The Intact Posterior Cruciate Ligament Not Only Controls Posterior Displacement but Also Maintains the Flexion Gap

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Abstract

Background The PCL is a strong stabilizer of the knee and provides posterior stability to the tibia. However, sagittal alignment of the PCL with the knee at 90° flexion suggests the PCL might play a role not only in posterior stabilization but also in maintaining the flexion gap.

Questions/purposes We determined whether the intact PCL helps maintain the flexion gap.

Methods We examined axial radiographs and gravity sag views of 17 patients with chronic isolated unilateral PCL injury. The flexion gap was defined as the mean value of the medial and lateral distances between the femoral and

tibial bones on the axial radiograph. Increase in the flexion gap and posterior laxity were determined by comparing the patients' injured and contralateral uninjured knees.

Results The flexion gap of PCL injured knees (median, 7.5 mm; range, 5.3–11.5 mm; medial median, 6.2 mm; medial range, 3.7–8.3 mm; lateral median, 7.9 mm; lateral range, 5.3–11.5 mm) was larger than that seen in uninjured knees (median, 5.0 mm; range, 4.0–7.6 mm; medial median, 4.6 mm; medial range 3.4–7.1 mm; lateral median, 5.6; lateral range, 4.5–11.2 mm). The increment in the medial distance was similar to that in the lateral distance. Posterior laxity of injured knees was 9.1 (median); 5.4 to 15.2 (range) mm greater than that of uninjured knees. We found no correlation between posterior laxity and the flexion gap increment.

Conclusions Our data suggest the intact PCL controls posterior displacement and maintains the flexion gap.

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Each author certifies that his or her institution approved the human protocol for this investigation, that all investigations were conducted in conformity with ethical principles of research, and that informed consent for participation in the study was obtained.

This study was performed at Osaka Rosai Hospital, Sakai City, Osaka, Japan.

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Introduction

The PCL is a strong stabilizer of the knee and provides posterior stability to the tibia. However, sagittal alignment of the PCL with the knee at 90° flexion shows the PCL might play a role not only in posterior stabilization but also in maintaining the flexion gap (Fig. 1) [14]. The flexion gap is difficult to observe and few studies have investigated it in normal knees. Tokuhara et al. [21] observed the flexion gap (the distance between the femoral posterior condyles and the tibial boney surface) under valgus-varus stress by using MRI, and found the lateral flexion gap was larger than the medial flexion gap in normal knees. Although MRI enables three-dimensional observation of the knee at various flexion angles and under various forces, the procedure is time-consuming, costly, and can be painful

Fig. 1 Posterior laxity is sagittal laxity (how the tibia moves posteriorly from the femur). The flexion gap is the vertical distance between the femoral posterior condyle and the tibial plateau, and is the average of the medial and lateral distance.

for patients with knee pain. Kanekasu et al. [9] obtained plain radiographs with the x-ray beam projected along the femoral shaft (axial radiography) to evaluate the rotational position of the femoral component after TKA, and suggested this technique might be useful to evaluate the flexion gap. Similarly, Tokuhara et al. [20] reported axial radiography had similar accuracy to MRI in determining the flexion gap, and was useful in evaluating the flexion gap in normal knees.

Some studies [6, 7, 12, 23] have suggested the PCL plays a role in maintaining the flexion gap because the flexion gap increased after resection of the PCL in TKA. It is uncertain, however, whether the intact PCL maintains the flexion gap because the PCL is influenced by conditions such as arthritis and by anesthesia during TKA. Therefore, we sought to confirm whether the intact PCL maintains the flexion gap by using axial radiography.

Patients and Methods

We retrospectively reviewed the radiographs of 17 patients (13 men, four women; mean age, 36 years; age range, 18–57 years) with isolated PCL injuries between March 2007 and May 2011. All patients reported having a sense of unclear knee instability in activities of daily living, despite receiving nonoperative medical treatment (eg, NSAIDs, physical therapy, braces). The time from injury to evaluation ranged from 3 months to 25 years. All patients had a positive traumatic history suggestive of PCL injury, and PCL tear was diagnosed by physical examination and confirmed by MRI and/or arthroscopy. The diagnosis was confirmed independently by three special sports orthopaedic surgeons (SH, YT, YY); if all three doctors came to the same conclusion, an isolated PCL injury was diagnosed. Thirteen of 17 patients had a posterior drawer Grade 2 PCL injury (6- to 10-mm posterior tibial translation) [2],

Fig. 2 Each patient was instructed to sit on the table with his or her lower legs hanging and to relax the thigh muscle. A 1.5-kg weight then was attached to the patient's ankle. The broken black arrow shows the path of the beam.

whereas the other four patients had a Grade 3 injury (> 10-mm posterior tibial translation). We excluded patients with fracture, osteoarthritis, or other ligament lesions, such as those of the medial collateral ligament or posterolateral structures, with the use of multiple radiographs, MR images, and physical examinations. Regardless of the amount of posterior laxity, all 17 patients were scheduled for PCL reconstruction owing to their symptoms related to knee instability in activities of daily living.

