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Original article

The mechanism of "killer turn" causing residual laxity after transtibial posterior cruciate ligament reconstruction

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Abstract

Background: The residual laxity after transtibial posterior cruciate ligament (PCL) reconstruction has been reported by several authors. The sharp angle where the graft exits the tibial tunnel, which is known as "killer turn", is believed to be the main reason. The purpose of this study was to reveal the mechanism of "killer turn" and its effect on both graft and tunnel inlet.

Methods: A total of 60 New Zealand white rabbits were included. All transtibial PCL reconstructions were performed *in vitro* using Achilles tendon autograft. The cyclic loading tests were conducted when reconstructed knees were subjected to 1500 cycles of tensile force of 50 N with the angle of pull at 45° to the tibial plateau. The tunnel inlet enlargement, graft elongation, stiffness, graft displacement, load to failure, and failure site were all recorded and analysed.

Results: Fifty-eight New Zealand white rabbits were available for biomechanical evaluation. The subjects had significant graft elongation and tunnel enlargement. The graft displacement increased by a mean of 0.92 ± 0.36 mm (16.70%). At the 1500th cycle, the grafts were significantly elongated by 5.59 ± 4.98%, and the tunnel inlet diameter was also significantly enlarged by 12.08 ± 4.31%. There was a linear correlation between total graft displacement and the two variables ($R2 = 0.402$, $F = 18.515$, $p < 0.001$). The coefficient for tunnel inlet enlargement was 0.419 ($p = 0.006$), and for graft elongation was 0.583 ($p = 0.002$). At the load-to-failure test, the failure load was 81.19 ± 20.13 N. Of the 58 grafts, 31 (53.45%) failed at the "killer turn", 13 (22.41%) for the para-tunnel fracture, seven (12.07%) for the graft pull-out, and the remaining seven (12.07%) for the rupture at the mounting site.

Conclusion: The mechanism of "killer turn" compromising posterior stability was that the repetitive friction between graft and tunnel inlet not only attenuated the graft, but also enlarged the tunnel inlet, leading to the displacement of the graft.

Keywords: biomechanical; osteoporosis; posterior cruciate ligament

Introduction

Transtibial is a popular technique of posterior cruciate ligament (PCL) reconstruction. Unfortunately, the clinical outcome was not always affirmed, because several authors reported the residual laxity after surgery. The residual laxity was a multifactorial issue, the graft type, tunnel placement, femoral impingement, and the "killer turn" were all considered relevant.[1–4] The "killer turn" where the graft makes an acute bend around the proximal posterior tibia frequently causes wearing of the graft and is thought to be one of the main risk factors of residual laxity.[4–12]

In the literature, it is the elongation and thinning of the graft that has been most frequently discussed.[3,4,7, 9,13–22] However, as "forces always come in pairs", it is reasonable to assume that the repetitive abrasion between graft and bone not only compromises the graft tissue, but also enlarges the tunnel inlet. To our knowledge, there was little evidence focusing on the

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tunnel inlet enlargement of transtibial PCL reconstruction, especially the effect of tunnel inlet enlargement on the graft displacement.

The purpose of this study was to: (1) prove the elongation of the graft after cyclic loading test; (2) detect if there was tunnel inlet enlargement; and (3) determine the correlation between graft elongation and total graft displacement and between tunnel enlargement and total graft displacement. We hypothesized that the total graft displacement was contributed to by both graft elongation and tunnel inlet enlargement.

Methods

A total of 60 skeletally mature, female New Zealand white rabbits were included in the study. The mean body weight was 3.5 ± 1.1 kg, with a mean body length of 31.5 ± 5.4 cm. The mean age was 16.4 ± 0.2 months. All rabbits were allowed free access to water and standard commercial rabbit feed during the acclimatization period of 1 week. All rabbits were then sacrificed for *in vitro* transtibial reconstruction. The leg side was determined randomly by a self-designed software.

On the tibia specimens, the native PCL footprint was identified before the PCL fibres were removed, leaving the remnants of the fibrous attachments intact.[9] The Achilles tendon autograft was harvested. A tunnel was drilled with 3.0 mm K-wire from the anteromedial cortex of the tibia to the centre of the native footprint at an angle of 60 degrees. The Achilles tendon autograft was then fashioned to a diameter of approximately 3.0 mm, with the calcaneus carefully removed. Braded with 4-0 Ethibond (Ethicon Inc., Somerville, NJ, USA), the graft was pulled through the tunnel and fixed with a self-made interference screw on the anteromedial cortex of tibia.

