Do Bone–Patellar Tendon–Bone ACL-Reconstructed Knees Have More Signs of Patellofemoral Posttraumatic Osteoarthritis Than Their Uninjured Contralateral Knees at 2 Years?

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Background: The prevalence of patellofemoral joint (PFJ) osteoarthritis ranges from 8% to 47% at 7 to 10 years after anterior cruciate ligament reconstruction (ACLR) using bone–patellar tendon–bone (BTB) autograft. In performing BTB ACLR, some hypothesize that either trauma caused by harvest of the BTB autograft or altered biomechanics contributes to PFJ posttraumatic osteoarthritis.

Purpose/Hypothesis: To determine whether knees with ACLR using a BTB autograft show early signs of posttraumatic osteoarthritis as compared with the contralateral uninjured knee 2 years after ACLR. We hypothesized that a BTB autograft will not increase the prevalence of PFJ osteoarthritis.

Study Design: Cohort study; Level of evidence, 3.

Methods: Bilateral knee 3-T magnetic resonance imaging (MRI) scans were collected in 57 patients (mean age, 20.3 years; 28 men) from a single site at a minimum of 2 years after ACLR. Structural MRI assessment of the knees was performed using the MRI Osteoarthritis Knee Score semiquantitative scoring system by a board-certified musculoskeletal radiologist. The presence of cartilage defects in the patellofemoral compartment was compared between the reconstructed and contralateral uninjured knees using logistic regression analyses.

Results: There were no significant differences in the prevalence of cartilage defects (full thickness or any thickness) in the PFJ between the BTB ACLR knees and the contralateral control knees: 38.6% of BTB ACLR knees had PFJ cartilage defects versus 31.6% of contralateral control knees (P > .391). The 95% CI for the difference between these groups was –9.0% to 23.0%.

Conclusion: When comparing BTB ACLR knees with the uninjured contralateral knees in the study patients, we failed to observe statistically significant differences in the prevalence of PFJ cartilage lesions of full thickness or any thickness. These results should be used in shared decision-making with athletes when choosing the appropriate autograft during reconstruction. Our wide 95% CIs secondary to a smaller sample size demonstrate a need for larger studies in this area to more accurately describe the difference between the operative and contralateral knees.

Keywords: ACLR; patellofemoral joint; osteoarthritis
cartilage defects. In comparing OA among different graft types, Li et al.\textsuperscript{13} found a 17\% prevalence of radiographic PFJ OA in the hamstring ACLR group versus 30\% prevalence in the BTB ACLR group at 7 years postoperatively. However, graft type was not a significant predictor of radiographic OA after applying multivariable modeling. Similarly, Keays et al.\textsuperscript{12} reported a 30\% prevalence of radiographic PFJ OA after hamstring autograft ACLR versus 41\% prevalence after BTB autograft ACLR 6 years postoperatively, which was not a statistically significant difference. In a recent systematic review, there were mixed results showing a significant versus nonsignificant relationship between BTB ACLR and PFJ OA, with the authors concluding that there was limited evidence of no relationship.\textsuperscript{16}

To study PFJ OA after ACLR, we used the Multicenter Orthopaedic Outcomes Network (MOON) consortium cohort. This is a prospective cohort with standardized reporting of intraoperative findings and patient factors, rehabilitation protocols, and postoperative imaging techniques. The current article reports data from the MOON's nested cohort: a group of younger patients injured during sports, with previously uninjured bilateral knees and without any preexisting risk factors for OA or prior surgical treatments. This subgroup is ideally suited to evaluate the initiation, progression, and modifiable risk factors of early OA after ACLR.\textsuperscript{21}

Our study investigates how BTB autograft ACLR affects the PFJ cartilage morphology and OA based on 3-T magnetic resonance imaging (MRI) at a minimum of 2 years postoperatively. We hypothesize that a BTB autograft harvested using a minimally traumatic technique will not increase the risk of PFJ PTOA signs on MRI as compared with the uninjured contralateral knee.

