Unique Anatomic Feature of the Posterior Cruciate Ligament in Knees Associated With Osteochondritis Dissecans

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Background: Osteochondritis dissecans (OCD) of the knee is a disorder in juveniles and young adults; however, its etiology still remains unclear. For OCD at the medial femoral condyle (MFC), it is sometimes observed that the lesion has a connection with fibers of the posterior cruciate ligament (PCL). Although this could be important information related to the etiology of MFC OCD, there is no report examining an association between the MFC OCD and the PCL anatomy.

Purpose: To investigate the anatomic features of knees associated with MFC OCD, focusing especially on the femoral attachment of the PCL, and to compare them with knees associated with lateral femoral condyle (LFC) OCD and non-OCD lesions.

Study Design: Case-control study; Level of evidence, 3.

Methods: We retrospectively reviewed 39 patients (46 knees) with OCD lesions who had undergone surgical treatment. Using magnetic resonance imaging (MRI) scans, the PCL attachment at the lateral wall of the MFC was measured on the coronal sections, and the knee flexion angle was also measured on the sagittal sections. As with non-OCD knees, we reviewed and analyzed 25 knees with anterior cruciate ligament (ACL) injuries and 16 knees with meniscal injuries.

Results: MRIs revealed that the femoral PCL footprint was located in a significantly more distal position in the patients with MFC OCD compared with patients with LFC OCD and ACL and meniscal injuries. There was no significant difference in knee flexion angle among the 4 groups.

Conclusion: The PCL in patients with MFC OCD attached more distally at the lateral aspect of the MFC compared with knees with LFC OCD and ACL and meniscal injuries.

Keywords: osteochondritis dissecans; posterior cruciate ligament; femoral footprint; anatomic feature

In 1870, Paget first described loose bodies within a joint.[23] Following this report, König considered this condition resulted from either acute osteochondral fracture, minor trauma that leads into osteonecrosis and fragmentation, or spontaneous development without remarkable evidence of trauma. In 1888, König[18] defined this nontraumatic condition as osteochondritis dissecans (OCD). From the accumulation of knowledge over the past century, OCD has become recognized as an acquired lesion of subchondral bone leading to articular cartilage degeneration.[10] However, even in the 21st century, the etiology and natural history of OCD still remain elusive.

According to the histological evaluation of the lesion, osteonecrosis led by poor vascular supply has commonly been described as the cause of this problem. However, it is very difficult to determine whether this histological condition is a primary or secondary condition.[25] Also, limited research has been reported demonstrating the histological findings related to osteonecrosis.[5,20] Currently, repetitive microtrauma has become a more widely accepted etiology

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of the OCD in its development. The high incidence of this disorder in the highly active athletic population supports this concept.[13,30]

In Western countries, the most common site of OCD lesion in the knee is the medial femoral condyle (MFC).[4,14] Several hypotheses, such as microtrauma, increased external torsion of the tibia, and axis deviation in the frontal plane, have been proposed as the cause of OCD at the MFC.[3,16,24] However, it is thought that multiple factors are involved in the formation of OCD in the knee, and we have not achieved a conclusive answer for this problem.

An interesting finding of MFC OCD is that orthopaedic surgeons have empirically known that the lesion is located very close to the femoral posterior cruciate ligament (PCL) footprint from arthroscopic findings (Figure 1A). Also, Smillie[26] described the loose body lying in the intercondylar notch attached by a few strands of the PCL as frequently occurring at the classical (ie, the MFC OCD) lesion. These facts lead us to the hypothesis that the PCL might have some role for the induction of repetitive stress and lesion development at the MFC OCD. However, as far as we know, there have been no reports proposing an association between the MFC OCD and the PCL.

In this study, we hypothesized that the PCL could have unique anatomy in patients with medial OCD. The aim of this study was to evaluate the femoral PCL attachment in patients with MFC OCD lesion compared with patients with lateral femoral condyle (LFC) OCD and non-OCD lesions and to examine the possible association between the MFC OCD and the PCL.

