>(14.7)</td>
<td>(18.0)</td>
<td>(17.8)</td>
<td>(22.0)</td>
<td>(18.9)</td>
<td>(22.3)</td>
</tr>
<tr>
<td>60°/s flexion</td>
<td>136±34</td>
<td>129±35</td>
<td>124±42</td>
<td>109±32</td>
<td>115±34</td>
<td>105±29</td>
</tr>
<tr>
<td>CoAc</td>
<td>(8.0)</td>
<td>(9.5)</td>
<td>(10.1)</td>
<td>(7.5)</td>
<td>(10.3)</td>
<td>(9.2)</td>
</tr>
<tr>
<td>240°/s extension</td>
<td>143±44</td>
<td>159±46</td>
<td>132±59</td>
<td>126±54</td>
<td>133±57</td>
<td>122±56</td>
</tr>
<tr>
<td>CoAc</td>
<td>(17.1)</td>
<td>(15.6)</td>
<td>(21.2)</td>
<td>(17.3)</td>
<td>(16.5)</td>
<td>(21.0)</td>
</tr>
<tr>
<td>240°/s flexion</td>
<td>130±37</td>
<td>122±42</td>
<td>118±40</td>
<td>106±37</td>
<td>112±26</td>
<td>104±29</td>
</tr>
<tr>
<td>CoAc</td>
<td>(13.3)</td>
<td>(12.6)</td>
<td>(17.8)</td>
<td>(16.5)</td>
<td>(15.4)</td>
<td>(14.6)</td>
</tr>
</tbody>
</table>

Note: peak torque is presented in N m, coactivation and force ratios are presented as percentages. MVC: maximal isometric voluntary contraction torque;

NS: not significant. Values are expressed as mean ± standard deviation.
Coactivation

In all groups, coactivation was lower during knee flexion than during knee extension (P < 0.05). Coactivation was lower in the dominant than in the nondominant leg only during eccentric 60°/s flexion (P < 0.05; Table 2). Coactivation was lower in symptomatic players than in controls during isometric and eccentric 60°/s extensions and was lower in asymptomatic players than in controls during concentric 60°/s and 240°/s flexion and eccentric 60°/s\(^1\) extension (Table 2; P < 0.05). Coactivation did not differ significantly between the symptomatic and asymptomatic players and was not significantly associated with knee pain.

Proprioception

There were no significant differences in matching the actual extension torque to a required torque between groups and no significant associations between the ability to match torque and knee pain (Fig. 3). Matching was better in the dominant than in the nondominant leg only in extension (P < 0.001). Matching was better during flexion in symptomatic players than in controls (P < 0.05, Fig. 3).

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Figure 3. Comparison of the error magnitude during repeated contractions at 20% maximal voluntary isometric contraction (MVC) between groups for the dominant and nondominant legs in controls, symptomatic, and asymptomatic basketball players. A, leg extension; B, leg flexion; *: different from controls at P < 0.05; #: difference between dominant and nondominant legs at P < 0.05. The data are presented as mean ± standard deviation.

Neuromuscular control

There were no significant side differences or side × group interactions in the neuromuscular control tests. Controls scored higher than the basketball players for the single-leg squat and jump and active straight-leg raise tests (P < 0.05, Fig. 4). When the data were combined for athletes and nonathletes, the poorer scores in the active straight-leg raise test were strongly associated with having a knee disorder, as shown by a more than twofold elevation in the odds ratio (95% CI 1.1–4.2; P < 0.05).
Figure 4. Comparison of visual inspection of stability during movement in controls, symptomatic (S), and asymptomatic (A) basketball players. *: significantly different from controls at $P < 0.05$. The data are presented as mean ± standard deviation.

Knee joint morphology

There were no significant side differences or side × group interactions for patella tendon morphology. Of the 176 knees inspected with ultrasound, 26 showed morphological abnormalities. Out of these 26, 22 were painful, but 4 were without pain (2 from non-symptomatic basketball players and 2 from non-athletes). There were also 20 painful knees without morphological abnormalities. The most frequent changes in the 26 knees with morphological abnormalities were characteristic of Osgood–Schlatter syndrome (50%), semimembranosus bursitis (19%), bipartite patella (15%), and patellar tendonitis (15%). The odds ratio of morphological abnormalities in participants with knee pain was elevated by 8.6-fold (95% CI 3.7–19.5; $P < 0.001$). Patella tendon CSA and thickness did not differ significantly between groups (Fig. 5).
Figure 5. Comparison of patellar tendon cross-sectional area (A) and thickness (B) in controls, symptomatic, and asymptomatic basketball players.

**DISCUSSION**

We found that adolescent basketball players with knee pain were taller and more frequently showed morphological abnormalities of the knee joint. Although these abnormalities may be related to greater stress on their knees compared with the other groups, there were no significant differences between asymptomatic and symptomatic players in the ability of their knee extensors and flexors to develop torque. There was also no evidence that the side-to-side differences in muscle strength contributed to the development of anterior knee pain in these adolescent basketball
players. Because a taller player has a longer femur and tibia, it is possible that the longer bones but similar muscle strength as in asymptomatic players placed greater stress on the knee joint because of the longer lever arm of the bones.

Consistent with previous investigations (Daneshjoo, Mokhtar, Rahnama, & Yusof, 2012; Śliwowski, Jadczak, Hejna, & Wieczorek, 2015), our study found that the dominant leg was stronger in each group. Bilateral strength differences were similar between groups, and there was no association between leg dominance and pain symptoms. These results are also consistent with data from upper body measurements showing that side-to-side differences between arms are unlikely to contribute to throwing-related pain in children (Mickevičius et al., 2016).