**METHODS**

**Patients and Data Collection**

This study was approved by our hospital's institutional review board. Patients were enrolled from the practice of a single senior surgeon (K.P.S.) from within the MOON's prospective nested cohort. These patients underwent BTB autograft ACLR between December 1, 2006, and November 1, 2008. Sample size was determined by including all possible patients in this date range based on the following criteria: an injury sustained while participating in a sport, a primary ACLR without any other concomitant ligamentous surgery, no previous surgery on the contralateral knee, no subsequent revision ACLR on the index knee at the time of follow-up, and age <36 years at the time of 2-year follow-up. At the time of surgery, patients completed standardized forms with information including demographics and patient-reported outcome measures.\textsuperscript{15} At the same time, the surgeon filled out a standardized data collection form detailing meniscal and articular cartilage status/treatment. Every patient was given a standardized rehabilitation protocol outlined by Wright et al.\textsuperscript{22}

Patients returned for on-site follow-up as part of the nested cohort at a minimum of 2 years after primary ACLR for radiographs, physical examination, and completion of the same set of patient-reported outcome instruments as had been completed at baseline. Additionally, these individuals had MRI scans of their operative and contralateral nonoperative knees as part of a bioabsorbable screw study.\textsuperscript{3}

Patient enrollment and follow-up are highlighted in Figure 1.

**Surgical Technique**

A 2-incision ACLR technique with BTB autograft was used for all patients in the cohort.\textsuperscript{14} A BTB autograft was harvested from the ipsilateral knee using a single longitudinal incision. After dissection of the patellar tendon, the central third of the tendon was used with a 20 to 25 mm–long bone block from the patella and tibial tubercle. For patellar bone block harvest, steps were taken to prevent iatrogenic compression of the patellar and trochlear articular cartilage: a narrow blade oscillating saw was used to a depth of 6 to 7 mm angled to create a trapezoidal bone block; the cuts were not extended to the posterior cortex of the patella; and the patellar bone block was...
MRI and Scoring at 2 Years

Imaging was performed on the surgical and contralateral knees on 3-T MRI (Philips Achieva) with a dedicated knee coil. The protocol included intermediate-weighted sagittal turbo spin echo imaging (repetition time [TR] / echo time [TE] = 2930/30 ms; voxel, 0.3 × 0.3 × 2.5 mm³), fat-saturated turbo spin echo imaging (axial, TR/TE = 3269/30 ms [voxel, 0.5 × 0.5 × 4 mm³]; coronal, TR/TE = 2270/30 ms [voxel, 0.5 × 0.5 × 4 mm³]), and 3-dimensional coronal gradient echo imaging (fast-field echo; TR/TE = 20/7.6 ms; voxel, 0.3 × 0.3 × 1.5 mm³). The MRI Osteoarthritis Knee Score (MOAKS) semiquantitative scoring system was used to score the surgical and contralateral knees. A board-certified musculoskeletal radiologist (F.A.) performed all scoring. ACL status could not be blinded as a result of the ability to see tunnel remnants and hardware in the surgical knee. Scored components on each MRI scan included the following: cartilage defects in each knee compartment (medial, lateral, patellofemoral), meniscal tears (medial, lateral; yes/no), bone marrow edema–like lesions (medial, lateral, patellofemoral; yes/no), effusion (yes/no), and Hoffa synovitis (yes/no). In the PFJ, knees were graded by percentage of surface area involved with cartilage loss of any thickness or full thickness, and this was summarized and reported as prevalence of cartilage lesions in each knee. Bone marrow edema–like lesions in each subregion were also counted as the total number of discrete lesions.

Statistical Analysis

Descriptive statistics were used to summarize demographics, MOAKS differences between surgical and contralateral knees, and patient-reported outcome changes over time. To compare MRI differences in the entire knee between surgical and contralateral knees, hypothesis testing was performed using logistic regression analysis. The PFJ was 1 of the 3 compartments analyzed (the other being the medial and lateral compartments). Logistic regression models were fit where the dependent variable was cartilage loss (presence or absence) and the independent variable was knee status (surgical or contralateral). Two models were created: 1 with any full-thickness cartilage loss and 1 with any cartilage lesions in the patellofemoral compartment. The knee was used as the unit of analysis. Generalized estimating equations were used to account for the correlation between knees from the same patient. P < .05 was considered statistically significant. The modeling results and statistical testing presented in this article are for the PFJ.

RESULTS

A total of 57 patients with BTB ACLR returned on-site at a minimum of 2 years postsurgically (mean ± SD time from surgery to testing, 2.9 ± 0.1 years). The mean age of the patients was 20.3 years, with 28 men in the group (Table 1). During baseline diagnostic arthroscopy, 4 patients had Outerbridge grade 1 and 2 changes on the patella, and 1 patient had Outerbridge grade 4 changes on the trochlea. All other patients had normal patellofemoral joint cartilage morphology on diagnostic arthroscopy at baseline.

