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Title:

Does meniscal pathology alter gait knee biomechanics and strength post ACL reconstruction?

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ABSTRACT

Purpose: Individuals following anterior cruciate ligament reconstruction (ACLR) with concomitant meniscal pathology have a higher risk of developing knee osteoarthritis (OA) compared to those with isolated ACLR. Knee extensor weakness and altered dynamic knee joint biomechanics have been suggested to play a role in the development of knee OA following ACLR. This study investigated whether these factors differ in people following ACLR who have concomitant meniscal pathology compared to patients with isolated ACLR.

Methods: Thirty-three patients with isolated ACLR and 34 patients with ACLR and meniscal pathology underwent strength and gait assessment 12-24 months post-operatively. Primary measures were peak isometric knee extensor torque and knee adduction moment (peak and impulse). Secondary measures included peak knee flexion moment and knee kinematics (sagittal and transverse).

Results: There were no between-group differences in knee extensor strength (mean difference (95%CI) 0.09 (-0.23 to 0.42) Nm/kg, n.s.), peak knee adduction moment (-0.02 (-0.54 to 0.49) Nm/(BW×HT)%, n.s.) or knee adduction moment impulse (0.01 (-0.15 to 0.17) Nm/(BW×HT%, $p = \text{n.s.}$). No between group differences were found for any secondary measures.

Conclusions: No evidence was found to suggest that the higher prevalence of OA in patients with ACLR and meniscal pathology compared to patients with isolated ACLR is attributed to reduced knee muscle strength or altered knee joint biomechanics assessed 1-2 years post-surgery. Given that there is a higher incidence of knee OA in patients with concomitant meniscal pathology and ACLR, further investigation is needed so that population-specific rehabilitation protocols can be developed.

Keywords: knee moments; anterior cruciate ligament reconstruction; meniscus; strength

Level of Evidence: III

INTRODUCTION

Knee osteoarthritis (OA) is reported in 50-70% of patients with anterior cruciate ligament or meniscal injury 10-15 years following trauma [4,26]. Meniscal injuries are often observed at the time of anterior cruciate ligament reconstruction (ACLR), and there is a higher prevalence of knee OA in patients with ACLR and concomitant meniscal pathology compared to those with isolated ACLR [26,29]. Two prominent factors thought to play a role in the pathogenesis of OA are quadriceps muscle weakness [30] and altered knee biomechanics [2].

In ACLR patients, knee extensor weakness is reportedly associated with tibiofemoral joint space narrowing [42] which is of concern given that knee extensor weakness may persist up to five years following surgery [31,42]. Theoretically, patients with greater knee trauma, such as with combined ACL and meniscal pathology, have greater arthrogenic muscle inhibition compared to patients with isolated ACLR, which may impair maximal knee muscle strength [32]. Some studies indicate that knee extensor strength is comparable in patients with isolated ACLR and in patients with combined meniscal pathology [25, 29, 37]. Nonetheless, these studies evaluating strength are somewhat heterogeneous as it is unclear whether patients with isolated ACLR had stable meniscal tears [25] and whether those with concurrent trauma also had chondral lesions and collateral ligament damage [29]. Further research is required in homogenous samples to investigate the effect of meniscal pathology on knee muscle strength.

External knee joint moments are widely used to infer loading of internal knee structures and are of clinical interest. Specifically, a higher external knee adduction moment has been associated with incidental knee pain [1] and medial tibiofemoral structural disease onset and progression [5,8,9,17,28], albeit not specifically in ACLR patients. Medial tibiofemoral compartment OA is more prevalent than lateral compartment OA [4] and is possibly

associated with progressive varus malalignment [11]. Few studies have evaluated frontal plane moments during gait in people following ACLR, with inconsistent findings. Studies have reported no difference [15,43,48], a higher [7] and a lower [45] peak adduction moment. Although the mechanism is unclear, one explanation for these inconsistencies may include the presence of meniscal pathology. A small study suggests that patients following ACLR with concomitant meniscal pathology may have higher knee joint moments compared to patients with isolated ACLR [43]. Conversely, some evidence implicates the role of reduced loading in structural tibiofemoral joint deterioration following ACLR [46,33]. Overall these findings provide rationale for further investigation to compare knee joint moments in patients with isolated ACLR to patients with combined ACLR and meniscal pathology.

