PODCAST 5-in-5

The Predicted Position of the Knee Near the Time of ACL Rupture Is Similar Between 2 Commonly Observed Patterns of Bone Bruising on MRI

Sophia Y. Kim-Wang,*† PhD, Charles E. Spritzer,‡ MD, Kwadwo Owusu-Akyaw,* MD, James A. Coppock,*† MS, Adam P. Goode,*§¶ DPT, PhD, Zoë A. Englander,* PhD, Jocelyn R. Wittstein,* MD, and Louis E. DeFrate,*†||# ScD
Investigation performed at Duke University, Durham, North Carolina, USA

Background: Bone bruises observed on magnetic resonance imaging (MRI) can provide insight into the mechanisms of noncontact anterior cruciate ligament (ACL) injury. However, it remains unclear whether the position of the knee near the time of injury differs between patients evaluated with different patterns of bone bruising, particularly with regard to valgus angles.

Hypothesis: The position of the knee near the time of injury is similar between patients evaluated with 2 commonly occurring patterns of bone bruising.

Study Design: Descriptive laboratory study.

Methods: Clinical T2- and T1-weighted MRI scans obtained within 6 weeks of noncontact ACL rupture were reviewed. Patients had either 3 (n = 20) or 4 (n = 30) bone bruises. Patients in the 4–bone bruise group had bruising of the medial and lateral compartments of the femur and tibia, whereas patients in the 3–bone bruise group did not have a bruise on the medial femoral condyle. The outer contours of the bones and associated bruises were segmented from the MRI scans and used to create 3-dimensional surface models. For each patient, the position of the knee near the time of injury was predicted by moving the tibial model relative to the femoral model to maximize the overlap of the tibiofemoral bone bruises. Logistic regressions (adjusted for sex, age, and presence of medial collateral ligament injury) were used to assess relationships between predicted injury position (quantified in terms of knee flexion angle, valgus angle, internal rotation angle, and anterior tibial translation) and bone bruise group.

Results: The predicted injury position for patients in both groups involved a flexion angle <20°, anterior translation >20 mm, valgus angle <10°, and internal rotation angle <10°. The injury position for the 3–bone bruise group involved less flexion (odds ratio [OR], 0.914; 95% CI, 0.846-0.987; P = .02) and internal rotation (OR, 0.832; 95% CI, 0.739-0.937; P = .002) as compared with patients with 4 bone bruises.

Conclusion: The predicted position of injury for patients displaying both 3 and 4 bone bruises involved substantial anterior tibial translation (>20 mm), with the knee in a straight position in both the sagittal (<20°) and the coronal (<10°) planes.

Clinical Relevance: Landing on a straight knee with subsequent anterior tibial translation is a potential mechanism of noncontact ACL injury.

Keywords: anterior cruciate ligament injury; bone contusion; kinematics; MRI

An estimated 400,000 anterior cruciate ligament (ACL) injuries occur annually in the United States.[28] Of these injuries, 70% occur via a noncontact mechanism.[5] Currently, surgical ACL reconstruction and rehabilitation are a mainstay of treatment. However, pain, knee instability, and osteoarthritis may occur regardless of surgical treatment.[1,21,42] Despite efforts at prevention,[44,49] the incidence of ACL injury is increasing.[2,7,36,55] Furthermore, the mechanisms of noncontact ACL injury remain controversial.[11,26,41,47] An improved understanding of the mechanisms of noncontact ACL rupture may improve efforts toward injury prevention, thus reducing injury rates and the consequences of ACL injury.

Importantly, there are limited in vivo data to describe the motions that lead to noncontact ACL injury. Studies have estimated the position of the knee near the time of ACL rupture by analyzing video footage of injuries.[9,27,33,38] Although these studies have provided important insight into the position of the knee near the time of

The American Journal of Sports Medicine
2023;51(1):58–65
DOI: 10.1177/03635465221131551

ACL injury, they are limited by the inherent sensitivity of these analyses to the position of the camera relative to the injured knee.[16] Furthermore, it is not possible to determine the exact moment of ACL rupture in these video analyses.[33,38] Therefore, the knee angles observed in these analyses may be the result of an ACL tear rather than the cause.[38]

As an alternative to studying videos of injury, investigators have analyzed the locations of bone bruises observed by magnetic resonance imaging (MRI) of ACL-injured patients.[24,35,43,48,53] Bone bruises are hypothesized to represent edema and trabecular microfracture resulting from impact between the femur and tibia near the time of injury.[24,48,53] Previous work has suggested that different patterns of bone bruising may result from different noncontact ACL injury mechanisms.[24,40,48] More recently, studies of bone bruises in ACL injury patients have quantitatively predicted the position of the knee near the time of ACL injury using 3-dimensional (3D) modeling and numerical optimization techniques.[29,39] Specifically, these studies predicted knee kinematics near the time of ACL rupture by positioning the femur and tibia to maximize the overlap of corresponding bone bruises.[29,39]

Previous studies have predicted the position of the knee near the time of injury in patients evaluated with 4 bone bruises using these optimization techniques.[29,39] Specifically, this pattern includes bruises on the medial and lateral femoral condyles (MFCs and LFCs, respectively) and the medial and lateral tibial plateaus (MTPs and LTPs, respectively). This is the most frequently observed pattern of bone bruises in noncontact ACL-injured patients and is identified in approximately 35% of injuries.[30] However, it is possible that different bone bruising patterns are indicative of different mechanisms of injury.[12,48]

Therefore, the purpose of this study was to determine the position of the knee near the time of noncontact ACL injury in patients evaluated with the second most commonly observed bone bruise pattern.[30] This pattern involves 3 bone bruises on the LFC, MTP, and LTP and is observed in approximately 24% of patients.[30] Specifically, we aimed to compare the predicted position of injury for this pattern of 3 bone bruises with that predicted for patients evaluated with 4 bone bruises.[29,39] On the basis of previous work,[29,39] we hypothesized that the predicted position of injury would involve a low flexion angle (extended knee) and large anterior tibial translations in patients displaying both of these patterns of 3 and 4 bone bruises.