The BTB ACLR knees had more cartilage defects in the tibiofemoral medial and lateral compartments and more meniscal tears, bone marrow edema, effusion, and Hoffa synovitis than the contralateral knees (Table 2). There

| TABLE 1 | Patient Characteristics |
|---------|-------------------------|
| Age, y  | 20.3 ± 5.12 (13-32)     |
| Body mass index, kg/m² | 23.9 ± 3.64 (18.3-37.7) |
| Female: male | 29:28 (50:9:49.1) |
| Smoking status, never:quit >6 moccurrence | 49:6:2 |
| Education, y | 12.5 ± 3.03 (7-19) |
| Meniscal treatment, no tear:tear | 31:26<sup>a</sup> |
| Medial | 16:4:1<sup>b</sup> |
| Lateral |

<sup>a</sup>Tear: 8, no treatment; 12, repair; 2, abrade and trephine; 4, excision.
<sup>b</sup>Tear: 13, no treatment; 2, repair; 2, abrade and trephine; 24, excision.
were no statistically significant differences in the prevalence of cartilage defects (any lesions or full-thickness loss) in the PFJ between the surgical BTB ACLR knees and the uninjured contralateral control knees: 15.8% of BTB ACLR knees had full-thickness PFJ cartilage defects versus 12.3% of contralateral control knees ($P = 0.478$) (Table 3). Similarly, 38.6% of BTB ACLR knees had any PFJ cartilage lesion as compared with 31.6% of contralateral control knees ($P = 0.391$) (Table 4). The 95% CI for the difference between these groups was –9.0% to 23.0%. If knees did have any cartilage lesion, they were most prevalent in the medial patella. Figure 2 shows examples of pathology in the PFJ on MRI for the reconstructed and contralateral uninjured knees. At a minimum of 2-year follow-up, outcomes on the KOOS (Knee injury and Osteoarthritis Outcome Score) for Symptoms, Sports, Pain, and Activities of Daily Living scores were maintained at a high level, with means ranging from 87 to 98.

**DISCUSSION**

The purpose of this study was to determine whether any PFJ differences were present between knees in a prospective cohort of 57 young, active patients undergoing BTB ACLR who were followed up a minimum of 2 years after surgery. With the patients available in this study, we failed to observe a difference in the prevalence of MRI-detected PFJ cartilage defects between BTB ACLR knees and contralateral nonoperated knees 2 years after surgery with the aforementioned BTB harvest technique. There was no difference found between BTB ACLR knees and contralateral knees in the presence of any full-thickness cartilage defect or any cartilage defect in the PFJ. However, the wide 95% CI of –9.0% to 23.0% for any-thickness cartilage defects, which is a factor of the smaller sample size, indicates that a larger study is necessary to make an accurate statement regarding the difference in prevalence between the operative and contralateral knees. There was a relatively high prevalence of PFJ cartilage lesions in both groups, with 38.6% of BTB ACLR knees and 31.6% of contralateral control knees having a lesion of any thickness. Regardless of the prevalence of PFJ OA, patients maintained high levels in patient-reported outcomes, with KOOS means ranging from 87 to 98 (of 100 points) for Symptoms, Sports, Pain, and Activities of Daily Living.

Radiographic PFJ PTOA has been demonstrated across numerous studies regardless of graft type, with a median prevalence of 36% at 2 to 15 years after ACLR. Specifically
examining BTB autograft ACLR, Järvelä et al\(^{10}\) showed a 47% incidence of radiographic PFJ PTOA 7 years postsurgically, and Ahn et al\(^{1}\) reported a 7.6% prevalence of radiographic PFJ PTOA 10 years after surgery. These radiographic studies have been informative in demonstrating the impact of PFJ PTOA with a longer follow-up, but they do not address the clinically important question about the impacts of early, preradiographic PTOA on articular cartilage after ACLR. As used in the current study, MRI is ideally suited to answer questions about early PFJ PTOA, as it has shown a greater sensitivity than radiographs to the early changes of OA.\(^{20}\)

Various study designs using MRI have been employed to investigate early PFJ PTOA after ACLR, with most showing a high prevalence at an early time point. Culvenor et al\(^{4}\) compared hamstring autograft ACLR knees with uninjured matched controls using MOAKS 1 year after surgery, and they found a prevalence of 17% PFJ PTOA as compared with uninjured controls, who had no PFJ OA features.