METHODS

Patient Population

Institutional review board approval was obtained for this retrospective case-control study. We reviewed 39 patients (47 knees) with OCD lesions who were surgically treated at our hospital from September 2010 to July 2015. The lesion location of the OCD in the knee was classified according to the Aichroth classification.[1] We included patients who had a single lesion at the MFC or LFC even if they had an additional OCD lesion in the trochlear groove or the patella. Patients with 2 or more lesions at the medial and lateral condyles and osteoarthritic change defined according to a consensus of Kellgren-Lawrence (K-L) grade $\geq$2 were excluded. Two control groups, matched for age but not sex, were also evaluated. One group consisted of patients who had sustained an ACL rupture; the other group was composed of patients who had sustained a medial or lateral meniscal tear.

Image Analysis Using Magnetic Resonance Imaging Scans

To evaluate anatomic features of the femoral PCL footprint in each patient from 4 groups, the preoperative coronal and sagittal magnetic resonance imaging (MRI) slices were reviewed retrospectively. Although the MRI images were acquired in a varied fashion, as different protocols were used depending on the institute where each patient initially visited, a minimum magnet strength of 1.5 T was used in all MRI studies. T2-weighted sequential slices were used for the measurement of indices described below.

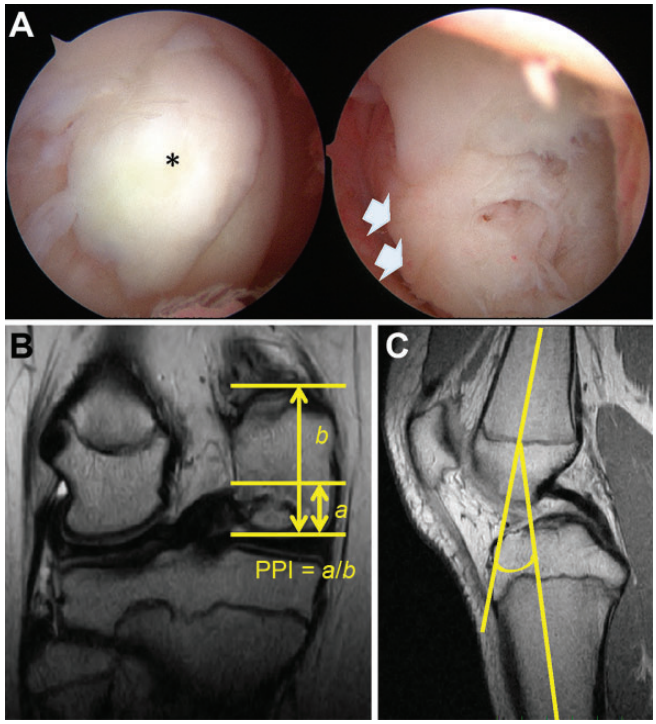

Figure 1. (A) Arthroscopic images of a 16-year-old boy with osteochondritis dissecans of the medial femoral condyle (MFC) in the left knee. Shown is the unstable osteochondritis dissecans lesion (asterisk); the attachment of the posterior cruciate ligament (PCL) fibers on the lesion can be seen (arrows). (B) Measurement of the PCL position index (PPI): The distance from the distal margin of the MFC to the proximal point of the PCL attachment at the lateral aspect of the MFC was measured (distance *a*). Also, using the same line of distal margin of the MFC, the length of the MFC was measured from the line of proximal margin of the MFC (distance *b*). The PPI was calculated as the ratio of *a* to *b*. (C) Measurements of the flexion angle. Using a sagittal section, the angle between the lines from the femoral and tibial shaft was measured.

PCL Position Index. For evaluation of the PCL footprint at the lateral surface of MFC and its variation on the coronal sections, the distance between the lines that were drawn at the articular margin of the distal end of the MFC and at the most proximal point of the femoral PCL attachment at the lateral surface of MFC was measured from the best coronal slice that described the femoral PCL attachment and the articular margin of the MFC (Figure 1B, distance *a*). In addition, the length of the MFC was measured using the same line at the articular margin of the distal end and the most proximal margin of the MFC (Figure 1B, distance *b*). Then, the PCL position index (PPI) was produced from the ratio of *a* to *b*.