Kinematic adaptations following ACLR may be associated with a transfer of knee joint cartilage contact locations, which are in turn, related to cartilage morphology [23,35]. Several studies have reported kinematic differences in gait for patients following ACLR [15,20,34]. Patients typically demonstrate greater external tibial rotation [34] and less knee flexion [20,15]. However, it is unclear if the populations studied had meniscal pathology [15,34]. Thus, it remains to be investigated if differences in knee joint kinematics during gait exist between patients with isolated ACLR and ACLR with concomitant meniscal pathology.

Given the evidence implicating the knee adduction moment and knee extensor weakness with knee joint OA, the aim of this study was to test the hypotheses that patients with ACLR and meniscal pathology would demonstrate i) greater knee adduction moment during gait and; ii) lower knee extensor strength compared to individuals with isolated ACLR. Secondary exploratory aims of this study were to determine between-group differences in knee joint kinematics (sagittal and transverse) and the peak knee flexion moment were apparent.

MATERIALS AND METHODS

Participants aged 18-50 years who had undergone ACLR surgery with a hamstring autograft 12-24 months previously were recruited from two orthopaedic surgeons (HGM and TSW) in Melbourne, Australia, who specialise in ACL surgery. Participants were excluded if they presented with any of the following: previous ACLR surgery, knee surgery since ACLR, history of injury or surgery in the contralateral knee, grade III collateral ligament damage, posterior cruciate ligament injury, positive pivot test or symptoms of knee instability (e.g. clicking, catching) during activities of daily living, hopping jumping or plyometric activity, or any musculoskeletal, cardiovascular or neurological conditions influencing walking, sports activity or daily function.

Surgical procedure

Using the same arthroscopic technique, all surgical procedures were performed by one of the two surgeons. An ipsilateral, four-strand semitendinosus and gracilis tendon graft was harvested through a 3 to 4cm incision over the pes anserinus. The graft was attached to a 15mm Closed Loop (CL) Endobutton (Smith and Nephew Endoscopy, Mansfield, Mass., USA). A similar anatomical tunnel placement was performed in all participants. A FastFix 360 meniscal suture was utilised. An interference screw ensured tibial fixation. Indications for a meniscal repair or partial meniscectomy were determined on the appearance of the tear at the time of surgery. Partial meniscectomy was performed in participants with non-repairable meniscal injuries that were deemed to be potentially symptomatic.

Strength assessment

Maximal isometric knee extensor strength was assessed in the reconstructed leg. As previously described [41], participants were seated on a small platform placed on the seat of a 125-APM isokinetic Kin-Com dynamometer system (Kin-Com, USA). In this position the

hip was at 90° flexion and the knee joint at 60° flexion. Following familiarisation, participants performed two, five-second maximal knee extensor isometric contractions with a 40 second rest period between each repetition. Participants received a standardised script of strong verbal encouragement and were instructed to push as hard as they could in addition to receiving real-time visual feedback of torque signals. The peak torque from two trials was recorded, corrected for gravity and normalised to body mass (Nm/kg).

Motion analysis

Kinematic (120 Hz) and kinetic data (1080 Hz) were collected using a 12-camera Vicon motion analysis system (Oxford, UK) synchronized with three AMTI force plates (AMTI, Inc., Watertown, MA, USA) embedded in the floor. Following the application of 40 reflective markers (13 millimetre diameter), participants completed a calibration trial that was used to define lower limb joint centres [36]. The knee joint centre was defined as the mid-line between the medial and lateral epicondyles. All participants were instructed to walk at a normal self-selected pace, while wearing Straprunner IV running sandals (Nike, Beaverton, US). For each task, three trials were acquired for the ACLR leg where there was a complete foot strike on a single force plate.

