Association of Smoking With Graft Rupture After Anterior Cruciate Ligament Reconstruction

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Background: The effect of smoking on graft rupture after anterior cruciate ligament (ACL) reconstruction is not well understood.

Hypothesis: It was hypothesized that there will be no relationship between tobacco use and graft rupture after ACL reconstruction, as reflected by postoperative magnetic resonance imaging (MRI) and arthroscopic examination.

Study Design: Cohort study; Level of evidence, 3.

Methods: Included were 233 patients who received primary ACL reconstruction with hamstring tendon autograft between January 1, 2013, and December 31, 2019, and who underwent MRI evaluation at 20.2 ± 1.9 months postoperatively. The patients were categorized by smoking history into 2 groups: 39 smokers and 194 nonsmokers. The 2 groups did not differ significantly in age, sex, operative technique, preinjury Tegner score, or mean time until postoperative MRI. The primary outcome was graft rupture rate, with rupture confirmed by either arthroscopic assessment or postoperative MRI diagnosis. The secondary outcome measure was degree of graft ligamentization, evaluated by measuring the signal-to-noise quotient (SNQ) of the graft.

Results: The overall ACL graft rupture rate was 6.0%. The rupture rate was significantly higher in smokers than in nonsmokers (12.8% vs 4.6%, respectively; $P = .0498$). Smokers also had a significantly higher whole-graft SNQ compared with nonsmokers (4.7 ± 4.4 vs 3.3 ± 3.7, respectively; $P = .028$), suggesting less satisfactory ligamentization in smokers.

Conclusion: Smoking was associated with a higher risk of graft rupture of ACL reconstruction and a higher SNQ of the intact graft as shown on postoperative MRI.

Keywords: anterior cruciate ligament reconstruction; graft rupture; MRI; smoking

Anterior cruciate ligament (ACL) tear is a common orthopaedic injury, with a population-based incidence of 68.6 per 100,000 person-years in the United States.[32] A total of 129,836 ACL reconstructions were performed in 2006 in the United States.[25] The incidence increased from 61.4 per 100,000 patient-years in 2002 to 74.6 in 2014.[15] However, the graft rupture rate is significant, with an overall rupture risk of 7.9% in 10 years,[23] and the rate of revision surgery within 2 years is nearly 5%.[30] This creates significant personal and social financial burden.[35] Younger age, participation in competitive sports, and the use of allograft are known to be the risk factors for ACL graft rupture.[18,36]

The prevalence of smoking in young, active individuals in Hong Kong is 10%.[6] Tobacco use is linked with an increased rate of complications in orthopaedic surgery.[3,4,17,28,29] Smoking is associated with a 50% increase in postoperative complications in ankle fracture treatment,[28] doubling the need for an ipsilateral revision superior labral anterior to posterior repair or ipsilateral revision to a biceps tenodesis[3] and causing a 15% decrease in rate of healing in rotator cuff repair.[29] Tobacco impairs the blood flow to cutaneous tissues and increases platelet aggregation, forming thrombi in vessels and subsequently limiting soft tissue perfusion and lymphocyte delivery to the wound area.[4] In addition, the inhibition effect of nicotine on the collagen protein could increase the likelihood of connective tissue injury.[17]

A study by Cancienne et al[4] showed that tobacco use was associated with a nearly 2-fold increase in the risk of ipsilateral revision ACL reconstruction and contralateral primary ACL reconstruction. However, the study by Cancienne et al[4] was a large database study, and the authors did not capture graft ruptures that did not undergo revision. As no other study has shown the effect of smoking on ACL graft rupture, further research is needed to fully establish the correlation between smoking and ACL graft rupture.

The purpose of this study was to investigate the effect of smoking on graft rupture after ACL reconstruction. It was

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hypothesized that there will be no relationship between smoking and graft rupture after ACL reconstruction. The hypothesis was tested in a sample of patients who received primary ACL reconstruction with hamstring autograft.

