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Physiologic Preoperative Knee Hyperextension is a Predictor of Failure in an ACL Revision Cohort:

A Report from the MARS Group

Abstract

Background—The occurrence of physiologic knee hyperextension in the revision anterior cruciate ligament (ACL) reconstruction population, and its effect on outcomes, has yet to be reported.

Hypothesis/Purpose—The prevalence of knee hyperextension in revision ACL reconstruction, and its effect on 2-year outcome were studied with the hypothesis that preoperative physiologic knee hyperextension 5 degrees is a risk factor for ACL graft rupture.

Study Design—Cohort study.

Methods—Revision ACL reconstruction patients were identified and prospectively enrolled between 2006 and 2011. Study inclusion criteria were patients undergoing single bundle graft reconstructions. Patients were followed up at 2 years, and asked to complete the identical set of outcome instruments (IKDC, KOOS, WOMAC, and Marx activity rating score), as well as to provide information regarding revision ACL graft failure. A regression model using graft failure as the dependent variable included age, gender, graft type at the time of the revision ACL surgery, and physiologic preoperative passive hyperextension greater than or equal to 5 degrees (HE 5; "yes/no"), to assess these potential risk factors for clinical outcomes 2 years after revision ACL reconstruction.

Results—There were 1,145 subjects included in the analyses. Two-year follow-up was attained on 91%. The median age was 26, with age being a continuous variable. Those below the median were grouped as "younger" and those above "older" (Age of 25/75 quartiles: IQR= 20, 35), and 42% were female. There were 50% autografts, 48% allografts, and 2% which had a combination autograft plus allograft. Passive knee HE 5 degrees was present in 374 (33%) of our revision cohort, with 52% being female. Graft rupture at 2-year follow-up occurred in 34 cases in our entire cohort, of which 12 were in our HE 5 group (3.2% failure rate) and 22 were in the non-hyperextension group (2.9% failure rate). The median age of subjects that failed was 19, compared to 26 for those with intact grafts. Three variables included in our regression model were significant predictors of graft failure: younger age (odds ratio [OR] = 3.6; 95% CI: 1.6, 7.9; p= 0.002), use of allograft (OR = 3.3; 95% CI: 1.5, 7.4; p= 0.003), and HE 5 degrees (OR = 2.12; 95% CI: 1.1, 4.7; p= 0.03).

Conclusion—This study found that preoperative physiologic passive knee hyperextension greater than or equal to 5 degrees is present in 1/3 of patients who undergo revision ACLR. HE 5 was found to be an independent significant predictor of graft failure after revision ACLR with a >2X odds ratio) of subsequent graft rupture in revision ACL surgery.

Clinical Relevance—Future study of these variables is warranted, and reports on ACL reconstruction results should separately evaluate the group of knees with HE 5.

Keywords

Anterior Cruciate Ligament; Knee Hyperextension; Graft Failure; Graft Tensioning

Introduction

The Multi-center ACL Revision Surgery cohort was conceived to evaluate the outcomes of revision ACLR when it was determined that existing prospective cohorts not specifically focused on revision ACLR lacked sufficient numbers of revision cases for efficient and meaningful analysis^{6,16,17}. Previous outcomes studies of anterior cruciate ligament reconstruction (ACLR) have generally reported results of the entire patient cohort, and few have considered the variable of passive knee hyperextension as a risk factor. ¹⁴ The risk factors that predispose a person to an ACL injury vary. It is well known that many knees passively extend past 0 degrees and that some, but not all, of these knees may have generalized laxity^{9,13–15,23}. Knee hyperextension (HE) has been previously reported to be an intrinsic factor contributing to ACL injury^{13,14}. The occurrence of knee hyperextension in the revision ACL reconstruction (ACLR) population and its effect on outcomes have yet to be reported. As such, the purpose of this study was to determine the prevalence of preoperative physiologic knee hyperextension in a large revision ACL reconstruction population, and to determine its effect on 2-year outcome. The study hypothesis was that hyperextension 5 degrees is an independent risk factor for ACL graft rupture 2 years following revision ACL surgery.