The femoral trochlea was the most affected region in that study, and similar to our findings, it showed that trochlear-sided defects were more prevalent in the ACLR knee than the contralateral control knee (Tables 3 and 4). It is possible that the patellar cartilage, which is thicker, may be more resilient to major changes over the course of 2 years, but the trochlea, with thinner cartilage, could break down faster. Frobell\(^{7}\) used serial quantitative MRI to study ACL-injured knees treated with reconstruction with hamstring or BTB autografts or nonoperatively over 2 years. The study found significant cartilage thinning occurring in the trochlea; and
interestingly, after 2 years, operative versus nonoperative treatment was not related to any differences in cartilage morphology. Last, Patterson et al. found progression of PFJ PTOA using MOAKS from 1 to 5 years after hamstring ACLR, with worsening of cartilage defects in 44% of patients over the course of 4 years. These studies reported hamstring autograft or mixed graft ACLR in the patient population, which makes it difficult to draw conclusions about early PFJ PTOA when using BTB autograft. Our study is unique to previous MRI PTOA studies in that our cohort includes only patients who have undergone BTB autograft ACLR and we have compared the findings with the contralateral uninjured knees. The contralateral uninjured knee serves as an ideal control in that it perfectly matches the injured knee’s age, sex, body mass index, smoking status, and activity level. Like prior MRI studies, our study demonstrated a higher-than-anticipated 38.6% prevalence of any grade PFJ cartilage lesions 2 years after surgery. Importantly, there was no statistically significant difference found between BTB ACLR knees and contralateral knees in the prevalence of any full-thickness lesions and any cartilage defect in the PFJ, although this conclusion is made in the context of a wide confidence interval because of our sample size. With these results, factors other than graft type (eg, altered PFJ biomechanics or quadriceps dysfunction) could be causative of PFJ OA in injured and uninjured knees in these patients. When using MRI and dual-orthogonal fluoroscopy to study patellar biomechanics, there is persistent abnormal patellar rotation, tilt, and lateral shift in PFJ cartilage contact even after ACLR.6

The current study has several limitations. The sample size of 57 patients who had MRI scans on both knees was small, which led to the wide confidence interval when determining the difference between PFJ OA in the operative and contralateral knees. This sample size included all possible patients in the study period and was not easily expandable; however, a larger sample size would allow a stronger conclusion to be drawn. This study’s conclusions are hypothesis-generating for future studies, and counseling patients with these results is merely 1 factor in many that help surgeons determine graft type. Furthermore, this study’s analysis assesses only for the presence of full-thickness or any-thickness cartilage defects in the PFJ as a whole—not the number, severity, or size of cartilage defects. Evaluation of other risk factors associated with PTOA presence or severity was not conducted in this population. Future investigations can be conducted to further assess risk factors and PTOA severity.

CONCLUSION

When comparing BTB ACLR knees with the contralateral uninjured knees, we failed to observe statistically significant differences in the prevalence of PFJ cartilage lesions of any or full thickness with our BTB harvesting technique. However, our smaller sample size resulted in a large confidence interval, so a larger study is needed to more accurately describe the differences. These results can be valuable in counseling patients before ACLR and can be 1 factor among many in choosing the appropriate graft during reconstruction.