According to the testing protocol of experiments in the literature,[8,10,11,14,23] after the tibial side of the graft was fixed first, the other end of the graft was mounted onto an MTS model-858 Mini Bionix servohydraulic materials testing machine (MTS Systems, Minneapolis, MN, USA) for a cyclic loading test, and subjected to 1500 cycles of loading at 1 Hz. Our device employed an MTS Model 858.11 load unit that is fatigue rated at 10 kN, with a resolution of 0.001 N. This freestanding load unit can be operated at frequencies up to 30 Hz. The device can detect a displacement range of $\pm$ 50 mm, with a resolution of 0.01 mm. The instrumental error was $< 0.5\%$. The loading force was 50 N. Both tibias and calcaneus were secured with polymethyl methacrylate bone cement and then mounted on the device. The graft was secured at an angle of 45 degrees to the tibial plateau on the sagittal plane. After the cyclic loading test, the graft was loaded to failure. The ultimate failure load was then recorded. The graft displacement was recorded as the displacement of the cross-head of the device and can be read on the displacement-cycle curve. The elongation of the grafts (the length change of the mid-third segment of the graft), the total displacement of the grafts (the difference of graft displacement between the 20^{th} cycle and the 1500^{th} cycle at a loading of 50 N), the graft stiffness, and the tunnel inlet enlargement of the transtibial group were recorded and analysed. The tunnel inlet measurement was performed on the three-dimensional (3D) micro-CT reconstruction images (SKYSCAN 1172, Bruker microCT, Ghent, Belgium). The slice thickness was set as 1 μm. The diameter of the tunnel inlet was measured on the 3D-reconstruction model. The tunnel enlargement was expressed as the equation of difference of pre- and post-testing diameter divided by pre-testing diameter. In a qualification of this method, it showed an inter-group correlation coefficient of 0.935, and an intra-group coefficient of 0.973.

Statistical analysis

All data were expressed as average $\pm$ standard deviation. The variables included graft elongation, graft displacement, load to failure, stiffness, and the tunnel enlargement of the transtibial group. Confirmed by the Kolmogorov-Smirnov test, all variables were of normal distribution. The paired t test was utilised to analyse the tunnel enlargement of the transtibial group. Student t test was applied for the assessment of graft displacement, graft elongation, and stiffness. The Pearson correlation test and linear regression analysis were conducted between graft elongation and tunnel enlargement and graft total displacement. The level of significance was $p < 0.05$.

Results

There were a total of 60 transtibial PCL reconstructions performed. Among them, 1 subject failed at the 1200^{th} cycle and 1 failed at the 300^{th} cycle for the rupture at the "killer turn". At last, 58 subjects survived the cyclic loading test. The subjects had significant graft elongation and tunnel enlargement. The graft displacement at the 1500^{th} cycle was 16.70% greater than at the 20^{th} cycle, resulting in the mean total graft displacement of 0.92 ± 0.36 mm. At the 1500^{th} cycle, the grafts were significantly elongated by $5.59 \pm 4.98\%$, while the tunnel inlet diameter was also significantly enlarged by $12.08 \pm 4.31\%$. The biomechanical properties of transtibial PCL reconstruction grafts are illustrated in Table 1.

The Pearson correlation test revealed a significant correlation between both graft elongation ($p = 0.001$) (Figure 1) and tunnel inlet enlargement ($p = 0.004$) (Figure 2) and total graft displacement. A further linear regression was conducted, demonstrating the linear correlation between total graft displacement and the two variables ($R^2 = 0.402$, $F = 18.515$, $p < 0.001$). The coefficient for tunnel inlet enlargement was 0.419 ($p = 0.006$), and for graft elongation was 0.583 ($p = 0.002$).

At the load-to-failure test, the failure load was 81.19 ± 20.13 N. Of the 58 grafts, 31 (53.45%) failed at the "killer turn", 13 (22.41%) for the para-tunnel fracture, seven (12.07%) for the graft pull-out, and the remaining seven (12.07%) for the rupture at the mounting site. The failure load was 81.31 ± 19.57 N for "killer turn", 82.66 ± 23.17 N for "para-tunnel fracture", 70.37 ± 17.12 N for "graft pull-out" and 88.39 ± 19.70 N for "rupture at the mounting site". No significant difference was detected among the four subgroups.