TABLE 1
Patient Demographics[a]

	MFC OCD (n = 26)	LFC OCD (n = 20)	ACL (n = 25)	MM/LM (n = 16)
Sex, male/female, n	19/3	14/3	10/15	9/7
Age, y, mean ± SD (range)	15.7 ± 6.1 (8-36)	12.9 ± 3.6 (8-21)	17.2 ± 2.9 (11-24)	14.9 ± 1.7 (13-18)
Physis, open/closed, n	17/9	16/4	7/18	9/7
Lesion location, n (%)[b]	Classical: 11 (42.3) Extended classical: 12 (46.2) Inferocentral: 3 (11.5)	Inferocentral: 16 (80.0) Anterior: 4 (20.0)	—	—

[a]ACL, anterior cruciate ligament; LFC, lateral femoral condyle; LM, lateral meniscus; MFC, medial femoral condyle; MM, medial meniscus; OCD, osteochondritis dissecans.
[b]According to Aichroth classification.[1]

Flexion Angle. We considered that the knee flexion angle (FA) could affect the view of the coronal section and the measurement of the femoral PCL attachment using the geometry of the MFC. Therefore, the knee FA was measured and compared between groups on sagittal sections. The lines along the femoral and tibial shafts were drawn and its angle was measured (Figure 1C).

Statistical Analysis

All values are expressed as the mean ± standard deviation. The Kruskal-Wallis test was carried out to compare the PPI and FA in the 4 groups. The Mann-Whitney *U* test was used to compare PPI and FA values between patients with open and closed physes in the MFC and LFC OCD groups and classical lesion and extended classical lesion in the MFC OCD group.

Two orthopaedic surgeons (M.I., M.Y.) performed the PPI and FA measurements in a randomized order, twice by each rater, with at least 30 days' duration between the measurements. Each rater was blinded to the other rater's measurements and their previous measurement. Then, intra- and interobserver reliabilities were analyzed using intraclass correlation coefficients (ICCs). The ICC values were interpreted as follows: <0.40, poor agreement; 0.40 < ICC < 0.75, fair to good agreement; >0.75, excellent agreement.

All statistical analysis was conducted using GraphPad PRISM (GraphPad Software Inc). $P < .05$ was regarded as statistically significant.

RESULTS

From this review, 22 patients with 26 OCD lesions located at the MFC were identified and were classified as the MFC OCD group (19 males and 3 females; mean age, 15.7 years; range, 8-36 years; classical lesion in 11 knees, extended classical lesion in 12 knees, and inferocentral lesion in 3 knees). Seventeen patients with 20 OCD lesions at the LFC were presented and were classified as the LFC OCD group (14 males and 3 females; mean age, 12.9 years; range, 8-21 years; inferocentral lesion in 16 knees and anterior lesion in 4 knees). One patient who had MFC OCD was excluded because of osteoarthritic change demonstrating K-L grade ≥2. To compare the population without OCD lesion, as a control, 25 knees of 25 patients with ACL injury and 16 knees of 16 patients with medial or lateral meniscus (MM/LM) injuries were reviewed as well and classified as the ACL group (10 males and 15 females; mean age, 17.2 years; range, 11-24 years) and the MM/LM group (9 males and 7 females; mean age, 14.9 years; range, 13-18 years). Demographic information is provided in Table 1.

TABLE 2
PPI and FA Measurements: All Groups[a]

	MFC OCD (n = 26)	LFC OCD (n = 20)	ACL (n = 25)	MM/LM (n = 16)
PPI, %	**0.413 ± 0.041**	0.473 ± 0.034	0.490 ± 0.034	0.491 ± 0.027
FA, deg	12.3 ± 6.0	13.9 ± 7.5	13.4 ± 4.8	12.8 ± 6.1

[a]Values are reported as mean ± SD. Bolded text indicates statistical significance. ACL, anterior cruciate ligament; FA, flexion angle; LFC, lateral femoral condyle; LM, lateral meniscus; MFC, medial femoral condyle; MM, medial meniscus; OCD, osteochondritis dissecans; PPI, posterior cruciate ligament position index.

