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Lateral ulnar collateral ligament (LUCL) reconstruction for the treatment of recalcitrant lateral epicondylitis of the elbow: a comparison with open débridement of the extensor origin

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Background: Based on the controversy over whether the extensor tendon is the only lesion of lateral epicondylitis of the elbow and numerous reports of concomitant lateral collateral ligament involvement, potential damage to the lateral collateral ligament complex should be considered for the treatment.

Methods: About 25 elbows in 23 patients (débridement group) and 22 elbows in 20 patients (reconstruction group) who were diagnosed with lateral epicondylitis and had an average of 22 months of symptoms revealing anatomical lesion on MRI were included. The capitellum-sublime tubercle-radial head (CSR) angle was measured on both sides preoperatively, and the visual analog scale (VAS) and Mayo elbow performance score (MEPS) were measured over 12 months, postoperatively.

Results: The initial preoperative mean VAS was statistically significant with 4.6 in the débridement group and 6.5 in the reconstruction group ($P < .05$). Postoperative VAS was continuously decreased in both groups with no significant difference at each assessment period ($P < .05$) but showed more rapid improvement in the reconstruction group compared with the débridement group. For MEPS, the reconstruction group showed significant improvement during the follow-up periods, and at the final follow-up MEPS, 3 cases in the débridement group and 0 cases in the reconstruction group showed a poor result, which was considered as surgery failure. The CSR angle of the affected side (7.2 ± 1.9) was significantly larger than that of the normal side (3.6 ± 1.5) ($P < .05$) in the reconstruction group. Increased CSR by more than 5 degrees was identified as a significant predictive indicator for potential concomitant ligament insufficiency (area under curve = 0.875, $P < .001$) showing 80.9% of the sensitivity, 82.1% of the specificity.

Conclusions: In the surgical treatment of recalcitrant lateral epicondylitis, lateral ulnar collateral ligament reconstruction added to the débridement of extensor origin may provide better results for the patients with suspicious lateral ligament insufficiency or failed previous surgery.

Lateral epicondylitis is one of the common clinical conditions in the adult population with a significant prevalence rate of 1-3%.[5,43] Conservative therapy is recommended, as more than 80% of patients show improvements with conservative therapy within 1 year. However, about 10% of patients experience recalcitrant states that do not respond to various combinations of conservative treatment.[5,14] In such cases, operative treatment is considered.[5,10,28] With operative treatment, 80%-90% of patients showed improvement regardless of the type of operation, but some of them are still refractory to an operation.[23] The main pathology of lateral epicondylitis involves degenerative changes due to the accumulation of chronic microtrauma at the origin of the extensor carpi radialis brevis (ECRB). Facilitating the healing process of the normal tendon by removing immature degenerative granulation tissues resulting from the failure of healing arising from repetitive microtrauma has been widely accepted as a major component of operative treatment.[19,27,38] However, there has been a continuing

Inje University Haeundae Paik Hospital Institutional Review Board approved this study (HPIRB 2017-08-010-001).

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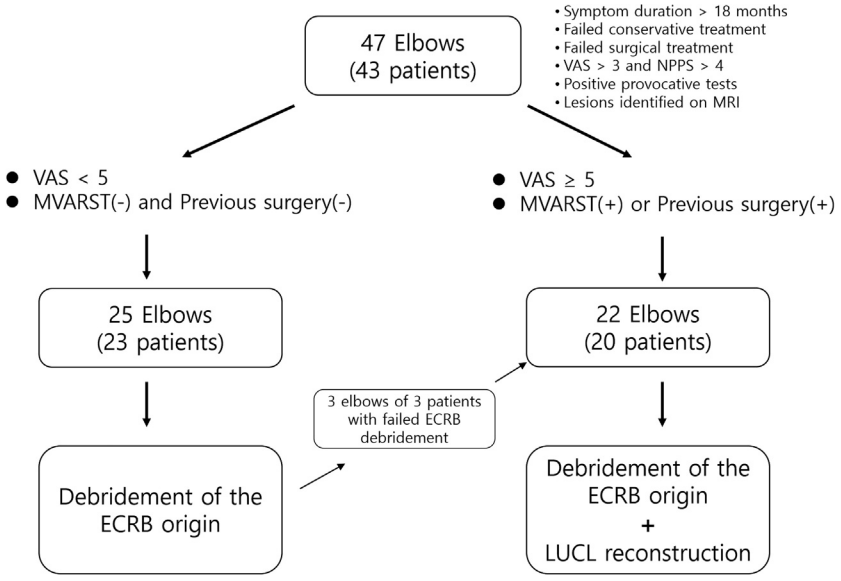