METHODS

Patient Selection and Study Design

The protocol for this study was approved by our ethics committee. A retrospective, observational study was conducted for patients receiving primary ACL reconstruction with hamstring tendon autograft in the author's institution (W.P.Y.) from January 1, 2013, to December 31, 2019. All operations were conducted either by or under supervision of the senior author (W.P.Y.). Concomitant anterolateral ligament (ALL) reconstruction was performed in a subgroup of young, active male patients aged from 18 to 40 years old. After ACL reconstruction, all patients received a standardized rehabilitation protocol and were advised not to return to pivoting sport for at least 9 months after the operation. Regular follow-up was performed at 3 months, 6 months, 9 months, and 1 year, and then annually after the operation. During each follow-up, International Knee Documentation Committee (IKDC) subjective scores were applied, and ACL deficiency signs were documented. Routine postoperative magnetic resonance imaging (MRI) of the operated knee was performed at 12 to 36 months after surgery.

The inclusion criteria were patients with (1) skeletal maturity, (2) isolated ACL injury, (3) primary ACL reconstruction, and (4) ipsilateral hamstring autograft. Participants were excluded for the following reasons: (1) diagnosed with inflammatory joint disease, (2) underwent multiligament reconstruction (other than concomitant ALL reconstruction) at the time of index surgery, (3) isolated anteromedial or posterolateral bundle reconstruction, (4) unknown smoking status, (5) no postoperative MRI performed or MRI performed <12 months postoperatively (unless graft rupture was arthroscopically confirmed), (6) suffered from significant complication leading to a change in rehabilitation protocol (eg, fracture), and (7) clinical follow-up of <9 months.

Smoking Status

Smoking was defined as the act of inhaling the fumes of burning tobacco products, including cigarettes, cigars, and smoking pipes. Smokers were classified as chronic smoker, social smoker, or ex-smoker. Chronic smokers were defined as active smokers who smoked regularly on a daily basis, regardless of the number of cigarettes consumed each day. Social smokers were defined as active smokers who smoked at an irregular frequency, regardless of the number of cigarettes consumed each time. Ex-smokers were those smokers who stopped smoking before the first consultation in the author's institution (W.P.Y.), regardless of the length of time since quitting smoking and the frequency of previous smoking.

Outcome Assessment

The primary study outcome was graft rupture rate, with ruptures detected by either arthroscopic assessment or MRI. The secondary outcome was the degree of graft ligamentization as shown on MRI scans. If the patient had >1 postoperative MRI scan, the initial set was used for assessment.

Knee MRIs were taken with a 1.5-T MRI machine without applying contrast. The MRI scans performed at our institution used a 1.5-T MRI system (SignaHD; General Electric) with a knee coil (General Electric). Six sequences, including T1- and T2-weighted images in the coronal, sagittal, and axial planes were performed (matrix, 256 × 256; field of view, 16 cm; thickness, 4 mm; and space, 0.5 mm). The axial scanning of the distal femur was performed along the anatomical axis of the distal femur. All MRI images were imported into a Digital Imaging and Communications in Medicine workstation and viewer (Miele-LXIV). Sagittal MRI images of the graft were graded for the presence of edema according to the modified Ahn classification (Figure 1).[1,22] Graft rupture was defined on MRI scans as modified Ahn grade 3 (Figure 1).

For the secondary outcomes, a subgroup analysis was performed on those patients with intact graft on postoperative MRI (as defined by modified Ahn grades 1 and 2). This included 36 smokers and 186 nonsmokers. The degree of ligamentization of the ACL graft on MRI was evaluated by measuring the normalized signal-to-noise quotient (SNQ) using a previously reported protocol (Figure 2A).[1] A T2-weighted sagittal image at the region of the ACL reconstruction graft was used for measurement of SNQ. A higher SNQ value indicates less graft ligamentization.

The graft signals were measured using 2 methods. In the first method, the mean signal at the proximal, middle, and distal portions of the intra-articular portions of the graft were defined as 3 mm–diameter circular regions of interest (ROIs), and the mean SNQ per ROI was measured. The

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Ethical approval for this study was obtained from University of Hong Kong/Hospital Authority Hong Kong West Cluster (ref No. UW 22-116).