In the MFC OCD group, the PPI was significantly lower; that is, the PCL footprint at the lateral surface of MFC was located at a significantly more distal position compared with the LFC OCD, ACL, and MM/LM groups (MFC OCD, 0.413% ± 0.041%; LFC OCD, 0.473% ± 0.034%; ACL, 0.490% ± 0.034%; MM/LM, 0.491% ± 0.027%) (Table 2 and Figure 2). On the other hand, there was no significant difference in FA among the 4 groups (MFC OCD, 12.3° ± 6.0°; LFC OCD, 13.9° ± 7.5°; ACL, 13.4° ± 4.8°; MM/LM, 12.8° ± 6.1°) (Table 2 and Figure 3).

To evaluate the influence of skeletal maturity on PPI and FA values, these measurements were first compared between all patients with open and closed physes. An open physis was confirmed in 49 knees, and 38 knees presented a closed physis (Table 1). The comparison revealed no significant difference in either the PPI or FA between these patient groups (PPI: 0.456% ± 0.052% vs 0.473% ± 0.041%, $P = .204$; FA: 12.8° ± 6.8° vs 13.7° ± 5.1°, $P = .235$; open vs closed, respectively) (Table 3). Second, we compared the PPI and FA among the OCD patients with open and closed physes. Thirty-three knees with an open physis and 13 knees with a closed physis were identified in