There is evidence that greater muscle strength is associated with a higher risk of injury. For instance, in adolescent female basketball players, greater hip abduction strength is associated with an increased risk for the development of patellofemoral pain (Herbst, 2015). Baseball players with greater throwing power are more prone to injury of the shoulder (Adirim & Cheng, 2003). At first glance, our data seem to support this notion because the basketball players had stronger knee extensors and flexors than controls. However, the knee extensors and flexors had a similar torque-generating capacity in asymptomatic and symptomatic players. Therefore, it seems unlikely that the knee pain in the symptomatic players was caused by the stronger knee extensors and flexors per se.

The lower coactivation during muscle contraction in players compared with controls is interesting because lower coactivation may be advantageous for the development of maximal voluntary contraction force. The cause of the lower coactivation in the basketball players is not known, but inappropriate coactivation of antagonist muscles may predispose the knee joint to a higher risk of injuries (Aagaard, Simonsen, Magnusson, Larsson, & Dyhre-Poulsen, 1998).
important perhaps is that inappropriate coactivation may have contributed to the impaired stability of trunk/lower-limb movements observed in the basketball players. Scattone and Serrão (2014) reported that impaired trunk/lower-limb kinematics are associated with an increased incidence of overuse knee injury in adolescent females. Consistent with this, we found that lumbopelvic balance during the active straight-leg raise test was associated with a more than twofold higher prevalence of knee pain in our sample. These observations suggest that particular emphasis should be placed on maintenance of core stability during athletic conditioning.

Both impaired stability of movement and limited ROM or hypermobility (extended ROM) have been reported to contribute to lower-extremity joint disorders (Tyler, Nicholas, Mullaney, & McHugh, 2006). In the symptomatic players in our study, limited ROM was greatest during the prone knee-bend test in the quadriceps. There is evidence that increased nerve mechanosensitivity plays a protective role in the prevention of muscle damage (Boyd et al., 2009). The smaller ROM was not only limited to the quadriceps, but also found in hip external rotation. Although a ROM deficit or hypermobility may contribute to knee problems in symptomatic players, these may not be directly related because ROM did not differ significantly between the symptomatic and asymptomatic players. Therefore, whether alterations in ROM are a prelude to knee problems remains unclear.

Similar to previous observations in bilateral sports (Zhang, Ng, Lee, & Fu, 2014), our study found no significant differences in PT CSA and thickness between groups. However, others have observed thickening and larger CSA in athletes in unilateral sports (Couppé et al., 2008) and female volleyball (bilateral) players (Toprak et al., 2012). The discrepancy between studies may be related to the type of sport and/or the sex of the athletes because sex and sport specificity are risk factors for patellar tendinopathy (van der Worp, van Ark, Roerink, Pepping, van den Akker-
Regardless of the reason for the discrepancies between studies, the lack of adaptation to the large stresses and strains on the tendon in the basketball players in the present study may have increased the risk for developing the clinical changes in the knee observed by ultrasound imaging in symptomatic players.

Ultrasound imaging revealed that 57% of the symptomatic group exhibited pathological changes in the knee joint. The odds ratios indicated that the prevalence of knee pain was 8.6-fold higher in athletes with morphological modifications in the knee joint. Interestingly, the ultrasound observations characteristic of Osgood–Schlatter syndrome were not accompanied by a lower torque-generating ability (data not shown). This lack of an association suggests that, despite these abnormalities, the tendon mechanical and material properties required to transmit force remain unchanged. Nevertheless, overuse or repetitive microinjuries can damage the muscle–tendon binding sites and, in turn, affect movement stability (Wild, Steele, & Munro, 2013). The observations that the symptomatic patients were taller and that 13 of the 26 detected abnormalities with ultrasound were characteristic of Osgood–Schlatter syndrome may indicate that the taller players were in their growth spurt. This syndrome is associated with more anterior knee pain because of inflammation of the PT at the tibial tuberosity beginning in late childhood.

None of the functional parameters differed significantly between symptomatic and asymptomatic players, and they are therefore unlikely to explain the development of knee pain in adolescent male basketball players. However, the symptomatic players were taller than the asymptomatic players, and it is possible that their greater height reflected a higher growth rate. This is significant because the incidence in the number of injuries during puberty (Brenner, 2007; Frank, Jarit, Bravman, & Rosen, 2007) is positively related to the rate of increase in height (Faulkner, Davison, Bailey, Mirwald, & Baxter-Jones, 2006).
One limitation of this study is that we did not assess the reproducibility of our ultrasound assessments and other tests. However, the literature (cited in the description of the different tests) shows good reliability and given that all procedures were performed by experienced researchers, we believe that the reliability of the different tests would have been as high as that reported. A second limitation is that anterior knee pain is multifactorial in nature, which makes assessment complicated. It is possible that recall of pain in the previous month introduced some bias, but this bias was minimized by asking the parents and coaches for confirmation of knee problems. In addition, it would be interesting if symptomatic players and asymptomatic players with morphological abnormalities were followed to assess whether the symptoms were transient and the morphological abnormalities preceded the development of symptoms. Finally, we did not assess the pubertal status of the participants, which would have helped us to better ascertain to what extent the pain was associated with accelerated growth.

**CONCLUSIONS**

Side-to-side differences of muscle strength, flexibility and neuromuscular control in 14–15-year-old male basketball players were not related to the early detection of anterior knee pain. However, the risk of knee pain was higher in tall players and was often accompanied by morphological abnormalities detected by ultrasound. The risk of pain was slightly related to a lower lumbopelvic balance, which highlights the need to maintain core stability during athletic conditioning.

**Conflict of interest**

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REFERENCES