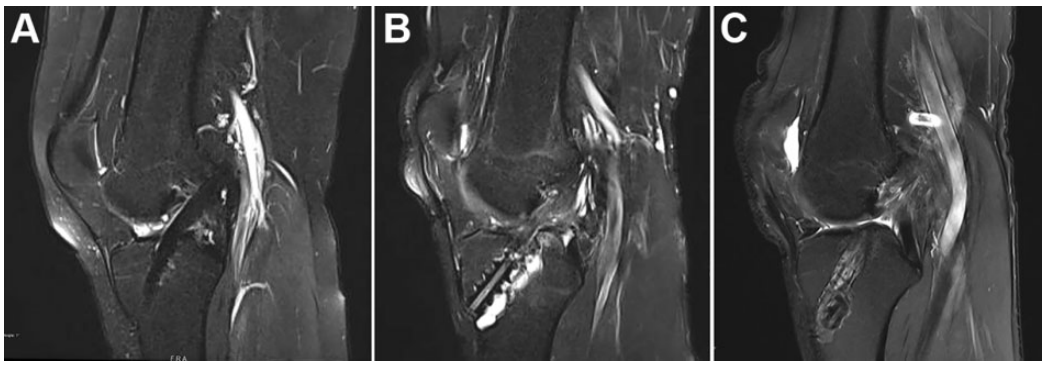

Figure 1. Modified Ahn classification on T2-weighted magnetic resonance imaging.[1,22] (A) Grade 1, near-completely to completely normal-appearing strands of hypointense graft without edema; (B) grade 2, heterogeneous graft with patchy or diffuse edema presented as hyperintensity; and (C) grade 3, no normal-appearing strands of ligament with generalized obvious hyperintensity or absence of graft.

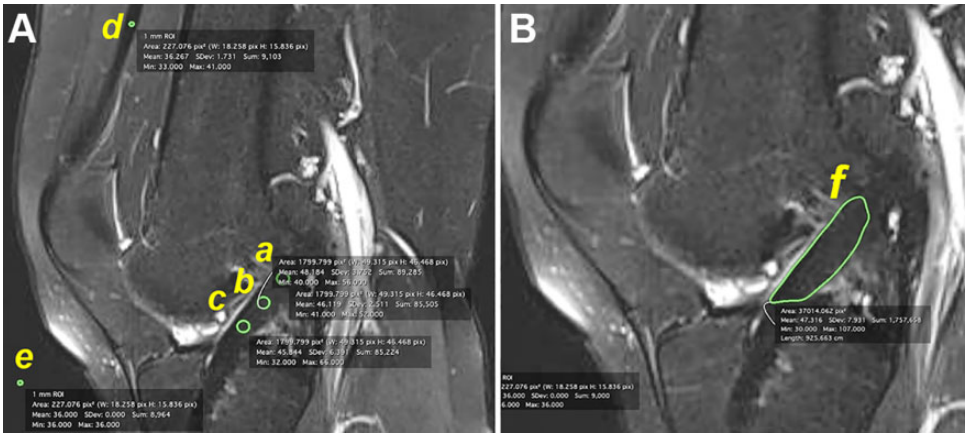

Figure 2. Postoperative sagittal T2-weighted magnetic resonance imaging of the knee showing the 2 methods of measuring graft signal-to-noise quotient (SNQ). (A) Graft signals measured at 3 regions of interest (green circles): proximal (*a*), middle (*b*), and distal (*c*) third of the intra-articular portions of the graft. Data normalized with the mean signal of the quadriceps tendon (*d*), and the background signal taken 2 cm anterior to the patellar tendon (*e*). (B) Mean normalized SNQ of the whole graft (*f*). ROI, region of interest.

data were normalized with the mean signal of the quadriceps tendon and the background (taken at $\sim$2 cm anterior to the patellar tendon) (Figure 2A). The normalized SNQ at the 3 representative regions of the graft was calculated according to the following equation.

$$SNQ = \frac{Signal\ of\ ACL\ graft}{Signal\ of\ background} - \frac{Signal\ of\ quadriceps\ tendon}{Signal\ of\ background}$$

In the second method, contour of the graft was marked, and the mean normalized SNQ per unit area of the graft was measured (Figure 2B).[2] Normalization of the signal intensity of the whole ACL graft was performed as described above.