METHODS

Inclusion Criteria

With approval from the institutional review board, we retrospectively reviewed clinical MRI scans of patients with noncontact ACL injuries. Our inclusion criteria were (1) MRI scan acquired within 6 weeks of injury, (2) noncontact injury, and (3) bone bruises visible in only the LTP, MTP, and LFC regions. These inclusion criteria were the same as those specified by previous studies,[29,39] except these patients had this pattern of 3 bone bruises, rather than 4 bruises. The sample size for this study was determined using previously collected data, whereby a minimum of 15 participants in each group provided sufficient power to detect differences of 3° of valgus between groups with 80% power.[39] Therefore, 20 patients who displayed the 3–bone bruise pattern and 30 patients from a previous study[39] who displayed the 4–bone bruise pattern were included in this analysis.

Sagittal plane clinical T2-weighted images with the following specifications were reviewed: magnetic field strength of 1.5 or 3.0 T, field of view of 140 to 180 mm, image matrices of 384 × 384 or 512 × 512 pixels, slice thickness of 3 or 4 mm, repetition time range of 3100 to 6016.7 ms, and echo time range of 63 to 75.4 ms.

Data Analysis

The cortical bone, articular cartilage, and the surfaces of the bone bruises on the femur and tibia were manually segmented by a single rater (S.Y.K.-W.) from the sagittal T2-weighted MRI scans of each patient's injured knee (Figure 1A). All manual bone, cartilage, and bruise surface segmentations were confirmed by the same musculoskeletal radiologist with more than 30 years of experience (C.E.S.). Importantly, during the segmentation review process, T2-weighted images were cross-referenced with corresponding T1-weighted images to confirm the focal regions of disruption on the outer margin of the bone (Figure 2). Segmentations were then compiled into wire frame models, which were then used to generate 3D surface models of the femur, tibia, and associated bone bruises (Figure 1B).[29,39]

To generate the predicted position of the knee near the time of injury, a numerical optimization algorithm was used to rigidly translate and rotate the tibia relative to

[#]Address correspondence to Louis E. DeFrate, ScD, Department of Orthopaedic Surgery, Duke University Box 3093, Durham, NC 27710 USA (email: lou.defrate@duke.edu).

*Department of Orthopaedic Surgery, Duke University School of Medicine, Durham, North Carolina, USA.

[†]Department of Biomedical Engineering, Duke University, Durham, North Carolina, USA.

[‡]Department of Radiology, Duke University School of Medicine, Durham, North Carolina, USA.

[§]Department of Population Health Sciences, Duke University School of Medicine, Durham, North Carolina, USA.

[||]Department of Mechanical Engineering and Materials Science, Duke University, Durham, North Carolina, USA.

[¶]Duke Clinical Research Institute, Duke University School of Medicine, Durham, North Carolina, USA.

Submitted December 10, 2021; accepted September 1, 2022.

One or more of the authors has declared the following potential conflict of interest or source of funding: This project was funded in part by the National Institutes of Health (AR065527, AR074800, AR079184, and AR075399). K.O.-A. has received support for education from Smith & Nephew and Fortis Surgical. J.R.W. has received support for education and speaking fees from Southtech Orthopaedics, and compensation for services other than consulting from Arthrex. AOSSM checks author disclosures against the Open Payments Database (OPD). AOSSM has not conducted an independent investigation on the OPD and disclaims any liability or responsibility relating thereto.

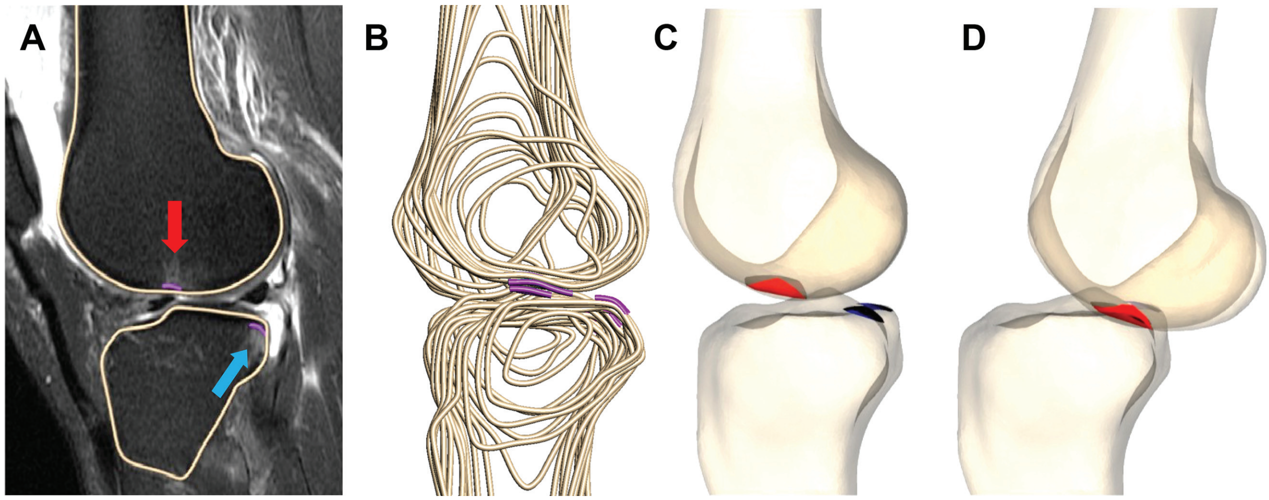

Figure 1. (A) The cortical bones (beige) and associated bone bruises (purple) were segmented from the T2-weighted magnetic resonance imaging (MRI). The segmentations were used to create (B) a wire frame and (C, D) 3-dimensional surface models of the bones and associated bone bruises. The position of the knee was reproduced in the (C) MRI reference position and (D) predicted position of injury. Blue indicates tibial bone bruises; red, femoral bone bruises.