Table 1
The biomechanical property of transtibial posterior cruciate ligament (PCL) reconstruction grafts.

	Value at 20th cycle	Value at 1500th cycle	Mean difference	Percentage	*p*
Graft displacement (mm)	6.07 ± 1.66	6.99 ± 1.61	0.92 ± 0.36 mm	16.70 ± 8.84%	< 0.001
Graft elongation (mm)	7.30 ± 1.70	7.70 ± 1.79	0.40 ± 0.30 mm	5.59 ± 4.98%	< 0.001
Tunnel inlet diameter (mm)	4.51 ± 1.12	5.06 ± 2.28	0.55 ± 0.36 mm	12.08 ± 4.31%	< 0.01
Stiffness (N/mm)	45.28 ± 14.47	54.76 ± 14.43	9.48 ± 6.74 N/mm	22.94 ± 22.64%	< 0.001
Failure load (N)	81.19 ± 20.13				

Discussion

The residual laxity after transtibial PCL reconstruction has been a well-identified problem. According to a systematic review, the overall rate of "abnormal" or "severely abnormal" was 25%. They concluded that, although the transtibial PCL reconstruction can successfully improve the posterior laxity, it still cannot restore normal stability.[20] In a clinical study of arthroscopic transtibial PCL reconstruction, Chen et al[15] reported that the rate of KT-1000 > 5 mm was 19% at 4-year follow up. Similarly, Norbakhsh et al[24] resulted in an "abnormal" rate of 19.3% at 4 years after autograft transtibial PCL reconstruction. Admittedly, the source of the residual laxity was multifactorial. Several factors may be responsible for it, such as allograft,[4,25] tunnel placement,[26] femoral impingement, tendon-bone healing impairment and wearing at the "killer turn". However, most authors believed that the "killer turn" was the main contributor.[3,4,10–12,17–21,23,26–28]

In the present study, the total graft displacement was observed to increase by nearly 17%. It could be assumed that the total displacement of the graft was composed of three parts: the graft elongation, the displacement at the fixation site, and the tunnel enlargement of the transtibial group. As the interference screw fixation has been proved to minimize the graft displacement,[21] the graft elongation and tunnel inlet enlargement became the main contributors.

Several biomechanical studies have verified the wearing of the graft at the "killer turn", but very few studies provided a quantitative evaluation of graft elongation. According to a study by Markolf et al,[23] the mean thinning at the "killer turn" of graft was 40.6%, which was consistent with the findings of Bergfeld et al.[14] The mean elongation was (9.8 mm/15 mm) 65.3%. Because of the different testing protocol and specimen, although the mean elongation in the present study was only 5.59%, the increment was still significant. In terms of the failure site, in a biomechanical study by Markolf et al,[23] with the transtibial technique, 10 of 31 grafts (32.2%) failed before the completion of the cyclic loading test. Similarly, in the 12 cadaveric knees in a study by McAllister et al,[11] two specimens failed before the cyclic loading test was completed. Both specimens ruptured at the killer turn near the tibial attachment of the graft. Consistent with the above studies, two grafts ruptured at the "killer turn" before the 1500th cycle, and more than 50% grafts ruptured at the "killer turn" in the load-to-