All MRI images were reviewed by 2 individual investigators who were blinded to the clinical history, arthroscopic findings, and initial radiological written report. One investigator was a fellowship-trained orthopaedic surgeon (W.P.Y.). The second investigator was a final-year medical student (Y.C.C.). Measurements were performed twice with an interval of 1 week. The inter- and intraobserver reliabilities of the modified Ahn grades were assessed by Cohen weighted kappa (interpreted as perfect if $>$0.8, substantial if 0.61-0.8, moderate if 0.41-0.6, fair if 0.21-0.4, and none to slight if 0.01-0.2). The inter- and intraobserver reliabilities of the SNQ calculations were assessed by the intraclass correlation coefficient (ICC; interpreted as good if $>$0.75, marginal if $\geq$0.4 and $\leq$0.75, and poor if $<$0.4).

Statistical Analysis

All statistical analyses were performed using SPSS (Version 27). The hypothesis of the negative effect of smoking on graft rupture after ACL reconstruction as compared with nonsmokers was examined by use of the chi-square test. The odds ratio of graft rupture in smokers versus

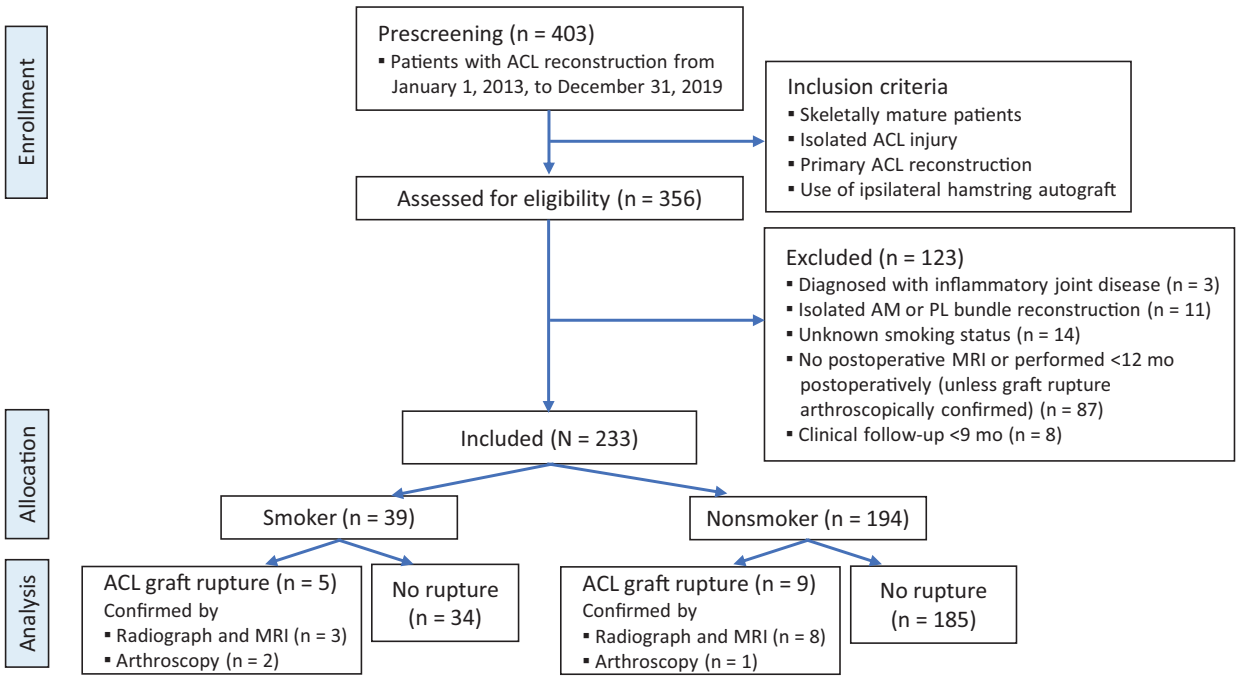

Figure 3. Enrollment and allocation of study groups. ACL, anterior cruciate ligament; AM, anteromedial; MRI, magnetic resonance imaging; PL, posterolateral.

nonsmokers was calculated. The 2-sample *t* test was used to compare the difference in the whole-graft SNQ between groups. Using the 1-sample Kolmogorov-Smirnov test, the whole-graft SNQ in smokers was compared with published data from Fukuda et al,[13] in which the SNQ of the reconstructed ACL graft reached a plateau value of 3.2 at 24 months postoperatively (reference SNQ). Statistical significance was set at $P < .05$.