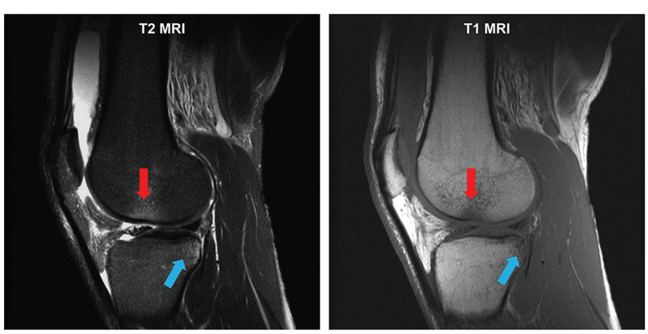

Figure 2. T2-weighted magnetic resonance imaging (MRI; left) and corresponding T1-weighted MRI (right). Arrows indicate positions of the femoral and tibial bone bruises. Note that the epicenters of the bone bruises are more apparent on the T1 images, whereas the bruises appear more diffuse on the T2 images.

the femur in 6 degrees of freedom. The goal of optimization was to minimize the distances between evenly spaced points across the surfaces of the tibial and femoral bruises.[29,39] Additionally, the optimization was constrained to minimize penetration of the bony surfaces of the femur and tibia.[29,39]

Because no MFC bruise was observed in the 3–bone bruise group, the previously described optimization algorithm used in the 4–bone bruise group[29] was modified. Specifically, the algorithm used in the 3–bone bruise group assumed that the MTP bruise resulted from contact with the MFC, rather than contact between the MTP and MFC bruises as in the 4–bone bruise case.[29,39] Thus, the optimization algorithm sought to minimize the distance between the MTP bruise and the nearest points on the surface of the MFC. After optimization, flexion angle, valgus angle, anterior tibial translation, and internal tibial rotation angle were measured relative to the unloaded MRI position (Figure 1, C and D) using a previously established coordinate system.[29,39]

Repeatability of Injury Prediction Algorithm Based on Locations of 3 Bone Bruises

Previous work assessed the repeatability of predicting the position of injury based on the locations of 4 bone bruises.[29] Here we performed a similar assessment of intrarater repeatability for this study. Specifically, a single investigator (S.Y.K.-W.) segmented the surfaces of the bone bruises on a single patient's knee in 3 independent trials. The coefficient of variation of the tibiofemoral bone bruise surface areas was within 3% of the mean total area. We then predicted the position of injury for each independent segmentation trial using the optimization algorithm described previously. Standard deviations in predicted position of injury across the 3 trials were flexion angle = 0.8°, valgus angle = 0.3°, anterior tibial translation = 0.03 mm, and internal tibial rotation angle = 0.1°. These analyses demonstrate that this methodology is repeatable in describing bruise areas, resulting in consistent predictions of the position of the knee near the time of ACL injury for patients with 3 bone bruises.

In the present study, a single investigator (S.Y.K.-W.) performed the bone bruise segmentations, which were reviewed by a musculoskeletal radiologist (C.E.S.). Nonetheless, we performed an additional analysis to assess the interrater repeatability of our segmentation technique. Specifically, 2 additional investigators (J.A.C., L.E.D.) segmented the bone bruises of 3 randomly selected participants. Interrater repeatability was assessed by quantifying the intraclass correlation coefficient (ICC; 2-way mixed-effects, absolute agreement) and the coefficient of variation of bone bruise surface

TABLE 1
Patient Characteristics and Predicted Injury Kinematics by Sex and Bone Bruise Pattern[a]

	3 Bruises (n = 20)	4 Bruises (n = 30)	*P* Value
Sex, female/male, n	11/9	15/15	.70
Age, y	25 ± 10	22 ± 6	.14
BMI	28.0 ± 5.6	24.9 ± 4.2	**.03**
Injury to MRI, d	8.4 ± 6.1	9.7 ± 6.9	.49
MCL injury, yes (%)	10 (50)	17 (57)	.51
Flexion, deg	11.6 ± 8.4	19.3 ± 10.9	**.008**
Valgus, deg	7.4 ± 3.1	8.2 ± 4.7	.51
Internal tibial rotation, deg	–0.1 ± 3.3	6.9 ± 7.9	**<.001**
Anterior tibial translation, mm	23.9 ± 5.1	25.7 ± 4.7	.20

[a]Data are presented as mean ± SD unless otherwise indicated. Significance assessed via Student *t* tests. Boldface type indicates statistical significance ($P < .05$). BMI, body mass index; MCL, medial collateral ligament; MRI, magnetic resonance imaging.

areas. Results from this analysis yielded an ICC(2, k) = 0.94 and a coefficient of variation in bruise surface areas within 4% of the mean total area.

Validity of Position of Injury Prediction Algorithm Based on Locations of 3 Bone Bruises

After establishing the inter- and intrarater repeatability of the algorithm used in patients with 3 bone bruises, we then assessed its validity in predicting the injury position. Specifically, we compared the predicted position of injury using the 3–bone bruise optimization algorithm with the position predicted by the 4–bone bruise optimization algorithm within the same patients. To do so, we sampled 8 patients from a previous study who had 4 bone bruises.[39] Based on previous work,[29] we estimated that for a paired design, 8 patients would provide 80% power to detect differences of >2° in valgus between analyses performed using 3 versus 4 bone bruises within the same patients.