Ground reaction forces and marker trajectories were filtered at 6Hz using a 2nd order, dual-pass Butterworth filter. Using a seven-segment lower limb direct kinematics and inverse dynamics model, net external moments were calculated in Vicon BodyBuilder (Oxford UK) according to Schache and Baker [36]. Knee flexion angle and rotation angle at initial contact and peak angle during stance were recorded. The first peak knee adduction moment, positive knee adduction moment impulse representing the positive area under the knee adduction moment curve and first peak knee flexion moment were normalised by dividing by body weight (N) times body height (m) and expressed as a percentage. The peak flexion moment

was also investigated, given evidence to suggest that it mediates medial knee contact force [44] and is related to cartilage changes in people with OA [9] and arthroscopic partial meniscectomy [17]. Dependent variables were extracted for each trial and averaged across trials.

Descriptive Data

Knee laxity

Using the instrumented KT1000TM knee arthrometer (Medmetrics Corp, San Diego, CA, USA) an experienced examiner assessed anterior knee joint laxity. With the participant supine, and the knee in 30° flexion, 133 Newtons (30 lbs) of anteriorly-directed force was applied and side-to-side difference was recorded.

Static alignment

Static alignment was assessed from the marker data, as participants stood comfortably in a neutral position barefoot. The angle of the knee-ankle joint vector was recorded in the laboratory frontal plane, where a negative value indicates a valgus position.

Self-reported measures

Participants completed the Cincinnati Knee Rating Scale [3] to assess symptoms, functional limitations with sports and daily activities, participant perception of knee conditions and sports and occupational activity levels. Physical activity levels were assessed using the Tegner Activity Scale [6]. The University of Melbourne Human Research Ethics Committee (ethics ID 1136167) approved this study, and all participants provided written informed consent.

Statistical analysis

Sample size estimates were performed on the primary variables (knee adduction moment and knee muscle strength). In patients with knee OA, a 22% greater knee adduction moment has been associated with OA progression risk of greater than 6 times [28]. Hence, a 10% change in peak knee adduction moment may be clinically relevant. Thus, we took a pragmatic approach and aimed to detect a large effect size of 0.8 with 80% statistical power. This required a sample size of 31 participants per group. For quadriceps strength, previous literature indicates that differences in knee extensor strength between ACLR patients with normal joint space width and ACLR patients with joint space narrowing yield an effect size of 0.86 [42]. Thus, 24 participants per group were required with 90% statistical power to detect an effect size of 0.86 in knee extensor strength between groups. Normal distribution of the data was assessed using Kolmogorov-Smirnov tests. Independent t-tests and chi-square tests were used to evaluate between-group differences as appropriate. To adjust for the confounding influence of walking speed on knee joint biomechanics [22], ANCOVA was also performed to evaluate group differences in knee joint biomechanics variables during gait. SPSS version 22.0 (SPSS, Chicago, IBM) was used to perform all statistical analyses with significance accepted at $p < 0.05$.

RESULTS

Of the 67 participants involved in this study, all had knee muscle strength data available for analysis, while 63 had gait data available (two participants did not undergo gait analysis, and data from two participants could not be used due to missing static markers and incomplete foot strikes on force plates). Two independent groups were investigated: one group included individuals who had undergone an isolated unilateral ACLR ($n = 33$) and a second group that included patients with a combination of unilateral ACLR and concurrent meniscal pathology ($n = 34$).

Participant characteristics

There were no significant differences in participant characteristics between the isolated ACLR group and combined ACLR and meniscus group (Table 1). The groups were similar regarding demographic profiles, surgical information and physical function. Information pertaining to the location and management of meniscal pathology of the ACLR group is presented in Table 2.

Knee extensor strength

Similarly, there were no between-group differences in maximal knee extensor strength (mean difference (95% CI) 0.09 (-0.23 to 0.42) Nm/kg, n.s.) (Figure1).

Joint moments

There were no between-group differences in walking speed, knee flexion moment, peak knee adduction, knee adduction moment impulse or during gait (Table 3). The ensemble curves indicate comparable magnitudes throughout stance for knee flexion moment and the knee adduction moment (Figure 2).

Joint kinematics

No between-group differences were observed for sagittal plane or transverse plane kinematics at initial contact or peak during stance phase (Table 3). The ensemble curves indicate similar trajectories throughout for knee flexion, and highlight variability in the transverse plane (Figure 3).