We performed a power analysis based on our pilot study of 98 patients (11 smokers and 87 nonsmokers), which showed that the group 1 proportion (smokers) was 15.4% and group 2 proportion (nonsmokers) was 3.3%. Power of the proposed hypothesis test (type II error) was set at 80% and the level of significance (type I error) was fixed at 5%. Based on the chi-square statistic (*Z* test), 185 patients were required for the current study to have adequate power.

RESULTS

Out of 403 patients initially identified, 233 patients with a mean age of 27 years (range, 15-56 years) were included. Overall, 46 (19.7%) of the patients were female, 71 (30.5%) had undergone double-bundle ACL reconstruction, and 72 (30.9%) had concomitant ACL and ALL reconstruction. The preinjury Tegner score was 6.4 ± 1.5. There were 39 patients in the smokers group (10 social smokers, 23 chronic smokers, and 6 ex-smokers regardless of packs per year) and 194 patients in the nonsmokers group (Figure 3).

There was no difference between the smoker and nonsmoker groups in terms of preoperative body mass index (25 ± 3 vs 24 ± 3 kg/m^2), preinjury Tegner score (6.4 ± 1.3 vs 6.4 ± 1.5), or preoperative IKDC score (62.9 ± 13 vs 61.9 ± 15). Demographic data were comparable between smokers and nonsmokers (Table 1). Postoperative MRI was performed on 231 (99%) of the 233 patients, at an average time of 20 ± 1.9 months after index surgery.

TABLE 1
Demographic Data of Study Participants (N = 233)[a]

	Smokers (n = 39)	Nonsmokers (n = 194)	*P*
Age at inclusion, y	28.2 ± 6.4	27.0 ± 8.9	.451
Sex, male: female	35: 4	152: 42	.103
Preinjury Tegner score	6.4 ± 1.3	6.4 ± 1.5	.947
Single-bundle: double-bundle	23: 16	139: 55	.117
Isolated ACL reconstruction: concomitant ACL and ALL reconstruction	29: 10	132: 62	.436
Length of follow-up, mo	38 ± 25	42 ± 26	.365
Time from index surgery to MRI, mo[b]	19.3 ± 7.3	20.3 ± 9.7	.548

[a]Data are reported as mean ± SD or No. of patients. ACL, anterior cruciate ligament; ALL, anterolateral ligament; MRI, magnetic resonance imaging.
[b]n = 231 patients.

Graft Ruptures

There were 14 patients (6%) with graft ruptures, while 219 patients (94%) had intact grafts. A significant difference in the graft rupture rate was found between smokers and nonsmokers (12.8% vs 4.6%; $P = .0498$; χ^2 test) (Figure 4). The

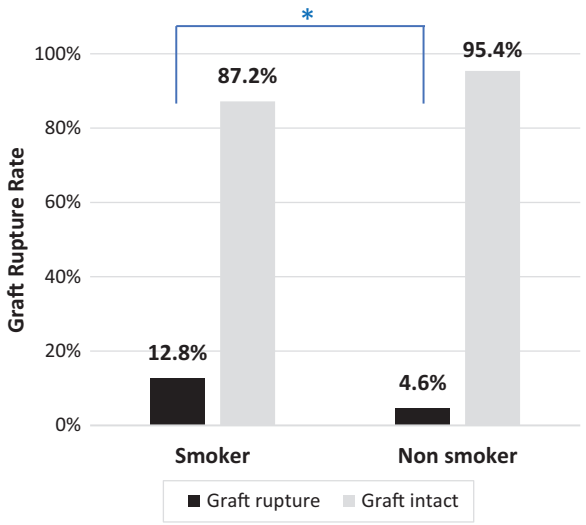

Figure 4. Graft rupture rate between smokers and nonsmokers. *Statistically significant difference ($P < .05$).