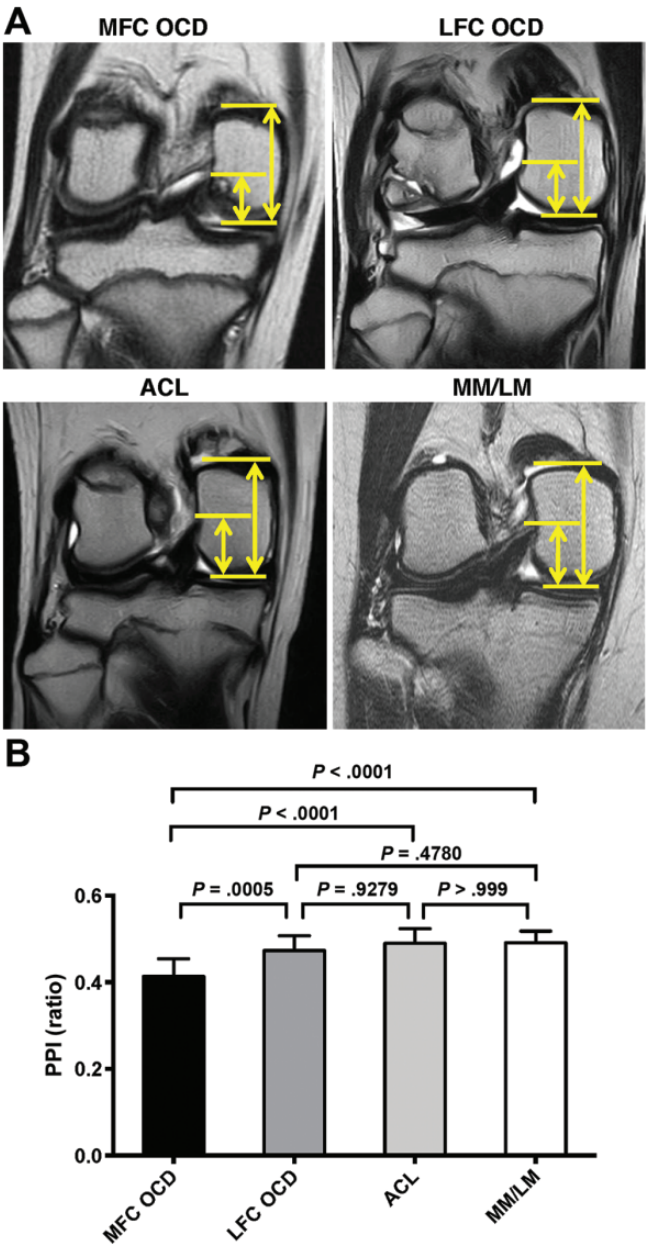

Figure 2. (A) Representative coronal sections from each group. (B) Comparison of the PCL position index (PPI) among the MFC OCD, LFC OCD, ACL, and MM/LM groups. The value of PPI in the MFC OCD group was significantly lower compared with the LFC OCD, ACL, and MM/LM groups. There was no significant difference between the LFC OCD, ACL, and MM/LM groups. Bars represent median ± standard deviation. ACL, anterior cruciate ligament; LFC, lateral femoral condyle; LM, lateral meniscus; MFC, medial femoral condyle; MM, medial meniscus; OCD, osteochondritis dissecans; PCL, posterior cruciate ligament.

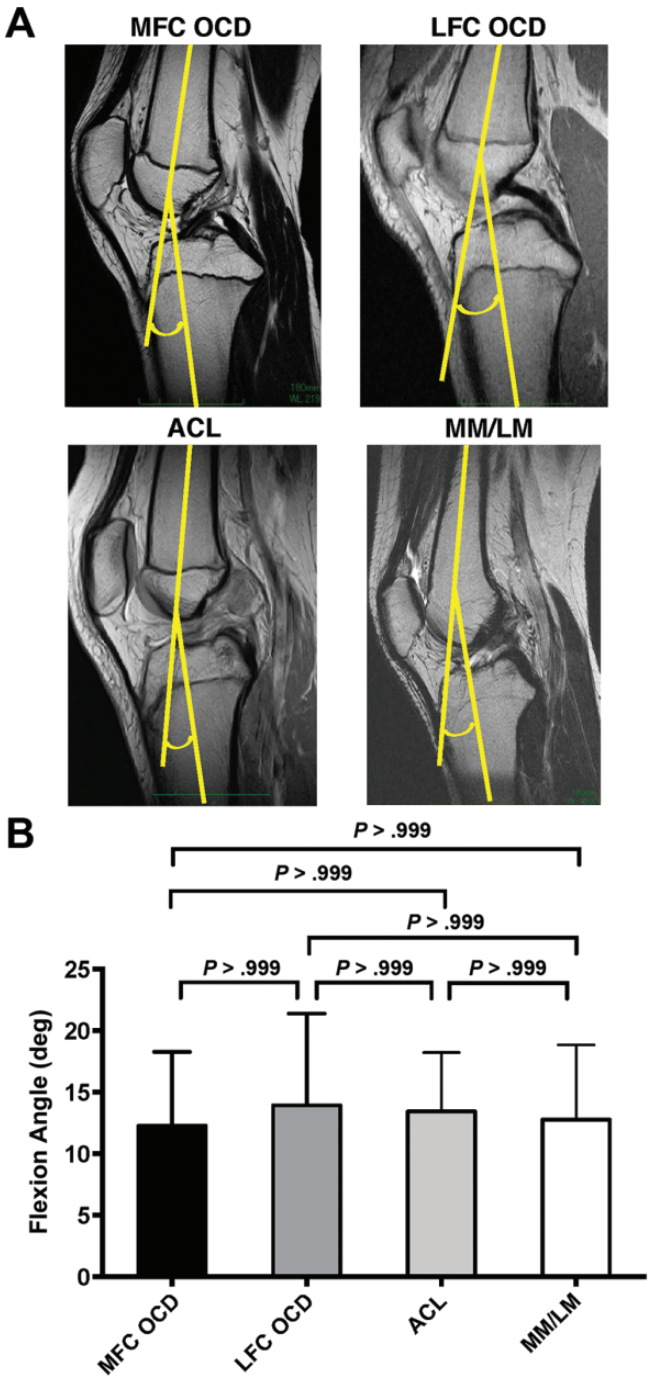

Figure 3. (A) Representative sagittal sections from each group. (B) Comparison of the flexion angle (FA) among the MFC OCD, LFC OCD, ACL, and MM/LM groups. There was no significant difference among the groups. Bars represent median ± standard deviation. ACL, anterior cruciate ligament; LFC, lateral femoral condyle; LM, lateral meniscus; MFC, medial femoral condyle; MM, medial meniscus; OCD, osteochondritis dissecans; PCL, posterior cruciate ligament.