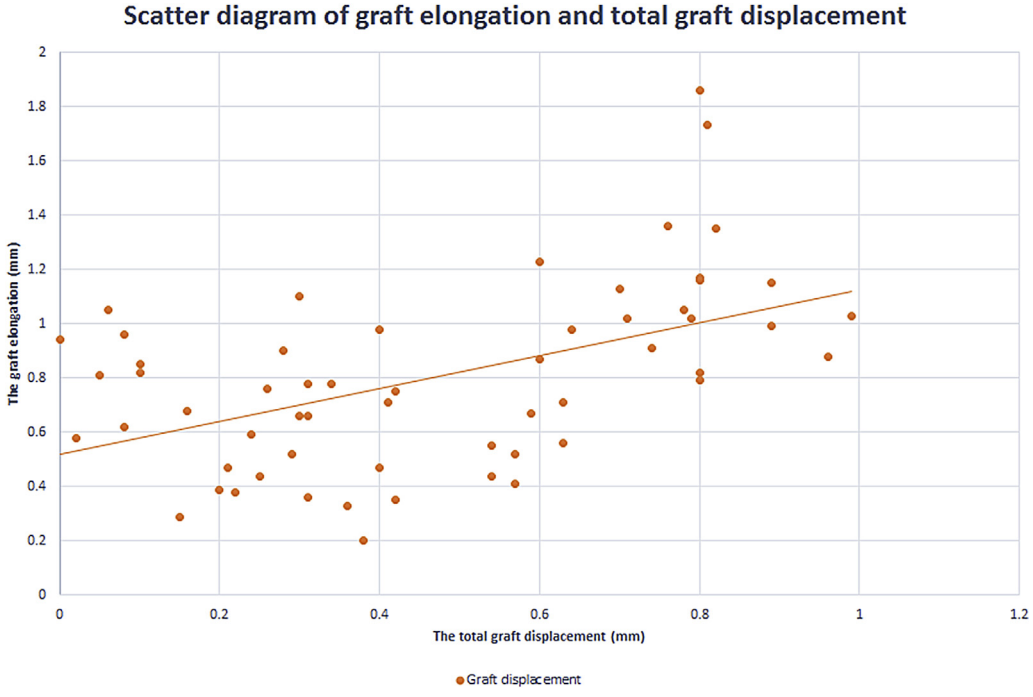

Figure 1. The scatter diagram of graft elongation and total graft displacement. There was a linear correlation between these two variables ($p = 0.001$).

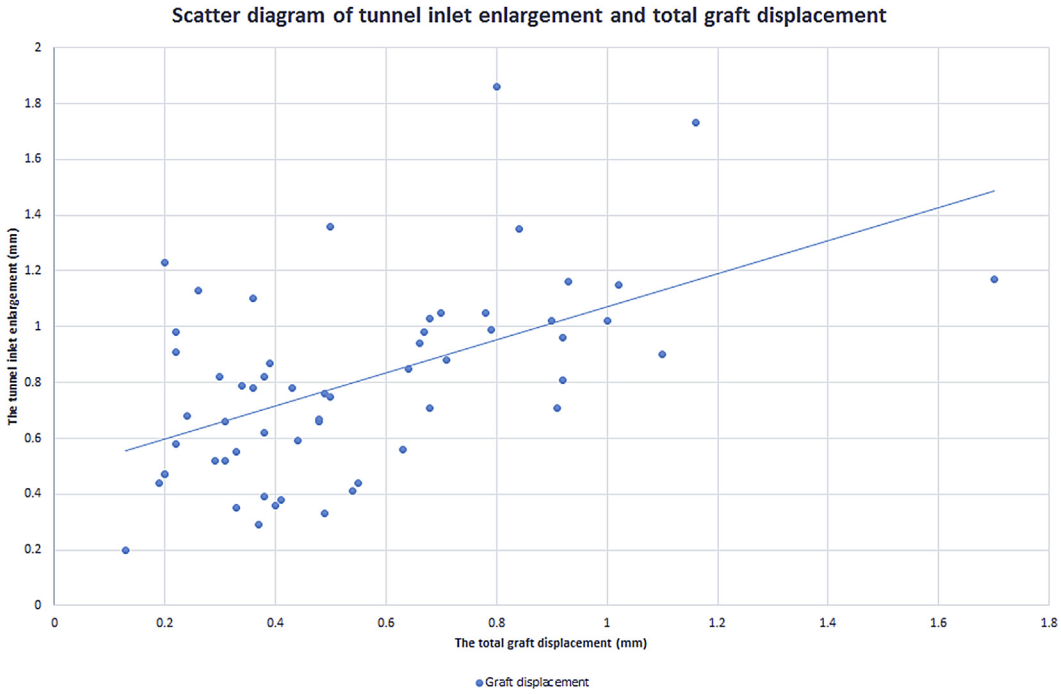

Figure 2. The scatter diagram of tunnel inlet enlargement and total graft displacement. There was a linear correlation between these two variables ($p = 0.004$).

failure test. Our study reached the same results as previous studies,[9–11,14,17] that the "killer turn" was a risk factor of transtibial PCL reconstruction failure.