Materials and Methods

Study Design

With the backing of the American Orthopaedic Society for Sports Medicine (AOSSM), this study began enrolling patients on March 1, 2006. This prospective longitudinal cohort design was established to determine prognosis and identify predictors of outcome of revision ACLR. The consortium consists of 83 enrolling surgeons at 52 sites in 28 American states and two Canadian provinces. Surgeons in this study practice in both academic (n=23; 44%) and private practice (n=29; 56%) sites. The epidemiology, demographics and specific methods of this consortium have been previously published ¹⁷.

Data Sources

Because double bundle reconstructions have been reported to potentially limit terminal extension and graft outcomes in patients with hyperextension ^{10,23}, only those patients who received a single bundle ACL graft at the time of their revision surgery were included for

this study. After obtaining informed consent, subjects were asked to complete a patient questionnaire that contains a series of validated patient-oriented outcome instruments, including the subjective International Knee Documentation Committee (IKDC), the Knee injury and Osteoarthritis Outcome Score (KOOS), and the Marx activity rating scale. The surgeon completed a form detailing the injury, treatment history of the knee, associated intra-articular injuries, a detailed examination under anesthesia according to IKDC guidelines, surgical technique, and graft utilized in the revision surgery. As such, all passive range of motion measurements were obtained under anesthesia by the participating study surgeon at the time of the revision surgery. Passive motion of both knees was documented. Knee HE was measured either with a goniometer or by bed-heel distance (1 cm = 1 degree).

Patient Follow-up

Patients were followed up for 2 years and asked to complete the identical set of outcome instruments. Patients were also contacted by telephone to determine if graft failure diagnosed by MRI and any subsequent surgeries had occurred since their initial revision reconstruction. If so, operative reports were obtained, whenever possible, to verify pathology and treatment.

Statistical analysis

To describe our patient sample, we summarized continuous variables with the median and inter-quartile ranges (IQR), and categorical variables with frequencies and percentages. These variables were compared using nonparametric statistics (Wilcoxon test for continuous variables, Pearson test for categorical variables). Graft failure or subsequent revision surgery at 2 years follow-up was the primary endpoint. A logistic regression model was used to analyze four independent variables: age, gender, graft type (autograft vs. allograft), and symmetrical knee HE 5 degrees vs. < 5 degrees, to assess these potential risk factors on outcomes 2 years after revision ACL reconstruction. Age was treated as a continuous variable, and the mean was 26 years. Those below the mean were grouped as "younger", and those above the mean were grouped as "older" (Age of 25/75 quartiles: IQR= 20, 35). The statistical model would support the analysis of only four independent variables. Statistical analysis was performed with free open source R statistical software (www.r-project.org).

Results

Baseline Characteristics (Table 1)

There were 1,145 subjects included in the analyses, as 54 were excluded from the overall cohort because a double bundle reconstruction was performed, and 6 subjects had missing range of motion values. Two-year follow-up was attained on 91%. The median age was 26 (IQR= 20, 35), and 42% were female. There were 50% autografts, 48% allografts, and 2% which had a combination autograft plus allograft. Passive knee HE 5 degrees was present in 374 (33%) of our revision cohort, with 52% being female. There was no difference in baseline characteristics between the two groups.

Graft Failure (Table 2; Figure 1)

Known graft rupture occurred in 34 cases in our entire cohort, of which 12 were in our HE 5 group (3.2% failure rate) and 22 were in our non-hyperextender group (2.9% failure rate). The median age of subjects that failed was 19, compared to 26 for those with intact grafts. Three variables included in our regression model were significant independent predictors of graft failure: younger age (odds ratio [OR] = 3.6; 95% CI: 1.6, 7.9; p= 0.002), use of allograft (OR = 3.3; 95% CI: 1.5, 7.4; p= 0.003), and HE 5 degrees (OR = 2.12; 95% CI: 1.1, 4.7; p= 0.03). Gender was not predictive of graft failure in our study.

Discussion

Our group has previously reported that young age and allograft use were predictive of graft failure in a revision ACL cohort^{3–6,16–20}. This report reaffirms that conclusion. Additionally, the hypothesis that HE 5 is predictive of graft failure at two years in a revision ACL cohort is supported by the results of this study. This is the first evidence based report of such an association.