DISCUSSION

The most notable finding of this study was that we found no evidence to suggest that knee extensor muscle strength, external knee joint moments or knee joint kinematics differ between patients with isolated ACLR and those with ACLR and meniscal pathology 12-24 months following surgery. Investigating these parameters could: 1) facilitate understanding of why patients following ACLR with combined meniscal pathology have a higher prevalence of knee OA than those with isolated ACLR, and 2) assist with the development of cohort-specific rehabilitation programs, given that both knee joint biomechanics and knee muscle strength are modifiable through conservative management.

Knee extensor strength is associated with knee OA development 2.5 to 14 years following ACLR in people with no radiographic or symptomatic knee OA at baseline [30], and joint space narrowing in ACLR patients at 4 years following surgery [42]. Furthermore, arthrogenic muscle inhibition has been found in patients following knee trauma, which included patients following ACLR [19], although the time from injury or surgery is unclear. Some authors suggest that greater knee trauma is associated with arthrogenic muscle inhibition which could impair knee muscle strength [32]. Nonetheless, our findings are consistent with previous research reporting no difference in knee muscle strength between those with isolated ACLR and patients with ACLR and concurrent injuries at 6 months, 1 year, 2 years [29] and 5-15 years [30,37] following surgery. Collectively, these results suggest that factors other than maximal knee muscle strength relatively early in the post-surgical period increase the risk of subsequent knee OA in people undergoing ACLR with combined meniscal pathology.

The knee adduction moment was investigated given its association with medial tibiofemoral knee OA onset and progression [5,8,9,17,28]. The knee adduction moment might have been expected to be higher with ACLR and meniscal pathology as previous literature reports higher peak knee adduction moment in people following arthroscopic partial meniscectomy [18,39]. Conversely, lower medial compartment compressive loading and knee adduction moment have been implicated in OA changes following ACLR [46]. Contrary to our hypothesis, and the findings of a previous pilot investigation in a smaller sample [43], we found no difference in the knee adduction moment during walking between the groups. Varma et al. [43] utilized a pragmatic sample that was relatively heterogeneous comparing patients with ACLR and ‘cartilage, meniscus or ligament damage’, to patients with isolated ACLR [43]. However, it is unclear from this study if higher frontal plane moments observed in the group with combined trauma were attributed to structural damage other than meniscal pathology.

Other factors should be considered when interpreting the kinetic findings of the current study. Walking speed was not standardised between the groups. While walking speed can affect the ground reaction force and thereby the knee adduction moment [22], there were no statistical between-group difference in walking speeds and results remained unchanged when controlling for walking speed. It is also of interest to note that no between-group difference in the peak knee extensor moment was observed, since higher external knee flexion moments have been associated with greater medial tibiofemoral joint contact force [44]. Knee joint moments have been widely used as an indicator of knee joint load as they have been positively related to adverse structural change [5,8,9,17,28]. However, although the external knee adduction moment provides an indication of mediolateral load distribution, we cannot account for the effect of meniscal pathology on the actual stresses within the knee joint.

50 External load measures would only detect changes due to these if the patients actually walked
51 differently. Muscle activation patterns that are likely to influence both the distribution and
52 magnitude of compressive joint load [27] and are altered in patients following ACLR [10,16].
53 Muscle activation patterns can be used in musculoskeletal modelling to estimate compressive
54 joint load; however such estimates do not directly assess or reflect impaired meniscal
55 function.

56
57 Although authors have reported kinematic differences in patients following ACLR [15,20,34]
58 and patients following partial meniscectomy [14,40] we observed no between-group
59 differences in kinematics associated with cartilage morphology [22,34]. Overall, we found no
60 evidence to suggest that the higher prevalence of OA in patients with ACLR and meniscal
61 pathology compared to patients with isolated ACLR is attributed to the variables assessed in
62 this study. Although not measurable in vivo, it is important to acknowledge that the integrity
63 of the meniscus to dissipate forces across the tibiofemoral joint is impaired following partial
64 meniscectomy [12,13]. Thus, increased joint contact stresses are likely higher in those with
65 concomitant meniscal pathology and ACLR compared to those with isolated ACLR. This
66 remains the most likely explanation for the increased subsequent OA in these individuals.
67 Additionally, future work should investigate other factors such as inflammation in patients
68 with concurrent meniscal pathology, which has also been suggested to play a role in
69 subsequent OA development [13]. Following ACLR, patients have chronically elevated pro-
70 inflammatory cytokines and matrix metalloproteinase [21] that may lead to osteoarthritic
71 changes. However, to our knowledge no study has investigated whether differential
72 inflammatory responses are apparent with the addition of meniscus pathology.