the OCD groups. No significant difference in PPI or FA could be confirmed (PPI: 0.435% ± 0.047% vs 0.451% ± 0.049%, $P = .513$; FA: 13.8° ± 7.1° vs 10.9° ± 4.7°, $P = .461$; open vs closed, respectively) (Table 3). Third, the PPI and FA in skeletally immature knees within the MFC and LFC OCD groups was analyzed. We identified 17 knees with an open physis in the MFC OCD group and 16 knees

TABLE 3
PPI and Flexion Angle Measurements: Association With Skeletal Maturity[a]

	All Knees			Knees With OCD Lesion			Knees With Open Physis		
	Open Physis (n = 49)	Closed Physis (n = 38)	*P*	Open Physis (n = 33)	Closed Physis (n = 13)	*P*	MFC OCD (n = 17)	LFC OCD (n = 16)	*P*
PPI, %	0.456 ± 0.052	0.473 ± 0.041	.204	0.435 ± 0.047	0.451 ± 0.049	.513	0.401 ± 0.038	0.470 ± 0.024	**<.0001**
FA, deg	12.8 ± 6.8	13.7 ± 5.1	.235	13.8 ± 7.1	10.9 ± 4.7	.461	13.1 ± 6.5	14.5 ± 8.0	.389

[a]Values are reported as mean ± SD. Bolded text indicates statistical significance. FA, flexion angle; LFC, lateral femoral condyle; MFC, medial femoral condyle; OCD, osteochondritis dissecans; PPI, posterior cruciate ligament position index.

TABLE 4
PPI and FA Measurements in the MFC OCD Group: Association With Lesion Location and Skeletal Maturity[a]

	Classical (n = 11)	Extended Classical (n = 12)	*P*
PPI, %	0.435 ± 0.031	0.390 ± 0.037	**.002**
FA, deg	9.8 ± 4.9	13.9 ± 5.7	.148
	Open Physis (n = 17)	Closed Physis (n = 9)	*P*
Lesion location, n (%)[b]	Classical: 3 (17.6) Extended classical: 11 (64.7) Inferocentral: 3 (17.6)	Classical: 8 (88.9) Extended classical: 1 (11.1) Inferocentral: 0 (0.0)	—
PPI, %	0.401 ± 0.038	0.436 ± 0.036	**.039**
FA, deg	13.1 ± 6.5	10.7 ± 4.8	.691

[a]Values are reported as mean ± SD. Bolded text indicates statistical significance. FA, flexion angle; PPI, posterior cruciate ligament position index.
[b]According to Aichroth classification.

in the LFC OCD group and found that the PPI in patients in the MFC OCD group was significantly lower than that in the LFC OCD group. There was no significant difference in FA between groups (PPI: 0.401% ± 0.038% vs 0.470% ± 0.024%, $P < .0001$; FA: 13.1° ± 6.5° vs 14.5° ± 8.0°, $P = .389$; MFC OCD vs LFC OCD, respectively) (Table 3).

In terms of lesion location in the MFC OCD group, we identified 11 knees with classical lesions and 12 knees with extended classical lesions. Interestingly, it was demonstrated that knees with an extended classical lesion (ie, lager lesion) had a significantly lower PPI compared with knees with a classical lesion (extended classical vs classical: 0.031% ± 0.435% vs 0.037% ± 0.390%, $P = .002$) (Table 4). Also, we confirmed there was no significant difference in FA between these 2 conditions (classical vs extended classical: 9.8° ± 4.9° vs 13.9° ± 5.7°, $P = .148$) (Table 4).