In each of the patients with 4 bone bruises, we removed the MFC bruise to simulate the 3–bone bruise pattern of interest. We then predicted the position of injury using the 3–bone bruise optimization algorithm as described previously and compared it with the position of injury as predicted by 4 bone bruises. Because the position of the knee was compared in the same knees for the 2 different bone bruising patterns (4 bone bruises and a simulation of 3 bone bruises), paired *t* tests were used to compare the results from both techniques. No statistically significant differences in the predicted kinematic variables were detected between the 2 methods ($P > .05$).

Statistical Analysis

Routine descriptive characteristics (mean ± SD) were used to summarize the data (Table 1). Normality assumptions and the presence of data outliers were tested via visual inspection of kernel density plots of the residuals and the inner and outer fences of the interquartile ranges of the residuals, respectively. Unpaired Student *t* tests were used to detect differences between the 3– and 4–bone bruise groups in the continuous variables (kinematic variables, age, body mass index [BMI]). Two-sample proportion tests were used to detect differences between the 3– and 4–bone bruise groups in the categorical variables (sex, medial collateral ligament [MCL] injury). Additionally, 4 multiple logistic regression models were constructed to investigate the relationship between each kinematic variable and the bruise patterns, while controlling for potential confounding variables (age, BMI, sex, MCL injury involvement).

With regard to MCL injury involvement, MRI scans were used to classify MCL integrity as grade 0 (n = 23), grade 1 (n = 26), or grade 2 (n = 1).[45] We conducted a sensitivity analysis to assess potential effects of differences in MCL grade by removing the 1 observation of a patient with a grade 2 MCL sprain and rerunning the logistic regression models. Removal of this 1 patient from the analyses resulted in trivial (<10%) changes in the odds ratios. Thus, the patient with the grade 2 MCL sprain was included with the grade 1 MCL sprain patients. For all logistic regression models, the 4-bruise pattern was used as the reference category. All analyses were conducted in R (R Core Team, 2020). Alpha was set a priori at $P < .05$.

RESULTS

A total of 20 patients (11 female, 9 male) who displayed the 3–bone bruise pattern and 30 patients (15 male, 15 female) from a previous study[39] who displayed the 4–bone bruise pattern were included in this analysis. Characteristics of the patient cohorts, including age, BMI, time between injury and MRI, and presence of MCL injury are presented in Table 1. No significant differences were detected in the proportion of male and female participants ($P = .70$) or the presence of an MCL injury ("yes" vs "no"; $P = .51$) between groups. We observed a significant difference in BMI between the 3– and 4–bone bruise patient cohorts ($P = .03$).

Additional imaging findings in the 3–bone bruise cohort included (1) MRI evidence[45] of grade 1 MCL sprain (intact ligament with periligamentous edema in 6 female and 3 male participants), (2) MRI evidence of grade 2 MCL injury (abnormal signal within the ligament with no ligamentous discontinuity in 1 male participant), (3) lateral meniscal

TABLE 2
Coefficient Estimations for Each Kinematic Variable Predicting the Presence of 3 Bone Bruises[a]

Kinematic Variable	OR (95% CI)	*P* Value
Flexion, deg	0.914 (0.846-0.987)	**.02**
Valgus, deg	0.978 (0.85-1.145)	.78
Internal tibial rotation, deg	0.832 (0.739-0.937)	**.002**
Anterior tibial translation, mm	0.946 (0.824-1.087)	.44

[a]Referent: 4 bone bruises. Models were generated for each kinematic variable, adjusted for sex, age, body mass index, and medial collateral ligament involvement. Boldface type indicates statistical significance ($P < .05$). OR, odds ratio.

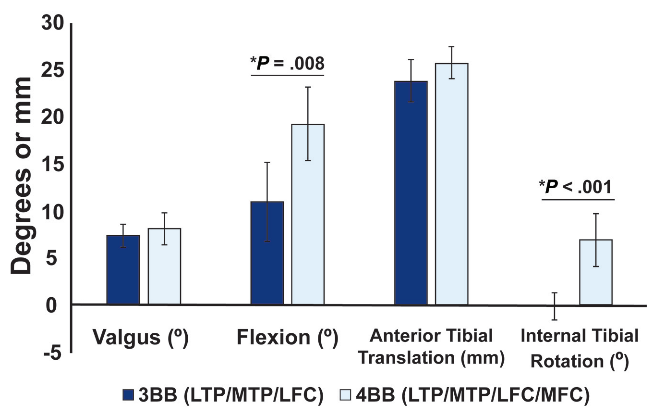

Figure 3. Predicted kinematics near the time of injury (mean ± 95% CI) for the 3–bone bruise (BB) (LTP/MTP/LFC) and 4-BB (LTP/MTP/LFC/MFC) groups. LFC, lateral femoral condyle; LTP, lateral tibial plateau; MFC, medial femoral condyle; MTP, medial tibial plateau. *Statistically significant at $P < .05$.

tear (2 female participants), and (4) medial meniscal tear (6 female and 8 male participants).

The predicted position of the knee near the time of ACL rupture is presented in Figure 3. In comparing the predicted injury positions between groups, the 3-bruise group demonstrated significantly less flexion (mean difference, 7.7°; P = .008) and internal tibial rotation (mean difference, 7.0°; $P < .001$) as compared with the 4-bruise group. No significant differences between groups were observed in the other kinematic variables (Table 1).