TABLE 2
Comparison Between Patients With Ruptured Versus Intact Grafts[a]

	Graft Rupture (n = 14)	Graft Intact (n = 219)	*P*
Smoking status, smoker: nonsmoker	5: 9	34: 185	**.0498**
Age at inclusion, y	26.5 ± 7.7	27.3 ± 8.6	.746
Sex, male: female	11: 3	176: 43	.870
Preinjury Tegner score	6.2 ± 1.7	6.4 ± 1.5	.677
Single bundle: double bundle	12: 2	150: 69	.175
Isolated ACL reconstruction: concomitant ACL and ALL reconstruction	9: 5	152: 67	.688
Length of follow-up, mo	39 ± 30	41 ± 25	.731
MRI from index surgery, mo	21.6 ± 10.0	20.0 ± 9.3	.542

[a]Data are reported as No. of patients or mean ± SD. Boldface *P* value indicates statistically significant difference between groups ($P < .05$). ACL, anterior cruciate ligament; ALL, anterolateral ligament; MRI, magnetic resonance imaging.

odds ratio of graft rupture in smokers versus nonsmokers was 2.76. Aside from smoking status, no other variables were significantly different between patients with ruptured versus intact grafts (Table 2).

Of the 14 graft ruptures, 9 were diagnosed on postoperative MRI scans, 3 were diagnosed arthroscopically, and 2 were diagnosed on a second MRI assessment at another institution after having reinjured their ACL. For these 2 patients, the graft was found intact (Ahn grade 2) on initial postoperative MRI. A total of 5 ruptures occurred in smokers (2 in social smokers and 3 in chronic smokers) whereas 9 occurred in nonsmokers. Among the 5 graft ruptures in smokers, 3 were diagnosed on MRI scans and 2 were diagnosed arthroscopically; among the 9 graft ruptures in nonsmokers, 8 were diagnosed on MRI scans and 1 was diagnosed arthroscopically. Regardless of the method of diagnosing graft rupture, there was no difference in the time of graft rupture between smokers and nonsmokers (22 ± 15 vs 20 ± 7 months, respectively).

For the patients whose diagnosis of graft rupture was made arthroscopically, 2 had reinjured their ACL before the postoperative MRI examination and had undergone diagnostic arthroscopy. The third patient reinjured his ACL after the postoperative MRI showed an intact graft, and the diagnosis of graft rupture was made during diagnostic arthroscopy.

At the most recent follow-up, 29 out of the 233 patients had a contralateral ACL injury: 4 of the patients were smokers and 25 were nonsmokers. No difference was found between the smokers and nonsmokers in terms of contralateral ACL tear.

TABLE 3
SNQ Overall and Compared Between Smokers and Nonsmokers[a]

SNQ	Overall (n = 222)	Smokers (n = 36)	Nonsmokers (n = 186)	*P*
Proximal third	2.63 ± 4.22	3.89 ± 5.82	2.38 ± 3.81	.093
Middle third	2.68 ± 3.46	3.76 ± 4.47	2.47 ± 3.20	.069
Distal third	3.06 ± 3.98	4.18 ± 4.99	2.84 ± 3.73	.155
Whole graft	3.51 ± 3.86	4.65 ± 4.42	3.28 ± 3.71	**.028**

[a]Data are reported as mean ± SD. Boldface *P* value indicates statistically significant difference between groups ($P < .05$). SNQ, signal-to-noise quotient.

Two-Year Tegner and IKDC Scores

For the 219 patients without graft rerupture, significant improvement in Tegner and IKDC scores were observed in both smokers (n = 34) and nonsmokers (n = 185). The 2-year Tegner and IKDC scores were 5 ± 1.9 and 85.2 ± 11 for smokers and 5.4 ± 1.9 and 87.4 ± 11 for nonsmokers, respectively, with no significant difference between the groups.

Graft Signal and SNQ

Among the 231 patients with available postoperative MRI, 148 (64%) were classified as having Ahn grade 1 signal intensity, 74 (32%) were classified as grade 2, and 9 (4%) were classified as grade 3 (ie, graft rupture). The SNQ was calculated on the 222 patients with intact graft shown on MRI (ie, Ahn grades 1 and 2) (Table 3). Smokers had a significantly higher whole-graft SNQ (4.65 ± 4.42) than nonsmokers (3.28 ± 3.71), suggesting suboptimal ligamentization in smokers ($P = .028$; independent *t* test) (Table 3). When comparing the SNQ of smokers with that of the "normal" ACL graft at 24 months after surgery (SNQ, 3.2),[13] the SNQ of smokers was significantly higher ($P < .001$; 1-sample Kolmogorov-Smirnov test).