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REFERENCES

1. Ahn JH, Kim JG, Wang JH, Jung CH, Lim HC. Long-term results of anterior cruciate ligament reconstruction using bone–patellar tendon–bone: an analysis of the factors affecting the development of osteoarthritis. Arthroscopy. 2012;28(8):1114-1123.
2. Claes S, Hermie L, Verdonk R, Bellermans J, Verdonk P. Is osteoarthritis an inevitable consequence of anterior cruciate ligament reconstruction? A meta-analysis. Knee Surg Sports Traumatol Arthrosoc. 2013;21(9):1967-1976.
3. Cox CL, Spindler KP, Leonard JP, Morris BJ, Dunn WR, Reinke EK. Do newer-generation bioabsorbable screws become incorporated into bone at two years after ACL reconstruction with patellar tendon graft? A cohort study. J Bone Joint Surg Am. 2014;96(3):244-250.
4. Culvenor AG, Collins NJ, Guermazi A, et al. Early patellofemoral osteoarthritis features one year after anterior cruciate ligament reconstruction: symptoms and quality of life at three years. Arthritis Care Res. 2016;68(6):784-792.
5. Culvenor AG, Cook JL, Collins NJ, Crossley KM. Is patellofemoral joint osteoarthritis an under-recognised outcome of anterior cruciate ligament reconstruction? A narrative literature review. Br J Sports Med. 2013;47(2):66-70.
6. de Velde SKV, Gill TJ, DeFrante LE, Papannagari R, Li G. The effect of anterior cruciate ligament deficiency and reconstruction on the patellofemoral joint. Am J Sports Med. 2008;36(6):1150-1159.
7. Frobell R. Change in cartilage thickness, posttraumatic bone marrow lesions, and joint fluid volumes after acute ACL disruption: a two-year prospective MRI study of sixty-one subjects. J Bone Joint Surg Am. 2011;93(12):1096-1103.
8. Hunter DJ, Guermazi A, Lo GH, et al. Evolution of semiquantitative whole joint assessment of knee OA: MOAKS (MRI Osteoarthritis Knee Score). Osteoarthritis Cartilage. 2011;19(8):990-1002.
9. Hunter DJ, Lo GH, Gale D, Grainger AJ, Guermazi A, Conaghan PG. The reliability of a new scoring system for knee osteoarthritis MRI and the validity of bone marrow lesion assessment: BLOKS (Boston Leeds Osteoarthritis Knee Score). Ann Rheum Dis. 2008;67(2):206-211.
10. Järvelä T, Paakkala T, Kannus P, Järvinen M. The incidence of patellofemoral osteoarthritis and associated findings 7 years after anterior cruciate ligament reconstruction with a bone–patellar tendon–bone autograft. Am J Sports Med. 2001;29(1):18-24.
11. Kaeding CC, Léger-St-Jean B, Magnussen RA. Epidemiology and diagnosis of anterior cruciate ligament injuries. *Clin Sports Med*. 2017;36(1):1-8.

12. Keays SL, Bullock-Saxton JE, Keays AC, Newcombe PA, Bullock MI. A 6-year follow-up of the effect of graft site on strength, stability, range of motion, function, and joint degeneration after anterior cruciate ligament reconstruction. *Am J Sports Med*. 2007;35(5):729-739.

13. Li RT, Lorenz S, Xu Y, Harner CD, Fu FH, Irgang JJ. Predictors of radiographic knee osteoarthritis after anterior cruciate ligament reconstruction. *Am J Sports Med*. 2011;39(12):2595-2603.

14. Magnussen RA, Spindler KP. Anterior cruciate ligament reconstruction: two-incision technique. *Oper Tech Sports Med*. 2013;21(1):34-39.

15. Marx RG, Stump TJ, Jones EC, Wickiewicz TL, Warren RF. Development and evaluation of an activity rating scale for disorders of the knee. *Am J Sports Med*. 2001;29(2):213-218.

16. Meer BLV, Meuffels DE, Eijsden WAV, Verhaar JAN, Bierma-Zeinstra SMA, Reijman M. Which determinants predict tibiofemoral and patellofemoral osteoarthritis after anterior cruciate ligament injury? A systematic review. *Br J Sports Med*. 2015;49(15):975-983.

17. MOON Knee Group, Jones MH, Oak SR, et al. Predictors of radiographic osteoarthritis 2 to 3 years after anterior cruciate ligament reconstruction: data from the MOON on-site nested cohort. *Orthop J Sports Med*. 2019;7(8):2325967119867085.

18. Øiestad BE, Engebretsen L, Storheim K, Risberg MA. Knee osteoarthritis after anterior cruciate ligament injury. *Am J Sports Med*. 2009;37(7):1434-1443.

19. Patterson BE, Culvenor AG, Barton CJ, et al. Worsening knee osteoarthritis features on magnetic resonance imaging 1 to 5 years after anterior cruciate ligament reconstruction. *Am J Sports Med*. 2018;46(12):2873-2883.

20. Rowbotham EL, Grainger AJ. Magnetic resonance imaging of arthritis of the knee. *Semin Musculoskelet Radiol*. 2017;21(2):113-121.

21. Spindler KP, Parker RD, Andrish JT, et al. Prognosis and predictors of ACL reconstructions using the MOON cohort: a model for comparative effectiveness studies. *J Orthop Res*. 2013;31(1):2-9.

22. Wright RW, Haas AK, Anderson J, et al. Anterior cruciate ligament reconstruction rehabilitation. *Sports Health*. 2015;7(3):239-243.