When discussing physiologic knee hyperextension in relation to ACL tears, there are several topics for consideration; 1) Prevalence in an ACL tear cohort, 2) Relative risk for ACL tear, 3) Association with increased AP knee laxity, 4) Relative risk for ACL graft rupture after primary ACLR, 5) Relative risk for ACL graft rupture after revision ACLR, 6) Does restoration of full HE increase the risk of ACL graft rupture in primary or revision ACLR?, 7) Does loss of full HE after primary or revision ACLR lead to knee OA?

Published reports suggest that knee HE is not necessarily associated with increased AP laxity or increased risk of ACL tear^{1,2,10,13,14,21,23}. Double bundle ACLR has been shown to capture the knee and limit HE after ACLR, as compared to single bundle ACLR^{21–24}. Changes of OA after ACLR are related to loss of full knee HE. There is some suggestion that the loss of HE may be causal of OA, and not simply a result of the OA^{24–26,28,29}.

Many surgeons have the opinion, based on their clinical experience, that it is more difficult to achieve long-term ACL graft integrity in a knee that hyperextends significantly. However, there has been relatively little investigation into this subset of ACL patients, so evidence based data is lacking. Few have studied preoperative knee HE as separate group when reporting results. Benner, et al. reported that knee hyperextension was not associated with an increased risk of graft rupture in a large cohort of primary ACLRs treated by a single surgeon with a consistent surgical technique using BTB autograft, graft tensioning in full HE and fixation with sutures tied over ligament buttons^{2,29}. This contrasts with our findings in a cohort with numerous surgeons and techniques. It is possible that the less rigid fixation and tensioning in full HE could reduce the risk of excessive graft tension and failure using the methods described by Benner, et al^{2,29}. It is possible that certain graft tensioning and fixation techniques might increase the risk of graft rupture as compared to others. This might be the reason that our data supports HE 5 as an independent risk factor for graft rupture in a revision ACL cohort. However, our methods do not address this issue.

This study includes numerous surgeons using differing graft choices, knee flexion angles when tensioning and methods of fixation $^{3-6,16-20}$. The methods and power of this cohort study allow for the multivariate analysis of the binary comparison of HE <5 vs. HE 5, but do not allow the analysis of HE as a continuum of numbers. The cutoff of 5 degrees was an arbitrary decision based on the hypothesis. This multivariate data analysis yielded significant findings, and the use of multivariate analysis is essential to identify the independent risk factors, for the seemingly small difference between 2.9 and 3.2 percent graft failure in the two groups can be affected by more than one independent variable. The increased risk (odds ratio = 2.12) of graft failure in HE knees in our cohort raises the suspicion that surgical technique in the recurvatum knee will affect risk of graft failure. Future study is warranted, and importantly, to determine the ideal graft, knee flexion angle for graft tensioning, and fixation method 29 . Although the OR of graft failure in HE knees (2.12) was not as striking as in the younger age (3.6) and allograft (3.3) groups, it was significant (p 0.03) when using the previously published regression model used in all reports from this cohort.

This study provides a high level of evidence that HE 5 is an independent risk factor for failure in revision ACL surgery. This revision ACL cohort had a large number of HE 5 knees (33%), and it is possible that there would be more HE 5 knees in a revision population if in fact HE is a risk factor for failure in ACL surgery. Knowing this to be the case, we retrospectively reviewed the MOON cohort and found that 32% of the knees in a primary ACLR cohort had HE 5³. It is beyond the scope of this study to compare the two groups or to determine if HE 5 is a risk factor in primary ACLR, but it is certainly of note that roughly 1/3 of knees in both a primary and a revision ACLR cohort demonstrate passive HE 5.

Physiologic hyperextension is an important variable in the surgical technique of ACLR, given that several studies have shown increasing tension and elongation in the native ACL in terminal extension^{7,8,11,12,27}. However, none of these studies looked at extension past 10 degrees, and only a few examined extension past 0 degrees. Larson recently reported on outcomes in ACL reconstruction comparing generalized laxity and knee HE >10 and found that these variables increased the rates of both graft failure and contralateral ACL tear. The knee HE + generalized laxity group had triple the risk of graft rupture (24.4% vs 7.7%) in a large cohort with 6-year follow-up¹⁴. When considering combined graft failure plus contralateral ACL tear rates, the knee HE + generalized laxity group had much greater risk also (34% vs 12%)¹⁴.