The use of axial radiography, with the knee at 90° flexion and the x-ray beam projected along the femoral axis, has been reported in previous studies [3, 5, 9, 13, 20] (Fig. 2). This view can be used to evaluate the flexion gap and rotational position of the femoral component after TKA. The method for obtaining axial radiographs in our study was as follows: Each patient sat on a table with his or her lower legs hanging, and a 1.5-kg weight attached to the ankle (Fig. 2). The marker used to calculate radiograph

magnification (a 50-mm-diameter stainless steel disc) was attached to the lateral side of the knee. The patient was instructed to relax the leg muscles, especially the quadriceps. We explained the procedure, waited a few minutes until the patient was relaxed, and confirmed relaxation from clinical palpation of the thigh. We then obtained axial radiographs of the injured and contralateral uninjured knees to evaluate the increment of the flexion gap (Fig. 3).

All measurements were obtained at least twice by two observers (YM, YK). We measured the medial and lateral vertical distances from the midpoint of each condyle to the tibial bony surface on the axial radiograph; the flexion gap was expressed as the average of these distances (Fig. 4). Then, the increment of the flexion gap was determined by comparing the radiographs of the patients' injured and contralateral uninjured knees. We also evaluated posterior laxity on the gravity sag view [19]. The method for obtaining the gravity sag view was as follows (Fig. 5): The patient was placed supine with both hips flexed at 45° and both knees at 90° flexion. The x-ray beam was projected from the lateral side. Side-to-side differences of the tibia-femur step-off were measured as posterior shift, and posterior laxity was calculated as the amount of posterior shift in the injured knee minus that in the contralateral uninjured knee. We found interobserver and intraobserver variability in measuring the flexion gap on axial radiography to have intraclass correlation coefficients of 0.970 and 0.956, respectively.

Image data were transferred to a personal computer (Let's Note; Panasonic Corporation, Kadoma, Japan), and the flexion gap and posterior laxity were measured manually using an image analysis program (Vectorworks; A&A

Co, Ltd, Tokyo, Japan). The program could change magnification, contrast, and brightness of the radiographs. We could manually detect the edges of measurement accurately by the program. The unit of measurement was 0.1 mm. Results were analyzed using StatView® 5.0 (SAS Institute Inc, Cary, NC, USA). Differences in instability between the injured and contralateral uninjured knees were analyzed by using the Wilcoxon signed-rank test, and Spearman's rank correlation coefficient test was used to analyze the correlation between the increment of the flexion gap and posterior laxity.

Fig. 4 We measured the medial and lateral vertical distances from the midpoint of each condyle to the tibial bony surface. Flexion laxity was expressed as the average of the medial and lateral distances.

Fig. 3A–B Axial radiographs of (A) the PCL-injured knee and (B) the contralateral uninjured knee were used to evaluate the increment of the flexion gap. The magnification is the same because it was adjusted by using a 50-mm-diameter stainless steel disc as a marker.

Fig. 5A–B Gravity sag views of (A) the contralateral uninjured knee and (B) the PCL-injured knee were taken in the supine position with both hips flexed at 45° and both knees at 90° flexion. Posterior shift is the side-to-side difference of the tibia-femur step-off.

Fig. 6 A scatterplot shows the increment of posterior laxity versus the increment of the flexion gap. R_s is Spearman's rank correlation coefficient. No correlation was seen between the increment of posterior laxity and the increment of the flexion gap.

Results

The intact PCL appears to maintain the flexion gap: the flexion gap of the PCL-injured knees was greater ($p < 0.001$) than that of the uninjured knees. The median flexion gap of uninjured knees was 5.3 mm (range, 4.0–7.6 mm; medial median, 4.6 mm; medial range, 3.4–7.1 mm; lateral median, 5.6 mm; lateral range, 4.5–11.2 mm), whereas that of PCL-injured knees was 7.5 mm (range, 5.5–9.1 mm; medial median, 6.2 mm; medial range, 3.7–8.3 mm; lateral median, 7.9 mm; lateral range, 5.3–11.5 mm). The median flexion gap increment was 2.0 mm (range, 0.5–3.8 mm; medial median, 2.0 mm; medial range, 0.0–3.8 mm; lateral median, 2.3 mm; lateral range, –1.2 to 5.1 mm). We found no difference ($p = 0.543$) between the increment of the medial distance and that of the lateral distance. Median posterior laxity was 9.1 mm (range, 5.4–15.2 mm).