Although there were several studies focusing on the effect of "killer turn", from our perspective, the mechanism of "killer turn" causing residual laxity was still uncertain. It is the wearing of the "graft" that was well-discussed, while the abrasion of "bone" was seldom mentioned. As "forces always come in pairs", the abrasion of bone, which was manifested as the tunnel inlet enlargement, shall not be ignored. Adding that the tunnel enlargement after anterior cruciate ligament (ACL) reconstruction has been well discussed,[29,30] it is reasonable to extrapolate that the tunnel inlet enlargement after transtibial PCL reconstruction was also existent.

According to the literature, the prevalence of tibial tunnel volume enlargement after ACL reconstruction was from 48.6–54.2%.[31] The prevalence of enlargement > 10% was reported to be 53.2%.[32] However, as far as we were concerned, very few studies[33] had focused on the tunnel inlet enlargement after PCL reconstruction. In a clinical study on the evaluation of tunnel volume enlargement after isolated PCL reconstruction using the arthroscopic transtibial technique with allograft, Kwon et al[33] reported that the incidence of tibial tunnel enlargement was 5.4% (3 of 56 patients). The definition of enlargement in their study was defined as a volume increase > 44%, which was a relatively high standard, so the incidence was as low as only 5.4%. At 1-year follow-up, the mean increment of tibial tunnel volume was 9.9% in the allograft group and 11.2% in the mixed graft group. In the present study, it was the diameter of the tunnel inlet instead of the volume that was measured. The tunnel inlet was enlarged by 12.08%.

In the present study, the graft displacement was regarded as a more direct measurement to reflect the mechanism of "killer turn" than anteroposterior laxity. We detected that both graft elongation and tunnel inlet enlargement were linearly correlated with total graft displacement. The coefficient of elongation and tunnel enlargement was close (0.583 vs. 0.419), indicating a comparable contribution of both factors. Traditionally, the "killer turn" was only believed to cause the abrasion and attenuation to the graft.[27] However, in the present study, we proposed that the "killer turn" also compromised the posterior proximal tibial cortex. The mechanism of the tunnel inlet enlargement would be a result of the competition of graft and proximal tibial cortex at the "killer turn". In the first place, according to the three-point bend mode theory,[3] the compressive force on the cortex of the "killer turn" was the resultant force of the axial load along the proximal and distal components of the graft. The compressive force may potentially cause abrasion of both graft and tunnel inlet abrasion at the graft tunnel margin at the proximal tibia after PCL reconstruction during the cyclic loading test. Second, the pressure on the tunnel inlet was determined, not only by the compressive force, but also the area of the contact surface. In the present study, the diameter of the Achilles tendon was fashioned to approximately 3 mm. Taking the posterior tibial fossa into consideration, the contact area would be very limited, making the pressure relatively high. Third, it was also assumed that the proximal tibial wall of the tunnel with low bone mineral density (BMD) would be more vulnerable to be oscillated and thinned by repetitive micromotion of the graft in the tunnel. In the literature, it had been proven that low BMD is a risk factor of tunnel enlargement after ACL reconstruction.[34]

The clinical relevance of the present study was based on the enlightenment of a new mechanism of "killer turn" causing residual laxity. First, when dealing with the tibia of low BMD, the inlay technique might be indicated to avoid excessive residual laxity. Second, if the inlay technique is contraindicated, the effect of "killer turn" should be eliminated by anterolateral tunnel direction,[18] the remnant preserving technique,[35] smoothing the posterior tibial facet,[27] and more conservative rehabilitation protocol.

There were some limitations in the present study. In the first place, this is an *in vitro* study evaluating the biomechanical property in an ideal model. Because the present study was *in vitro*, there seemed to be less reliability than an *in vivo* study with muscles and capsule intact and a healing process. Second, the biomechanical testing protocol varied greatly between different studies. The results in the present study can only be interpreted into a finding of a new mechanism of "killer turn" causing residual laxity. The data cannot be directly applied to clinical practice.

The conclusion of this study was that both graft elongation and tunnel inlet enlargement were contributors in the mechanism of "killer turn" causing residual laxity.

Conflicts of interest

The authors have no conflicts of interest relevant to this article to declare

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