Akelman reported that there was no difference in clinical outcomes between a low graft tension group as compared to a high graft tension group in long-term follow-up¹. The methods of this study did not seek to assess the relative risk of graft tensioning or fixation as a function of knee position. We believe that this issue is an important subject for future study. Additionally, the biomechanics literature is deficient related to knee kinematics and ACL tension patterns in the HE 5 knee. Yet, fully one-third of primary and revision ACL reconstruction patient populations are in this category.

There are limitations to this study that need to be addressed. Wide variability exists between surgeons related to the knee flexion angle for ACL graft fixation. In our study's surgeon

group this varied from a position of full passive hyperextension to 20 or 30 degrees of knee flexion. With wide variability in HE, one must realize the adverse effects on graft excursion, tension and even overload to failure that may be caused by the combination of hyperextension and a graft that has increasing strain in terminal extension, yet is fixed with the knee in flexion. This is particularly concerning, given the recent trend for lower "anatomic" femoral tunnel positions, that do create slightly more graft excursion, as compared to "higher" AM femoral bundle techniques 1,7,8,11,27. Secondly, we did not have MRI, physical examination, or objective laxity measurements (i.e. KT-1000) to determine knee laxity in the patient cohort. Rather, our determination of graft failure was based on patient telephone calls to determine the status of each graft. We considered MRI and/or revision surgical confirmation as evidence of graft failure. Examination of each patient by a single examiner would have been exceedingly difficult given the number of patients and the fact that they were treated at 52 sites in 28 American states and two Canadian provinces.

Conclusions

This study found that preoperative physiologic knee hyperextension greater than or equal to 5 degrees is present in 1/3 of patients who undergo revision ACLR. HE 5 was found to be an independent significant predictor of graft failure after revision ACLR, with a $>2\times$ odds ratio of subsequent graft rupture. Younger age (<26) and use of allograft tissues for ACLR are associated with a >3X odds ratio of subsequent graft rupture.

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References

- Akelman MR, Fadale PD, Hulstyn MJ, et al. Effect of Matching or Over-constraining Knee Laxity During Anterior Cruciate Ligament Reconstruction on Knee Osteoarthritis and Clinical Outcomes: A Randomized Controlled Trial with 84-Month Follow-up. Am J Sports Med. 2016 Jul; 44(7): 1660–70. [PubMed: 27159308]
- Benner RW, Shelbourne KD, Gray T. The degree of knee extension does not affect postoperative stability or subsequent graft tear rate after anterior cruciate ligament reconstruction with patellar tendon autograft. Am J Sports Med. 2016 Apr; 44(4):844–9. [Epub ahead of print]. [PubMed: 26801922]
- 3. Borchers JR, Kaeding CC, Pedroza AD, Huston LJ, Spindler KP, Wright RW, MOON Consortium and the MARS Group. Intra-articular findings in primary and revision anterior cruciate ligament reconstruction surgery: a comparison of the MOON and MARS study groups. Am J Sports Med. 2011 Sep; 39(9):1889–93. [Epub ahead of print]. [PubMed: 21646434]
- 4. Brophy RH, Haas AK, Huston LJ, Nwosu SK, MARS Group. Wright RW. Association of meniscal status, lower extremity alignment, and body mass index with chondrosis at revision anterior cruciate ligament reconstruction. Am J Sports Med. 2015 Jul; 43(7):1616–22. [Epub ahead of print]. [PubMed: 25899434]