The multiple logistic regression analyses (Table 2), which were adjusted for multiple confounding variables (age, BMI, sex, MCL involvement), were consistent with the results obtained from the unpaired *t* tests (Table 1). Specifically, a significant predictive relationship was detected between flexion and the 3–bone bruise pattern (OR, 0.914; 95% CI, 0.846-0.987; P = .02) and between internal tibial rotation and the 3–bone bruise pattern (OR, 0.832; 95% CI, 0.739-0.937; P = .002). These results suggest that for every 1° increase in knee flexion angle, there was an approximately 9% decrease in the odds of being evaluated with a 3–bone bruise pattern versus a 4–bone bruise pattern. Similarly, for every 1° increase in internal tibial rotation, there was a 17% decrease in the odds of being evaluated with the 3–bone bruise pattern as compared with the 4–bone bruise pattern. No significant relationships were detected between valgus or anterior tibial translation and bone bruise pattern.

DISCUSSION

The purpose of this study was to compare the predicted position of noncontact ACL injury in individuals evaluated with the 2 most common patterns of bone bruising (LTP/MTP/LFC and LTP/MTP/LFC/MFC)[30] using 3D modeling and numerical optimization techniques.[29,39] Importantly, previous work has suggested that different patterns of bone bruising may result from different noncontact ACL injury mechanisms.[24,40,48] However, although some differences were observed, the present study demonstrates that the position of the knee near the time of ACL rupture was similar in patients evaluated with these 2 patterns of bone bruises. Specifically, this study indicates that the knee was in a straight position in both the sagittal (<20° of flexion) and coronal (<10° of valgus) planes and underwent large anterior tibial translation (>20 mm) near the time of ACL rupture for both bone bruise patterns.

The predicted position of injury in patients with 3 bone bruises involved significantly less flexion as compared with those with 4 bone bruises. Nonetheless, the predictions using both 3 and 4 bone bruises indicated that the knee was positioned close to full extension (<20°) near the time of injury. Previous in vivo studies have suggested that ACL tension increases with decreasing flexion angle,[3,17] which may increase the ligament's vulnerability to injury. For example, studies of healthy participants that have combined static or high-speed biplanar radiography with MRI have demonstrated that ACL length and strain increase with decreasing knee flexion during lunging,[17] walking,[19] and jumping.[15] These studies are consistent with other in vivo studies that have combined motion capture, biplanar radiography, and MRI[50,51] or have used arthroscopically implanted strain transducers[3,4,10,23] to demonstrate that ACL strain increases with decreasing flexion angle. The findings regarding the relationship between ACL strain and knee flexion can be explained in part by the orientation of the line of action of the patellar tendon with respect to the tibia. Specifically, when the knee is extended, the patellar tendon is oriented to apply an anteriorly directed shear force to the proximal tibia.[13,17,18,20] As the flexion angle increases, the patellar tendon applies a posteriorly directed shear force on the tibia. Together with these investigations of in vivo ACL strain as a function of knee flexion angle,[15,17-19] the findings of the present study suggest that landing on an extended knee is a high-risk position for ACL rupture for both those with 3 and 4 bone bruises.

A primary function of the ACL is to resist anterior translation of the tibia relative to the femur.[22] Consistent with this biomechanical role, the results of this study demonstrate large anterior tibial translations (24-26 mm) near the time of injury in both the 3– and 4–bone bruise groups. Interestingly, a cadaveric model investigating quadriceps loading on an extended knee as a potential noncontact ACL injury mechanism measured average anterior translations of >20 mm when the ACL was completely ruptured.[14] Together with findings that quadriceps muscle activation can induce large compressive and anteriorly directed loads to the ACL when the knee is positioned near extension,[14,20,54] these findings further support the notion that the ACL may be at high risk of failure via excessive anterior tibial translation when landing on an extended knee.

Recent studies[25,46] have suggested that bruising results from impact between the femur and tibia with the knee in a more flexed position (36°-46°). However, in the present study we predicted that the knee was positioned closer to extension (<20°) near the time of injury. These differences may be due to variations in the techniques used to identify the extent and epicenter of the bone bruises on the MRI scans. In this study, we cross-referenced the T2- with the T1-weighted images to fully characterize the locations of the bone bruises. Specifically, we identified focal points of cortical bone disruption and infraction on the T1-weighted MRI scans in addition to the edema on the T2-weighted MRI scans. When compared with the T1-weighted images, the edema seen on the T2-weighted images extends significantly inferior to the point of cortical abnormality on the tibial plateau (Figure 2). When this difference is not accounted for, the perceived epicenter of the tibial impaction zone may be shifted. We hypothesize that this would result in overestimates of knee flexion in the predicted position of injury.

To test this hypothesis, we simulated the effect of shifting the perceived epicenter of impact on the predicted position of injury in 5 randomly selected patients from the 3- and 4-bruise groups. In this simulation, the surfaces of the medial and lateral tibial plateau bone bruises were extended beyond the articular surface onto the posterior vertical portion of the tibia. We then compared the position of injury determined by this shifted position with that identified by the original bone bruise locations. We found that extending the bone bruises inferiorly significantly increased the predicted flexion in both groups to 30° (P = .006; paired t test), which is similar to the 36° of knee flexion reported previously.[46] We believe that the method of characterizing bone bruise geometries outlined in the present study (specifically, cross-referencing the T2-weighted images with T1-weighted images) is more appropriate than only using the T2-weighted images and extending the bruises to the posterior aspect of the tibial plateau, which is far below the articular surface. Furthermore, the extended knee position predicted from the bone bruises characterized by referencing both the T1- and T2-weighted images is consistent with in vivo studies that have demonstrated increased ACL strain with knee extension.[15,17]

We observed a statistically significant difference in internal rotation between groups, with more internal rotation predicted near the time of injury in those with 4 bone bruises relative to those with 3 bone bruises. As previous work has noted that the tibia rotates internally with increasing flexion,[32] the increased internal rotation in those with 4 bone bruises is likely related to the increased flexion angle in the predicted position of injury in this group (Figure 3, Table 2). Additionally, there are varied data presented in the literature regarding the role of internal rotation in ACL injury. For example, studies that have analyzed video footage of ACL injuries have implicated both internal and external rotation of the tibia in the mechanism of injury.[31,33,38] On the other hand, in vivo studies using strain gauges attached to the anteromedial bundle of the ACL have suggested that internal rotation increases ACL loading.[23] Thus, even though the role of internal rotation in ACL injury remains to be investigated further, these data suggest the injury occurred with <10° of internal rotation in both groups.