The increment of the flexion gap did not correlate with posterior laxity ($R_s = 0.02$; Fig. 6).

Discussion

With the knee at 90° flexion, the PCL is inclined 65° relative to the tibial plateau [14]; as such, there is the possibility the PCL plays a role not only in posterior stabilization but also in maintaining the flexion gap. It has been difficult to evaluate this possibility, however, because of the difficulty in observing the flexion gap. Tokuhara et al. [20] reported the flexion gap obtained from axial radiography correlated with that measured by MRI. Therefore, we used axial radiography to confirm whether the intact PCL maintains the flexion gap.

We acknowledge several limitations to our study. First, previous reports [3, 5, 9, 13, 20] have suggested contraction of the quadriceps could affect the flexion gap; therefore, it is essential that subjects have relaxed leg muscles during the examination. We explained this to each patient and waited a few minutes until the patients were relaxed before obtaining the radiographs. We also confirmed muscle relaxation with clinical palpation and facial expression. Second, prior researchers [3, 9, 13] have tested various loads on the ankle, and found the gap width was not directly proportional to the amount of distraction force. They concluded a 1.5-kg weight was suitable in terms of the subjects' comfort and the reproducibility of the results and that the interobserver and intraobserver variabilities in measuring the flexion gap on axial radiography were small. In the current study, we also found small interobserver and intraobserver variabilities in measuring the flexion gap on axial radiography. Third, the number of patients in our study was small; however, all the results showed that, compared with contralateral uninjured knees, PCL-injured knees had an increased flexion gap. There was no exception

Table 1. Summary of selective PCL resection studies

Study	Number of knees	Increment of flexion gap (mm)	
		Medial	Lateral
Kadoya et al. [7]	18 TKAs	4.8 ± 0.4*	4.5 ± 0.4*
Yagishita et al. [23]	30 TKAs	2.7 ± 0.7*	2.9 ± 2.2*
Current study	17 PCL injuries	2.0 (0.0–3.8) [#]	2.3 (–1.2 to 5.1) [#]

* Values are expressed as mean ± SD; [#]values are expressed as median (range).

in the results. The result was reliable, even though the sample was small. Fourth, it was difficult to diagnose chronic isolated PCL injuries. The diagnosis was made independently by three sports orthopaedic surgeons who each had more than 10 years of experience. Each surgeon had to come to the same conclusion to diagnose a chronic isolated PCL injury. Four patients had a Grade 3 injury and some researchers [11, 17] have reported one should be suspicious for other ligament injuries in patients with Grade 3 injuries. However, the four patients in our study were diagnosed with an isolated PCL injury based on a thorough medical examination. Even if these four patients were excluded, the flexion gaps of the remaining 13 patients' PCL-injured knees increased, and the results would be the same.

Our data suggest the intact PCL not only controls posterior displacement but also maintains the flexion gap. Compared with the contralateral uninjured knees, all PCL-injured knees had an increased flexion gap. Some studies [7, 23] have reported PCL resection in TKA increases the flexion gap (Table 1); however, those researchers used intraoperative measurements and knees with osteoarthritis or rheumatoid arthritis. The intraoperative measurements might have been influenced by anesthesia, the incision, and/or release of the capsule. Our patients had one normal knee and one with isolated PCL injury, and our observations suggested the PCL in the normal knees played a role in maintaining the flexion gap.

Other factors reportedly influence flexion laxity. Saeki et al. [16] and Yagishita et al. [23] reported that selective medial collateral ligament release in TKA increases the flexion gap by using an intraoperative measurement. Furthermore, selective lateral collateral ligament release or popliteus tendon release increased the flexion gap in one cadaver study [8]. With the knee at 90° flexion, the patella tendon was inclined 90° relative to the tibial plateau in one MRI study [10]. Owing to these factors, we believe that compared with posterior laxity, the flexion gap increment in PCL-injured knees is small. The average flexion gap increment was slightly smaller than that in TKA [7, 23] (Table 1). The small differences might be related to the influence of anesthesia, incisions, and/or capsular release.

Regardless of the amount of posterior laxity, all patients were scheduled for PCL reconstruction owing to the symptoms related to knee instability. The surgical indication for chronic isolated PCL injury remains controversial [11]. Many reports [1, 4, 15, 18, 22] have noted athletes with PCL deficiencies function well despite obvious posterior laxities, while some studies [15, 18, 22] have indicated there is no correlation between the amount of posterior laxity and clinical knee function. Surgical decision making is difficult and based on many factors. In addition to our results, if it is clarified that an increase in the flexion gap influences the clinical results of PCL reconstruction, the flexion gap increase could be a factor to help clinicians choose a surgical intervention for treatment of PCL injuries.

Our observations suggest the intact PCL functions not only as a posterior stabilizer of the tibia but also maintains the flexion gap in normal knees.

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