- Brophy RH, Wright RW, David TS, McCormack RG, Sekiya JK, Svoboda SJ, Huston LJ, Haas AK, Steger-May K, Multicenter ACL Revision Study (MARS) Group. Association between previous meniscal surgery and the incidence of chondral lesions at revision anterior cruciate ligament reconstruction. Am J Sports Med. 2012 Apr; 40(4):808–14. [Epub ahead of print]. [PubMed: 22374942]
- 6. Chen JL, Allen CR, Stephens TE, Haas AK, Huston LJ, Wright RW, Feeley BT, Multicenter ACL Revision Study (MARS) Group. Differences in mechanisms of failure, intraoperative findings, and surgical characteristics between single-and multiple-revision ACL reconstructions: a MARS cohort study. Am J Sports Med. 2013 Jul; 41(7):1571–8. [Epub ahead of print]. [PubMed: 23698386]
- Cooper DE, Small J, Urea L. Factors affecting graft excursion patterns in endoscopic anterior cruciate ligament reconstruction. Knee Surg Sports Traumatol Arthrosc. 1998; 6(Suppl 1):S20–24. [PubMed: 9608459]
- 8. Cooper DE, Small J, Urea L. Factors affecting graft excursion patterns in endoscopic anterior cruciate ligament reconstruction: The effect of guide offset and rotation. Arthroscopy. 1998 Mar; 14(2):164–70. [PubMed: 9531127]
- Estes K, Cheruvu B, Lawless M, Laughlin R, Goswami T. Risk assessment for anterior cruciate ligament injury. Arch Orthop Trauma Surg. 2015 Oct; 135(10):1437–43. [Epub ahead of print]. [PubMed: 26198056]
- Ettinger M, Petri M, Guenther D, Liu C, Krusche C, Liodakis E, Albrecht UV, Krettek C, Jagodzinski M. Anatomic double-bundle ACL reconstruction restricts knee extension in knees with hyperextension. Knee Surg Sports Traumatol Arthrosc. 2013 Sep; 21(9):2057–62. [Epub ahead of print]. [PubMed: 22945469]
- Hoher J, Kanamori J, Fu FH, Woo SL. The position of the tibia during graft fixation affects knee kinematics and graft forces for anterior cruciate ligament reconstruction. Am J Sports Med. 2001; 29:771–776. [PubMed: 11734491]
- 12. Kennedy JC, Hawkins RJ, Willis RB. Strain Guage Analysis of Knee Ligaments. Clin Orthop Relat Res. 1977 Nov-Dec;(129):225–9.
- 13. Kim SJ, Moon HK, Kim SG, Chun YM, Oh KS. Does severity of specific joint laxity influence clinical outcomes of anterior cruciate ligament reconstruction? Clin Orthop Relat Res. 2010 Apr; 468(4):1136–41. [Epub ahead of print]. [PubMed: 19582525]
- Larson CM, Bedi A, Dietrich ME, et al. Generalized Hypermobility, Knee Hyperextension, and Outcomes After Anterior Cruciate Ligament Reconstruction: Prospective, Case Controlled Study with Mean 6 Years Follow-up. Arthroscopy. 2017 Oct; 33(10):1852–1858. [PubMed: 28599980]
- 15. Lin HC, Lai WH, Shih YF, Chang CM, Lo CY, Hsu HC. Physiological anterior laxity in healthy young females: the effect of knee hyperextension and dominance. Knee Surg Sports Traumatol Arthrosc. 2009 Sep; 17(9):1083–8. [Epub ahead of print]. [PubMed: 19575181]
- Magnussen RA, Trojani C, Granan LP, Neyret P, Colombet P, Engebretsen L, Wright RW, Kaeding CC, MARS Group. SFA Revision ACL Group. Patient demographics and surgical characteristics in ACL revision: a comparison of French, Norwegian, and North American cohorts. Knee Surg Sports Traumatol Arthrosc. 2015 Aug; 23(8):2339–48. [Epub ahead of print]. [PubMed: 24850239]
- 17. MARS Group. Wright RW, Huston LJ, Spindler KP, Dunn WR, Haas AK, Allen CR, Cooper DE, DeBerardino TM, Lantz BB, Mann BJ, Stuart MJ. Descriptive epidemiology of the Multicenter ACL Revision Study (MARS) cohort. Am J Sports Med. 2010 Oct; 38(10):1979–86. [Epub ahead of print]. [PubMed: 20889962]
- MARS Group; MARS Group. Effect of graft choice on the outcome of revision anterior cruciate ligament reconstruction in the Multicenter ACL Revision Study (MARS) cohort. Am J Sports Med. 2014 Oct; 42(10):2301–10. [Epub ahead of print]. [PubMed: 25274353]
- 19. MARS Group. Radiographic findings in revision anterior cruciate ligament reconstruction from the MARS cohort. J Knee Surg. 2013 Aug; 26(4):239–47. [Epub ahead of print]. [PubMed: 23404491]
- 20. Matava MJ, Arciero RA, Baumgarten KM, Carey JL, DeBerardino TM, Hame SL, Hannafin JA, Miller BS, Nissen CW, Taft TN, Wolf BR, Wright RW, MARS Group. Multirater agreement of the causes of anterior cruciate ligament reconstruction failure: a radiographic and video analysis of the MARS cohort. Am J Sports Med. 2015 Feb; 43(2):310–9. [Epub ahead of print]. [PubMed: 25537942]