Importantly, we did not identify a difference in valgus angle near the time of injury between bone bruise groups, and the predicted position of injury was associated with valgus angles of 7° to 8° in both groups. In contrast, analyses of 2-dimensional videos of ACL injuries have identified changes in valgus angle of approximately 40° during injury scenarios.[6,27] However, it is important to note that valgus angles measured from videos may be influenced by the perspective of the camera angle relative to the participant. Specifically, it has been shown that valgus angle measurements vary substantially when obtained from viewing angles that are not aligned with the participant's anatomic coordinate system[16] and therefore may not be indicative of true anatomic alignment.[33] Furthermore, although ACL rupture has been estimated to occur within 50 ms of initial ground contact,[33] the large valgus angles observed in video analyses[6,27] occurred later than this estimated time of ACL rupture. Moreover, because it remains unclear exactly when the ACL has ruptured in injury video footage, it is also possible that these valgus angles are not a cause of injury but rather a consequence of the knee buckling after the ligament has torn.[37,38] Studies of in vivo ACL strain as a function of knee position may also provide insight into the role of valgus in ACL injury. A previous in vivo imaging study demonstrated that ACL tension decreased when the knee was positioned to simulate a valgus collapse as compared with an extended position.[52] This finding is supported by strain gauge studies that found no significant influence of valgus moment on ACL strains.[23] Together, these findings suggest that valgus angles may not play as significant a role in the noncontact ACL injury mechanism as has been previously suggested.

It is important to note that predicting the position of injury based on the locations of bone bruises assumes they are formed during impact between the tibia and femur near the time of ACL injury. This assumption is in line with previous work that has contended that bone bruises provide information on the mechanism of ACL injury.[29,34,48,53,56] However, based on the large anterior tibial translations measured in the predicted position of injury, it is possible that the ACL has already torn before the impact resulting in the bone bruises.[25] As such, it is possible that these predictions are overestimates of the motion of the knee at the time of ACL rupture. Additionally, bone bruises may fade with time from injury.[8]

Therefore, in this study, we included patients with imaging obtained within 6 weeks of injury, and we observed no difference in time from injury between the groups with 3 and 4 bone bruises.

It must be noted that the algorithm used to predict the position of the knee near the time of injury in patients evaluated with 3 bone bruises assumes that the MTP bruise was formed by contact with its nearest point on the MFC. To test this assumption, we compared the predicted position of injury using 4 and 3 bone bruises within the same patients. In this analysis, we did not observe a difference in the predicted position of injury when the MFC bruise was removed from patients with 4 bone bruises. Future work may assess differences in predicted knee position when adopting different assumptions on the origin of the MFC bruise. Furthermore, in the present study, we predicted the position of injury based on bone bruise location for 2 specific patterns of bone bruises (LTP/MTP/LFC and LTP/MTP/LFC/MFC). Among a cohort of 136 patients with noncontact ACL injuries, these 2 bone bruising patterns comprised 59% of the patient cohort.[30] However, predictions from additional patterns of bone bruising remain to be investigated further.

CONCLUSION

The LTP/MTP/LFC bone bruise pattern is commonly observed in MRI scans of patients with noncontact ACL rupture.[30] Building on previous techniques,[29,39] we predicted the position of the knee near the time of ACL rupture in patients displaying this pattern of 3 bone bruises. This analysis predicted that the knee was positioned in extension (flexion angle, <20°), with minimal valgus angulation (<0°) and internal rotation (<10°), and underwent significant anterior translation (>20 mm) near the time of injury. This knee position is similar to that reported for ACL injury cases with 4 bone bruises.[29,39] Therefore, the findings of this study support the hypothesis that landing on a straight knee with subsequent anterior tibial translation is a potential mechanism for noncontact ACL injury in patients evaluated with these patterns of both 3 and 4 bone bruises.

ACKNOWLEDGMENT

We acknowledge Don S. Kirkendall for his editorial assistance. We also acknowledge the late Dr William E. Garrett for his contributions to this series of work.