Moreover, concerning the relationship with the status of physis in the MFC OCD group, 2 indices were compared between patients with open and closed physes. Open physis was confirmed in 17 knees (classical lesion, 3 knees; extended classical lesion, 11 knees; inferocentral lesion, 3 knees) and 9 knees presented with closed physes (classical lesion, 8 knees; extended classical lesion, 1 knee). The PPI was significantly lower in the open physis group compared with the closed physis group; however, there was no significant difference in FA between groups (PPI: 0.401% ± 0.038% vs 0.436% ± 0.036%, $P = .039$; FA: 13.1° ± 6.5° vs 10.7° ± 4.8°, $P = .691$; open vs closed, respectively) (Table 4).

Intra- and interobserver reliability in measuring PPI and FA was analyzed. For PPI, intraobserver ICCs were excellent (0.84; 95% CI, 0.74-0.90), and interobserver ICCs were fair to good (0.70; 95% CI, 0.48-0.84). For FA, intraobserver ICCs were excellent (0.91; 95% CI, 0.85-0.95), and interobserver ICCs were fair to good (0.66; 95% CI, 0.42-0.81).

DISCUSSION

This study demonstrated a significantly distally located femoral PCL footprint in patients with MFC OCD compared with patients with LFC OCD and non-OCD lesions. To the best of our knowledge, this is the first report to describe the unique anatomy of the PCL in the knee associated with MFC OCD from MRI studies.

Considering OCD lesions at the LFC, it has been proposed that repetitive stress on the LFC by the discoid lateral meniscus or anatomic features of the femur and tibia could play an important role in lesion development.[2,9,15,17,22] In addition, malalignment of the mechanical axis, valgus malalignment, has been positively associated with LFC OCD.[16] On the other hand, in 1933, Fairbank[11] proposed impingement of the tibial spine due to violent rotation inward of the tibia against the inner MFC as the cause of MFC OCD. Supporting this hypothesis, Smillie[27] described the same concept and mentioned the

pressure on the MFC by the ACL. From the standpoint of the mechanical axis of the leg, there was no significant evidence of the specific mechanical axis change accounting for MFC OCD, as shown in LFC OCD.[16] As an anatomic risk factor for MFC OCD, Chow et al[6] recently demonstrated that knees with MFC OCD lesions have significantly narrower intercondylar notch width compared with controls using MRI scans. This study proposed a new approach to investigate anatomic risk factors predisposing to MFC OCD lesions.[6] Thus, few concepts and findings have been introduced to unveil the etiological factor of MFC OCD; however, many things still remain unclear and have to be proven to elucidate the cause of MFC OCD.

The relevance between MFC OCD lesions and PCL was described by Smillie[26] in 1957. Since then, however, no studies have investigated the association between MFC OCD and the PCL as an etiological factor of this entity. Anatomically, the femoral PCL attachment is located on the lateral surface of the MFC in the intercondylar fossa. From a cadaver study it was demonstrated that caudal location of the PCL footprint is bound by the rim of the articular surface of the MFC, close to the MFC OCD lesion.[12] According to this anatomic information, we focused on the PCL, especially its femoral footprint.

In terms of biomechanics of the PCL, it was demonstrated that knee flexion would increase tensile force of the PCL.[21,32] Recently, Xu et al[34] presented a case of femoral osteochondral avulsion fracture of the PCL due to knee hyperflexion in adults, and this led to the concept that the PCL could induce traction force at the lateral wall of MFC with flexion. Of note, the lesion was located at the lateral surface of the MFC, which is similar to common MFC OCD lesions. Although cases of this condition in children are extremely rare,[7] it is not difficult to assume that the PCL could also induce stress at the lateral wall of the MFC in skeletally immature children during a knee-flexed position in sports activity such as sliding and kneeling in soccer or baseball games. As shown in our analysis, it was demonstrated that patients with MFC OCD lesions presented significantly lower PPI. Thus, the caudal location of the PCL is closer to the rim of the articular surface of the MFC in MFC OCD patients. Especially in skeletally immature children under strenuous athletic activity, the PCL might exert abnormal tensile forces at the MFC to develop a pathological condition related to osteochondrosis such as Osgood-Schlatter disease and Sever disease. From our analysis, patients with open physes had larger lesions, ie, extended classical lesions, with significantly lower PPI values compared with patients with closed physes in MFC OCD patients. Hence, we might be able to consider that the lower PPI associated with skeletal immaturity induces excessive stress on the MFC and could be a risk factor of MFC OCD in children.