Intra- and Interobserver Reliability

The intraobserver reliability of the SNQ measurements was good for the whole graft (ICC, 0.85) and the distal third

(ICC, 0.83) but was marginal for the proximal third (ICC, 0.55) and middle third (ICC, 0.67). The intraobserver reliability of Ahn grading was almost perfect (Cohen weighted $\kappa = 0.82$).

The interobserver reliability of the SNQ measurements was good for the whole graft (ICC, 0.96), distal third (ICC, 0.91), and middle third (ICC, 0.84), but was marginal for the proximal third (ICC, 0.74). The interobserver reliability of Ahn grading was substantial (Cohen weighted $\kappa = 0.70$).

DISCUSSION

The study results rejected our null hypothesis that smoking would have no effect on graft rupture after ACL reconstruction. Smoking was associated with an increased risk of ACL graft rupture ($P = .0498$). Smokers had a nearly 3-fold higher risk of ACL graft rupture when compared with nonsmokers (odds ratio, 2.76). In addition, the MRI analysis of intact grafts indicated that smokers had inferior graft ligamentization compared with both nonsmokers (whole-graft SNQ, 4.65 ± 4.42 vs 3.28 ± 3.71, respectively; $P = .028$) and with normal SNQ levels ($P < .001$).[13]

Our findings add further evidence to the association between smoking and ACL graft rupture. The only previous study on this topic that we are aware of was by Cancienne et al,[4] who used data retrieved from a national insurance database and reported a doubled rate of subsequent ipsilateral revision ACL reconstruction surgery and contralateral primary ACL reconstruction in smokers. Our findings echoed those of Cancienne et al,[4] by showing that smokers were associated with a 2.76-fold higher risk of ACL graft rupture when compared with nonsmokers. The reason leading to the revision ACL reconstruction (ie, whether the revision was due to graft rupture or other causes, such as persistent knee instability) was not reported in the Cancienne et al[4] study, likely because the database was initially designed for insurance purposes. In addition, those authors did not identify patients with graft rupture who did not undergo revision ACL reconstruction.

Effects of smoking on the functional result after ACL reconstruction have been supported by previous publications. Cigarette smoking was associated strongly with occupational disability after discharge in initial ACL injuries,[9] with a lower postoperative activity level,[10] suboptimal subjective functional assessment, and poorer IKDC score.[19,20,34]

The effect of smoking on ACL graft rupture is postulated to be related to a reduction in soft tissue perfusion and vascularization, which delays ligamentization.[4] In the current study, this was demonstrated as significantly higher whole-graft SNQ on postoperative MRI in smokers versus nonsmokers ($P = .028$). A few studies have shown a correlation between the degree of ACL graft remodeling and MRI signal intensity.[27,37] Weiler et al[37] showed a significant association between SNQ and the mechanical strength with degree of neovascularization of the primary ACL reconstruction graft in sheep. In human, Muramatsu et al[27] postulated that the increased enhancement in MRI indicated vascularization in immature ACL graft and the reduction in MRI signaling indicated a completed revascularization of the graft. However, on top of the issue of ligamentization, there are other factors possibly contributing to persistently high signal of the graft and increased SNQ after ACL reconstruction. These include repeated injury (with or without graft rupture), graft impingement, infection, and so forth.

Although there is no clear time frame for the ligamentization stages of an ACL graft,[7] most studies have found that ligamentization is complete at 24 months.[8,13,38] Dong et al[8] reported that the graft ultrastructural maturation could be reached at 24 months, with no significant further improvement at 39 months. Zdanowicz et al[38] demonstrated an initial increase in SNQ until 9 months, then SNQ continued to decrease until the final follow-up time point of 24 months. Fukuda et al[13] showed that, 24 months after index surgery, the SNQ of ACL graft would generally reach a plateau level of 3.2 to 3.4.

These previous results correlated with our findings that smokers had a significantly higher SNQ compared with both nonsmokers and with normal SNQ levels at 24 months after ACL reconstruction, indicating a delayed remodeling and ligamentization process. The cumulative effect of smoking on ACL reconstruction was shown to be halved in patients who had stopped smoking for 6 to 8 weeks preoperatively.[26] Therefore, it is reasonable that smoking cessation should be recommended preoperatively to reduce the ACL graft rupture rate. Smoking should be avoided up to 2 years after ACL reconstruction to minimize any impact on delayed graft ligamentization and graft rupture.