ORCID iD

Kwadwo Owusu-Akyaw https://orcid.org/0000-0002-3661-8844

REFERENCES

1. Andriacchi TP, Briant PL, Bevill SL, Koo S. Rotational changes at the knee after ACL injury cause cartilage thinning. *Clin Orthop Relat Res*. 2006;442:39-44.
2. Beck NA, Lawrence JTR, Nordin JD, DeFor TA, Tompkins M. ACL tears in school-aged children and adolescents over 20 years. *Pediatrics*. 2017;139(3):e20161877.
3. Beynnon B, Howe J, Pope MH, Johnson RJ, Fleming B. The measurement of anterior cruciate ligament strain in vivo. *Int Orthop*. 1992;16(1):1-12.
4. Beynnon BD, Johnson RJ, Fleming BC, Stankewich CJ, Renstrom PA, Nichols CE. The strain behavior of the anterior cruciate ligament during squatting and active flexion-extension: a comparison of an open and a closed kinetic chain exercise. *Am J Sports Med*. 1997;25(6):823-829.
5. Boden BP, Griffin LY, Garrett WE Jr. Etiology and prevention of noncontact ACL injury. *Physician Sportsmed*. 2000;28(4):53-60.
6. Boden BP, Torg JS, Knowles SB, Hewett TE. Video analysis of anterior cruciate ligament injury: abnormalities in hip and ankle kinematics. *Am J Sports Med*. 2009;37(2):252-259.
7. Bram JT, Magee LC, Mehta NN, Patel NM, Ganley TJ. Anterior cruciate ligament injury incidence in adolescent athletes: a systematic review and meta-analysis. *Am J Sports Med*. 2020;49(7):1962-1972.
8. Bretlau T, Tuxoe J, Larsen L, Jorgensen U, Thomsen HS, Lausten GS. Bone bruise in the acutely injured knee. *Knee Surg Sports Traumatol Arthrosc*. 2002;10(2):96-101.
9. Carlson VR, Sheehan FT, Boden BP. Video analysis of anterior cruciate ligament (ACL) injuries: a systematic review. *JBJS Rev*. 2016;4(11):e5.
10. Cerulli G, Benoit DL, Lamontagne M, Caraffa A, Liti A. In vivo anterior cruciate ligament strain behaviour during a rapid deceleration movement: case report. *Knee Surg Sports Traumatol Arthrosc*. 2003;11(5):307-311.
11. Chaudhari AM, Andriacchi TP. The mechanical consequences of dynamic frontal plane limb alignment for non-contact ACL injury. *J Biomech*. 2006;39(2):330-338.
12. DeAngelis JP, Spindler KP. Traumatic bone bruises in the athlete's knee. *Sports Health*. 2010;2(5):398-402.
13. Defrate LE, Nha KW, Papannagari R, Moses JM, Gill TJ, Li G. The biomechanical function of the patellar tendon during in-vivo weight-bearing flexion. *J Biomech*. 2007;40(8):1716-1722.
14. DeMorat G, Weinhold P, Blackburn T, Chudik S, Garrett W. Aggressive quadriceps loading can induce noncontact anterior cruciate ligament injury. *Am J Sports Med*. 2004;32(2):477-483.
15. Englander ZA, Baldwin EL III, Smith WAR, Spritzer CE, Garrett WE Jr, DeFrate LE. In vivo anterior cruciate ligament deformation during a -single-legged jump measured by magnetic resonance imaging and high-speed biplanar radiography. *Am J Sports Med*. 2019;47(13):3166-3172.
16. Englander ZA, Cutcliffe HC, Utturkar GM, Garrett WE, Spritzer CE, DeFrate LE. A comparison of knee abduction angles measured by a 3D anatomic coordinate system versus videographic analysis: implications for anterior cruciate ligament injury. *Orthop J Sports Med*. 2019;7(1):2325967118819831.
17. Englander ZA, Cutcliffe HC, Utturkar GM, et al. In vivo assessment of the interaction of patellar tendon tibial shaft angle and anterior cruciate ligament elongation during flexion. *J Biomech*. 2019;90:123-127.
18. Englander ZA, Foody JN, Cutcliffe HC, Wittstein JR, Spritzer CE, DeFrate LE. Use of a novel multimodal imaging technique to model in vivo quadriceps force and ACL strain during dynamic activity. *Am J Sports Med*. 2022;50(10):2688-2697.
19. Englander ZA, Garrett WE, Spritzer CE, DeFrate LE. In vivo attachment site to attachment site length and strain of the ACL and its bundles during the full gait cycle measured by MRI and high-speed biplanar radiography. *J Biomech*. 2020;98:109443.
20. Englander ZA, Lau BC, Wittstein JR, Goode AP, DeFrate LE. Patellar tendon orientation and strain are predictors of ACL strain in vivo during a single-leg jump. *Orthop J Sports Med*. 2021;9(3):23259671 21991054.
21. Fithian DC, Paxton LW, Goltz DH. Fate of the anterior cruciate ligament-injured knee. *Orthop Clin North Am*. 2002;33(4):621-636.
22. Fleming BC, Beynnon BD. In vivo measurement of ligament/tendon strains and forces: a review. *Ann Biomed Eng*. 2004;32(3):318-328.