The strengths of our study include a relatively large sample of patients who underwent similar surgical procedures using a hamstring graft, 12-24 months previously by one of two experienced surgeons. There are several limitations to this study, which may in part explain the absence of between-group significant differences. First, as this was an exploratory study that was powered to detect large effect sizes (≥ 0.80) it is possible that a larger sample size may have yielded smaller yet potentially relevant differences between groups. Second, we assessed isometric knee extensor strength which has been suggested by some authors as a less discriminating assessment of knee muscle strength compared to isokinetic strength in meniscectomy patients tears [38]. Thus, we cannot conclude that clinically relevant differences in isokinetic measures of strength were not present between the groups. Third, the combined group included patients with lateral and medial meniscal pathology that underwent both/either repair and partial meniscectomy, which may influence our observations. However, post-hoc analyses confirmed that knee moments and knee extensor muscle strength were not significantly different between those who had medial or lateral meniscal pathology. Although sample sizes are small, these findings are consistent with previous research in people undergoing arthroscopic partial meniscectomy without ACLR [40]. Furthermore, no differences were found between those with isolated ACLR and participants with ACLR combined with medial meniscal pathology. Thus, our data do not suggest that factors related to location of meniscal tear or surgical intervention were influential. Fourth, patients were assessed 12-24 months post-ACLR, and only during walking, which may have been too early after surgery [24,47] or insufficiently vigorous [14] to detect differences. Finally, marker-based gait analysis has known sources of measurement error including soft tissue artefact and marker misplacement. These combined sources of error may have masked any subtle changes in gait patterns between the groups. Lastly, the absence of preoperative data and a lack of

information regarding specific exercises (i.e., sets, repetitions and frequency) performed by participants as part of their post-surgery rehabilitation is a limitation.

CONCLUSION

In summary, there was no evidence to suggest that a higher prevalence of OA in patients with ACLR and meniscal pathology compared to patients with isolated ACLR was attributed to the variables assessed 1-2 years post-surgery. Our findings suggest that factors other than quadriceps strength and knee biomechanics during gait in the early post-ACLR years may contribute to the higher subsequent prevalence of knee OA in patients with ACLR and meniscal pathology compared to those with isolated ACLR.

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119 **CONFLICT OF INTEREST**

120 The authors declare that they have no conflict of interest.

121

122 Figure Legends

123 **Figure 1.** Peak isometric knee extensor muscle strength assessed at 60° knee flexion for the
124 isolated ACLR group and combined group (ACLR and meniscal pathology)

125

126 **Figure 2.** Ensemble average of the knee adduction moment ($\pm$ standard deviation) during
127 stance in the ACLR group (black line) and ACLR with combined meniscal pathology (red
128 line).

129 **Figure 3.** Ensemble average of the knee flexion moment ($\pm$ standard deviation) during
130 stance in the ACLR group (black line) and ACLR with combined meniscal pathology (red
131 line).