- 21. Matsubara H, Okazaki K, Tashiro Y, Toyoda K, Uemura M, Hashizume M, Iwamoto Y. Intercondylar roof impingement after anatomic double-bundle anterior cruciate ligament reconstruction in patients with knee hyperextension. Am J Sports Med. 2013 Dec; 41(12):2819–27. [Epub ahead of print]. [PubMed: 24099713]
- Morgan JA, Dahm D, Levy B, Stuart MJ, MARS Study Group. Femoral tunnel malposition in ACL revision reconstruction. J Knee Surg. 2012 Nov; 25(5):361–8. [Epub ahead of print]. [PubMed: 23150344]
- 23. Saito K, Hatayama K, Terauchi M, Hagiwara K, Higuchi H, Takagishi K. Clinical outcomes after anatomic double-bundle anterior cruciate ligament reconstruction: comparison of extreme knee hyperextension and normal to mild knee hyperextension. Arthroscopy. 2015 Jul; 31(7):1310–7. [Epub ahead of print]. [PubMed: 25801047]
- 24. Shelbourne KD, Urch SE, Gray T, Freeman H. Loss of normal knee motion after anterior cruciate ligament reconstruction is associated with radiographic arthritic change after surgery. Am J Sports Med. 2012 Jan; 40(1):108–13. [Epub ahead of print]. [PubMed: 21989129]
- 25. Shelbourne KD, Gray T. Minimum 10-year results after anterior cruciate ligament reconstruction: how the loss of normal knee motion compounds other factors related to the development of osteoarthritis after surgery. Am J Sports Med. 2009 Mar; 37(3):471–80. [Epub ahead of print]. [PubMed: 19059893]
- 26. Shelbourne KD, Freeman H, Gray T. Osteoarthritis after anterior cruciate ligament reconstruction: the importance of regaining and maintaining full range of motion. Sports Health. 2012 Jan; 4(1): 79–85. [PubMed: 23016073]
- 27. Takai S, Woo SL, Livesay GA, Adams DJ, Fu FH. Determination of the in situ loads of the human anterior cruciate ligament. J Ortop Res. 1993; 11:686–695.
- 28. Wright RW. Osteoarthritis classification scales: interobserver reliability and arthroscopic correlation. J Bone Joint Surg Am. 2014 Jul 16; 96(14):1145–1151. [Epub ahead of print]. [PubMed: 25031368]
- 29. Yazdi H, Moradi A, Sanaie A, Ghadi A. Does the hyperextension maneuver prevent knee extension loss after arthroscopic anterior cruciate ligament reconstruction? J Orthop Traumatol. 2016 Dec; 17(4):327–331. [Epub ahead of print]. [PubMed: 27164977]

What is known about the subject

It is known that knee hyperextension is common in an ACL tear cohort, and that this is more common in females. There are conflicting studies regarding knee HE and the risk of ACL tears. It is known that, although knee HE is common, it is not necessarily associated with increased AP laxity. There is Level 4 evidence to suggest that restoring full knee HE does not affect graft rupture risk and is associated with a lower risk of osteoarthritis at long-term follow-up. It has been shown that double-bundle ACL reconstruction (ACLR) techniques prevent recovery of full HE as compared to single-bundle ACLR techniques.