23. Fleming BC, Renstrom PA, Beynnon BD, et al. The effect of weight-bearing and external loading on anterior cruciate ligament strain. *J Biomech*. 2001;34(2):163-170.
24. Graf BK, Cook DA, De Smet AA, Keene JS. ''Bone bruises'' on magnetic resonance imaging evaluation of anterior cruciate ligament injuries. *Am J Sports Med*. 1993;21(2):220-223.
25. Grassi A, Agostinone P, Di Paolo S, et al. Knee position at the moment of bone bruise could reflect the late phase of non-contact anterior cruciate ligament injury rather than the mechanisms leading to ligament failure. *Knee Surg Sports Traumatol Arthrosc*. 2021;29(12):4138-4145.
26. Hewett TE, Lynch TR, Myer GD, Ford KR, Gwin RC, Heidt RS Jr. Multiple risk factors related to familial predisposition to anterior cruciate ligament injury: fraternal twin sisters with anterior cruciate ligament ruptures. *Br J Sports Med*. 2010;44(12):848-855.
27. Hewett TE, Torg JS, Boden BP. Video analysis of trunk and knee motion during non-contact anterior cruciate ligament injury in female athletes: lateral trunk and knee abduction motion are combined components of the injury mechanism. *Br J Sports Med*. 2009;43(6):417-422.
28. Kibler WB. *OKU Orthopaedic Knowledge Update: Sports Medicine 4*. American Academy of Orthopaedic Surgeons; 2009.
29. Kim SY, Spritzer CE, Utturkar GM, Toth AP, Garrett WE, DeFrate LE. Knee kinematics during noncontact anterior cruciate ligament injury as determined from bone bruise location. *Am J Sports Med*. 2015;43(10):2515-2521.
30. Kim-Wang SY, Scribani MB, Whiteside MB, DeFrate LE, Lassiter TE, Wittstein JR. Distribution of bone contusion patterns in acute noncontact ACL torn knees. *Am J Sports Med*. 2021;49(2):404-409.
31. Koga H, Nakamae A, Shima Y, et al. Mechanisms for noncontact anterior cruciate ligament injuries: knee joint kinematics in 10 injury situations from female team handball and basketball. *Am J Sports Med*. 2010;38(11):2218-2225.
32. Kozanek M, Hosseini A, Liu F, et al. Tibiofemoral kinematics and condylar motion during the stance phase of gait. *J Biomech*. 2009;42(12):1877-1884.
33. Krosshaug T, Nakamae A, Boden BP, et al. Mechanisms of anterior cruciate ligament injury in basketball: video analysis of 39 cases. *Am J Sports Med*. 2007;35(3):359-367.
34. Lattermann C, Jacobs CA, Reinke EK, et al. Are bone bruise characteristics and articular cartilage pathology associated with inferior outcomes 2 and 6 years after anterior cruciate ligament reconstruction? *Cartilage*. 2017;8(2):139-145.
35. Lynch TC, Crues JV III, Morgan FW, Sheehan WE, Harter LP, Ryu R. Bone abnormalities of the knee: prevalence and significance at MR imaging. *Radiology*. 1989;171(3):761-766.
36. Mall NA, Chalmers PN, Moric M, et al. Incidence and trends of anterior cruciate ligament reconstruction in the United States. *Am J Sports Med*. 2014;42(10):2363-2370.
37. Meyer EG, Haut RC. Anterior cruciate ligament injury induced by internal tibial torsion or tibiofemoral compression. *J Biomech*. 2008;41(16):3377-3383.
38. Olsen OE, Myklebust G, Engebretsen L, Bahr R. Injury mechanisms for anterior cruciate ligament injuries in team handball: a systematic video analysis. *Am J Sports Med*. 2004;32(4):1002-1012.
39. Owusu-Akyaw KA, Kim SY, Spritzer CE, et al. Determination of the position of the knee at the time of an anterior cruciate ligament rupture for male versus female patients by an analysis of bone bruises. *Am J Sports Med*. 2018;46(7):1559-1565.
40. Quatman CE, Hewett TE. The anterior cruciate ligament injury controversy: is ''valgus collapse'' a sex-specific mechanism? *Br J Sports Med*. 2009;43(5):328-335.
41. Renstrom P, Ljungqvist A, Arendt E, et al. Non-contact ACL injuries in female athletes: an International Olympic Committee current concepts statement. *Br J Sports Med*. 2008;42(6):394-412.
42. Roos H, Adalberth T, Dahlberg L, Lohmander LS. Osteoarthritis of the knee after injury to the anterior cruciate ligament or meniscus: the influence of time and age. *Osteoarthritis Cartilage*. 1995;3(4):261-267.
43. Rosen MA, Jackson DW, Berger PE. Occult osseous lesions documented by magnetic resonance imaging associated with anterior cruciate ligament ruptures. *Arthroscopy*. 1991;7(1):45-51.
44. Sadoghi P, von Keudell A, Vavken P. Effectiveness of anterior cruciate ligament injury prevention training programs. *J Bone Joint Surg Am*. 2012;94(9):769-776.
45. Sanders TG, Medynski MA, Feller JF, Lawhorn KW. Bone contusion patterns of the knee at MR imaging: footprint of the mechanism of injury. *Radiographics*. 2000;20:S135-S151.
46. Shi H, Ding L, Ren S, et al. Prediction of knee kinematics at the time of noncontact anterior cruciate ligament injuries based on the bone bruises. *Ann Biomed Eng*. 2020;49(1):162-170.
47. Shin CS, Chaudhari AM, Andriacchi TP. The effect of isolated valgus moments on ACL strain during single-leg landing: a simulation study. *J Biomech*. 2009;42(3):280-285.
48. Speer KP, Spritzer CE, Bassett FH III, Feagin JA Jr, Garrett WE Jr. Osseous injury associated with acute tears of the anterior cruciate ligament. *Am J Sports Med*. 1992;20(4):382-389.
49. Stevenson JH, Beattie CS, Schwartz JB, Busconi BD. Assessing the effectiveness of neuromuscular training programs in reducing the incidence of anterior cruciate ligament injuries in female athletes: a systematic review. *Am J Sports Med*. 2015;43(2):482-490.
50. Taylor K, Terry M, Utturkar G, et al. Measurement of in vivo anterior cruciate ligament strain during dynamic jump landing. *J Biomech*. 2011;44(3):365-371.
51. Taylor KA, Cutcliffe HC, Queen RM, et al. In vivo measurement of ACL length and relative strain during walking. *J Biomech*. 2013;46(3):478-483.
52. Utturkar GM, Irribarra LA, Taylor KA, et al. The effects of a valgus collapse knee position on in vivo ACL elongation. *Ann Biomed Eng*. 2013;41(1):123-130.
53. Viskontas DG, Giuffre BM, Duggal N, Graham D, Parker D, Coolican M. Bone bruises associated with ACL rupture: correlation with injury mechanism. *Am J Sports Med*. 2008;36(5):927-933.
54. Wall SJ, Rose DM, Sutter EG, Belkoff SM, Boden BP. The role of axial compressive and quadriceps forces in noncontact anterior cruciate ligament injury: a cadaveric study. *Am J Sports Med*. 2012;40(3):568-573.
55. Zbrojkiewicz D, Vertullo C, Grayson JE. Increasing rates of anterior cruciate ligament reconstruction in young Australians, 2000-2015. *Med J Aust*. 2018;208(8):354-358.
56. Zhang L, Hacke JD, Garrett WE, Liu H, Yu B. Bone bruises associated with anterior cruciate ligament injury as indicators of injury mechanism: a systematic review. *Sports Med*. 2019;49(3):453-462.