Smoking is associated with other risky behaviors, such as drug addiction and alcoholism.[14] It is reported that patients suffering from alcoholism and other psychoactive substance abuse are more prone to musculoskeletal injury.[33] This may negatively affect graft healing after ACL reconstruction.

The results of this study can be generalized to other patients who receive primary ACL reconstruction with ipsilateral hamstring autograft for the treatment of isolated ACL deficiency. However, it may not be applicable to patients having ACL reconstruction using graft other than hamstring autograft (eg, bone–patellar tendon–bone graft, quadriceps tendon graft, allograft, etc), revision surgery, multiple ligament injury, or concomitant inflammatory joint disease.

Limitations

One limitation of the current study was its retrospective nature, causing an incompleteness of data that could lead to an unclear definition of smoking history, such as the amount and the medium of smoking. Therefore, we could not comment on the effect of the amount of smoking on the ACL graft rupture rate. In addition, although data concerning ACL reconstruction were collected prospectively, comprehensive data on all important health- or lifestyle-related information were not available. Although it was important to know how long before the operation the former smoker had stopped smoking, and whether nonsmokers started smoking in the postoperation period, this information was

unavailable, introducing potential bias to the results. Furthermore, unlike in a prospective experimental trial, we could not control the number of participants in each group. To improve the completeness of information, a prospective case-control study should be conducted.

Second, we did not investigate other factors that have been shown to affect the graft rupture (eg, type of pivoting sport,[31] time of return to sport,[21] preoperative knee laxity,[12] and posterior tibial slope).[12] Therefore, we could not fully demonstrate the correlation of higher activity level, early return of sports, high-grade preoperative knee laxity, and steeper posterior tibial slope with ACL graft rupture, as shown in previous studies.[30] A third limitation was that the data in the current study were statistically fragile (fragility index, 0). Because we recruited the minimal number of patients that would provide enough power to this study, the effect of smoking on ACL graft rupture in the current study was only marginally statistically significant. A larger sample size could reject the null hypothesis with a greater power and reduce the fragility of the data. Another limitation of this study was the high rate of patients who did not undergo postoperative MRI, especially for smokers. Among the 87 patients who defaulted the postoperative MRI, 35.6% (31 patients) were smokers, which was much higher than the proportion of smokers included in our study (16.7%; 39 patients). This significant loss of follow-up in smokers could introduce bias in comparing the graft rupture rate in smokers and nonsmokers.

Fourth, the sample of the current study included 161 isolated anterior cruciate reconstructions and 72 combined ACL and ALL reconstructions. The inclusion of concomitant ACL-ALL reconstruction may have introduced bias to this study. However, whether concomitant ALL reconstruction offered additional benefit in reducing graft rupture is still under debate. Recent randomized controlled trials of short- to midterm follow-up suggested that ALL reconstruction is protective against graft rupture.[12,16] However, long-term clinical trials with follow-up of >10 years did not support this hypothesis.[5,24] We are not aware of any research studying the impact of smoking on graft rupture in patients receiving concomitant ACL-ALL reconstruction.

Fifth, other factors that may potentially affect SNQ (eg, impingement, repeated injury, partial tear, etc) were not evaluated in the current study. Potential bias was present. As a sixth limitation, tobacco consumption was limited to smoking. Impact due to other forms of tobacco consumption, such as tobacco chewing or sniffing of tobacco snuff, were not investigated. Finally, unlike previous studies,[8,11,22] the degree of ligamentization of ACL graft was examined with MRI imaging but not with histological investigation on the biopsy sample; thus, a quantitative histology result on the degree of ligamentization could not be fully obtained. However, given the invasiveness of biopsy sampling, this could further reduce the sample size, with decreased efficiency of statistical analysis, and the randomization of biopsy sampling could not conclusively represent the degree of ligamentization of the entire graft. Therefore, biopsy sampling was not considered.

Despite these limitations, we hope this can serve as an initial study and trigger the interest of the orthopaedic fraternity to further investigate this important health issue.

CONCLUSION

The present study compared the ACL graft rupture rate and the degree of ligamentization in smokers and nonsmokers. Smoking was associated with an increased risk of graft rupture as well as a higher SNQ in the intact graft on postoperative MRI scan at a mean of 20.2 months after index surgery.

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