What this study adds to existing knowledge

Knee HE 5 was present in one third of our cohort undergoing revision ACLR, and is an independent risk factor for subsequent graft rupture (odds ratio = 2.12; p=0.03). Given that prior literature has provided evidence that knee HE is not a risk factor for graft rupture in primary ACLR using BTB autograft with fixation in full extension, it may be that certain graft tensioning and fixation methods are more likely to affect the risk of graft rupture in revision ACLR surgery than others. This should be the subject of future investigation, and knees with HE 5 should be evaluated separately.

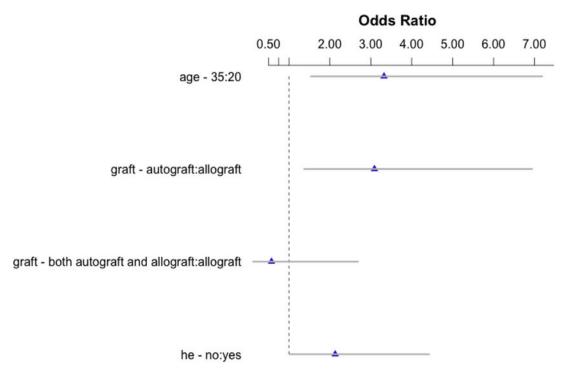


Figure 1. Odds Ratios for Age, Graft and HyperextensionMedian age was 26 (IQR 35:20). Increased odds ratio of graft failure was associated with young age (OR 3.6), allograft (OR 3.3) and HE 5 (2.12).

 Table 1

 Baseline Demographic and Clinical Characteristics at the Time of Revision ACL Reconstruction

Characteristic	HE Cohort (n=374)	Non-HE Cohort (n=771)	Entire Cohort (n=1145)
Age, y	18, 24, 34	20, 27, 35	20, 26, 35
Sex			
Male	48 (181)	62 (480)	58 (661)
Female	52 (193)	38 (291)	42 (484)
Body Mass Index	22.3, 25.1, 28.6	22.9, 25.1, 28.4	22.6, 25.1, 28.5
Revision Number			
1	86 (322)	90 (691)	88 (1013)
2	11 (43)	9 (67)	10 (110)
3 or more	2 (9)	2 (13)	2 (22)
Time from last ACLR, y	1.3, 3.0, 7.6	1.7, 4.0, 9.0	1.5, 3.7, 8.5
Graft Type			
Autograft	56 (210)	47 (361)	50 (571)
Allograft	42 (156)	51 (394)	48 (550)
Both autograft + allograft	2 (8)	2 (15)	2 (23)
Graft Source			
ВТВ	58 (216)	52 (403)	54 (619)
Soft tissue	42 (157)	47 (360)	45 (517)
Other (ie. both BTB + soft tissue)	<1 (1)	1 (6)	1 (7)
Hyperextension, degrees	5, 5, 8	0, 0, 0	0, 0, 5

Categorical values are expressed as % (n), while continuous variables are expressed as a,b,c, where "a" represents the lower quartile, "b" the median, and "c" the upper quartile. HE = hyperextension; BTB = bone-patellar tendon-bone.

ACL Graft Failures at 2 Year Follow-up

	HE group (n=12; 3%)	Non-HE group (n=22; 3%)	Combined Failure group (n=34; 3%)	Non-Failure group (n=1111; 97%)
Age, y	17, 18, 23	17, 23, 32	17, 19, 32	20, 26, 35
Sex				
Male	58 (7)	68 (15)	65 (22)	58 (639)
Female	42 (5)	32 (7)	35 (12)	42 (472)
Graft Type				
Autograft	33 (4)	36 (8)	35 (12)	50 (559)
Allograft	67 (8)	59 (13)	62 (21)	48 (529)
Both autograft + allograft	0	5 (1)	3 (1)	2 (22)

Table 2

Categorical values are expressed as % (n), while continuous variables are expressed as a,b,c, where "a" represents the lower quartile, "b" the median, and "c" the upper quartile. HE = hyperextension.