To evaluate the femoral PCL attachment, we used the coronal sections of MRI scans from routine studies and calculated the PPI with an original method. As this was a retrospective study, MRI images used in this study were not standardized for evaluation of ligament footprint. Of note, it was difficult to evaluate the exact footprint of the PCL on the sagittal view. The intrarater reliability in our study was excellent both for PPI and FA and, on the other hand, the interrater reliability was fair to good for both PPI and FA. These data showed that our method is reliable; however, for more valuable data accumulation, the technology of 3-dimensional computed tomography imaging might be helpful for visualization of the femoral PCL footprint and for quantitative analysis for future studies.[31] To prove the excessive force by the PCL at the MFC, cadaver study altering the femoral footprint and measuring an in situ tensile force by the reconstructed PCL or mathematical model, such as a 3-dimensional finite element model, will be considered for future studies.[19] Once confirmed, a new avenue of management for OCD treatment could be established according to the mechanism of this disorder, such as more effective conservative treatment with braces that control the force generation on the lesion by the PCL to avoid surgical intervention.

Several previous studies have discussed the association between the PCL and MFC OCD lesion. Wilson[33] described a test of physical examination to diagnose juvenile OCD, the so-called "Wilson test." In Wilson's report,[33] the patient associated with MFC OCD had a unique lateral rotation gait. Unfortunately, the Wilson test has been shown to have limited diagnostic value[8,14]; however, his observation about the abnormal gait (laterally rotated tibia in the patient with MFC OCD) suggests an indirect association with the PCL. Inspired by this, Bramer et al[3] used computed tomography analysis to demonstrate that patients with MFC OCD had increased external tibial torsion compared with LFC OCD. It was then proposed that this increased laterally rotated position of the tibia could be a unique clinical finding of the patient with MFC OCD. So far, tibial exotorsion has been considered as one of the causes of MFC OCD.[28,29] Moreover, Markolf et al[21] demonstrated that when the tibia was applied with 100 N of posterior force with 10 N·m of external torque, the mean resultant ligament force in the PCL was dramatically reduced at flexion angles less than 50°, and only 61% of the initial loading was observed even at 90° of flexion. On the other hand, with 10 N·m of internal torque, the ligament force in the PCL was increased through most of the range of flexion.[21] Taken together, from the increased external tibial torsion and the pattern of force generation in the PCL, this laterally rotated tibia could reduce a tensile force on the MFC OCD lesion by the PCL. Also, Wilson[33] stated that this unique presentation disappeared after removal of the loose body in patients with MFC OCD, and this may explain that release of the PCL from a medial lesion could improve symptoms. Combining these facts, it might be suggested that this external tibial rotation in patients with MFC OCD compensates for any pain induced by the PCL.

As with all clinical studies, there are some limitations to this study. First, the patient sample is small. The incidence of OCD in the knee is not high and the patient population analyzed was from only our institute. We are now continuing imaging analysis retro- and prospectively to achieve a more consolidated conclusion. Second, we could only conclude the significant difference in the femoral PCL footprint in patients with MFC OCD compared with those with LFC OCD and non-OCD lesions. Functional analysis to evaluate the ligament properties of the PCL-associated MFC OCD lesion was not performed. Further studies are required. Third, we did not

utilize data from a population with intact knees as a normal sample, although the significantly lower PPI value in the MFC OCD group was compared with the LFC OCD, ACL, and MM/LM injury groups. Volunteers with intact knees should be enrolled for further analysis to standardize our method using MRI scans and to obtain the mean value of the control group.

CONCLUSION

Our study is the first to evaluate the femoral PCL attachment in patients with OCD. We concluded that the femoral PCL footprint is located more distally in the knee with medial OCD compared with the knee with lateral OCD and non-OCD lesions. This unique anatomic feature could be a risk factor of MFC OCD and a key to bringing new insight for more understanding of this enigmatic disorder.

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