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Fig. 1

Fig. 2

Fig. 3

Table 1 Participant characteristics

Characteristic	ACLR (n=33)	ACLR & meniscus pathology (n=34)	<i>p</i> -value
Demographics			
Age, years	27.1 ± 6.3	29.2 ± 6.0	n.s.
Male, n (%)	18 (55%)	25 (74%)	n.s.
Body mass (kg)	77.9 ± 16.0	78.7 ± 13.5	n.s.
Body height (m)	1.76 ± 0.10	1.75 ± 0.10	n.s.
Body mass index (kg/m ²)	25.1 ± 3.5	25.6 ± 3.1	n.s.
Dominant side affected (yes:no)	32:1	27:7	n.s.
Laxity (deg)	2.4 ± 2.3	2.2 ± 2.5	n.s.
Static alignment (deg) [†]	-2.7 ± 2.1	-2.6 ± 1.8	n.s.
Surgical information			
Affected knee (right:left)	18:15	15:19	n.s.
Surgeon (TW:HM)	11:22	13:21	n.s.
Injury nature (contact:non-contact)	13:20	12:22	n.s.
Time from surgery (months)	16.7 ± 3.1	16.4 ± 2.8	n.s.
Time between injury and surgery (months)	5.8 ± 12.9	17.5 ± 39.0	n.s.
Chondral defects present			
Medial femoral condyle, n	2	8	n.s.
Medial tibial plateau, n	0	0	-
Lateral femoral condyle, n	1	7	n.s.
Lateral tibial plateau, n	1	3	n.s.
Physical function related information			
Current sport participation (yes:no)	25:8	22:12	n.s.
Previous sport participation (yes:no)	33:0	34:0	.
Return to same level of sport participation (yes:no)	13:20	7:27	n.s.
Tegner activity scale (1-10)	6 ± 2	6 ± 2	n.s.
Cincinnati Knee Rating Scale (6-100)	87.4 ± 13.8	85.0 ± 11.1	n.s.

Values are mean ± SD

ACLR: anterior cruciate ligament reconstruction

Cincinnati Knee Rating Scale (6-100), higher score indicates better function

Tegner 1-10, higher scores indicate higher levels of physical activity

TW: Tim Whitehead

HM: Hayden Morris

[†] negative value indicates valgus position

Table 2 Location and management of meniscus pathology in patient with combined ACLR and meniscus pathology (n=34)

	Total	Stable untreated tears	Repair	Partial meniscectomy
Medial meniscus pathology	14	1	7	6
Lateral meniscus pathology	11	-	1	10
Combined lateral and medial meniscus pathology	9	2	2	13

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Table 3 Means and standard deviation between groups for gait speed and internal knee moment

Gait variables	ACLR (n = 33)	ACLR and meniscus (n = 30)	Unadjusted mean difference (95% confidence intervals)		Adjusted mean difference (95% confidence intervals) [†]	
			ACLR minus ACLR and meniscus	p-value	ACLR minus ACLR and meniscus	p-value
Gait speed (m/s)	1.36 ± 0.10	1.38 ± 0.20	-0.02 (-0.08 to 0.03)	n.s.	.	.
<i>Knee Joint Kinematics</i>						
Knee flexion initial contact °	7.9 ± 3.4	8.9 ± 3.8	-0.8 (-0.1 to 2.5)	n.s.	-0.8 (-2.5 to 1.0)	n.s.
Peak knee flexion angle °	54.5 ± 6.2	52.2 ± 6.9	2.3 (-1.0 to 5.6)	n.s.	2.0 (-1.3 to 5.3)	n.s.
Knee internal rotation at initial contact °	4.1 ± 4.3	3.3 ± 5.1	0.9 (-1.5 to 3.3)	n.s.	0.9 (-1.5 to 3.4)	n.s.
Peak internal rotation angle °	7.8 ± 4.4	6.4 ± 4.2	1.4 (-3.5 to 0.8)	n.s.	1.5 (-0.7 to 3.7)	n.s.
<i>Knee Joint Kinetics</i>						
Peak KAM, Nm/(BW×HT)%	2.52 ± 1.12	2.54 ± 0.92	-0.02 (-0.54 to 0.49)	n.s.	0.03 (-0.48 to 0.54)	n.s.
KAM impulse, Nm.sec/(BW×HT)%	0.74 ± 0.33	0.73 ± 0.29	0.01 (-0.15 to 0.17)	n.s.	0.01 (-0.15 to 0.17)	n.s.
Peak KFM, Nm/(BW×HT)%	4.97 ± 1.26	5.22 ± 1.55	-0.26 (-0.97 to 0.45)	n.s.	-0.18 (-0.87 to 0.51)	n.s.

KAM: knee adduction moment; KFM: knee flexion moment; BW: body weight; HT: height [†] adjusted for walking speed

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