Figure 1 The flowchart for the patient selection and clinical process. *MVARST*, moving varus stress test; *NPPS*, Nirschl pain phase scale; *ECRB*, extensor carpi radialis brevis; *LCLC*, lateral collateral ligament complex; *VAS*, visual analog scale.

question as to whether ECRB damage is the only lesion in the lateral epicondylitis. With the recent advances in radiology, abnormalities of the lateral ulnar collateral ligament (LUCL), accompanied by ECRB involvement, have been continuously reported.[4,18,36] The main pathogenesis of lateral epicondylitis is the torque injury caused by eccentric contraction of the ECRB and varus loading, as in the backhand stroke in tennis.[40,41] In addition to such strenuous load, in daily life, gravitational varus force due to the weight of an object in the hand tenses the lateral collateral ligament complex (LCLC) of the elbow joint.[31] There have also been reports of LUCL damage showing macroscopic instability accompanied by lateral epicondylitis.[13,17] Based on these facts, the authors hypothesized that a type of lesion in which ECRB damage extends to the LCLC attachment might exist in the disease group commonly referred to as lateral epicondylitis. This type of LCLC injury could be a micro- or macro-tear of the ligaments including LUCL that has not yet progressed to apparent varus or posterolateral rotatory instability (PLRI) of the elbow. For this study, based on the concept of restoring the biomechanical function of the LUCL by soft tissue augmentation,[12,32] LUCL reconstruction accompanied by débridement of ECRB was carried out and evaluated in patients who have recalcitrant lateral epicondylitis with high symptom level associated with LCLC involvement.

Subjects and methods

This study was conducted in the form of a retrospective clinical study and approved by Inje University Haeundae Paik Hospital Institutional Review Board (HPIRB 2017-08-010-001). Between March 2011 and December 2018, 76 patients with recalcitrant lateral epicondylitis underwent extensor débridement or LUCL reconstruction in addition to débridement in a single institution under the care of a single elbow joint specialist surgeon. Of these, 43 patients (men : women = 24 : 19) excluding those over 65 years of age, those with periarticular degenerative changes on the radiography, those with symptoms of the ipsilateral shoulder and wrist, and those who were not followed up for more than 1 year were selected for the study. The study subjects were limited to those who experienced symptoms for 18 months or longer (mean 22.3 months, range 18-37 months), those who underwent a various combination of conservative treatments, including steroid injections (mean 2.6 times, range 1-7 times), those with a 3 or greater as per the visual analog scale (VAS),[35] those with a phase 4 or more regarding their limitations in daily life as per the Nirschl pain phase scale,[26] tenderness confined to lateral epicondyle, a positive resisted wrist extension test,[34] those who were diagnosed with lateral epicondylitis and underwent surgery but had no improvement for 18 months or more. Of 43 patients, we performed LUCL reconstruction accompanied by ECRB débridement for the patients meeting the following three criteria and classified as the reconstruction group: (1) those who have the positive moving varus stress test (MVARST) designed by one of the authors (Y. Kim), (2) those who were observed to have lesions of footprint of ECRB and LUCL at the same time on T1- and T2-weighted coronal MRIs, (3) those who had pain of 5 or greater by VAS. The other patients who showed the lesion limited to ECRB on the same MRIs or did not meet the conditions above were conducted ECRB débridement alone regardless of their VAS score, were classified as the débridement group (Fig. 1). Care was taken not to include patients with concomitant symptoms due to other diseases such as medial epicondylitis, plica syndrome, radial tunnel syndrome, calcific tendinitis of the elbow in both groups. Four cases with definite PLRI with the positive lateral pivot shift test[29] under anesthesia were excluded.

The débridement group included 25 elbows of 23 patients (men : women = 13 : 10, mean age 52.7 years) and the reconstruction group included 22 elbows of 20 patients (men : women = 11 : 9, mean age 49.4 years). For the débridement group, all patients underwent primary surgery. For the reconstruction group, 5 elbows of 5 patients who underwent ECRB débridement in different hospitals and 3 elbows of 2 patients who underwent ECRB débridement in our hospital by the same surgeon but showed no improvement of symptom were included as revision surgery.

Clinical scores: visual analog scale and Mayo elbow performance score

The VAS was measured immediately before surgery, and the ratios of change in pain intensity (%) were investigated in accordance with the patient's visit intervals. The perception of pain is a

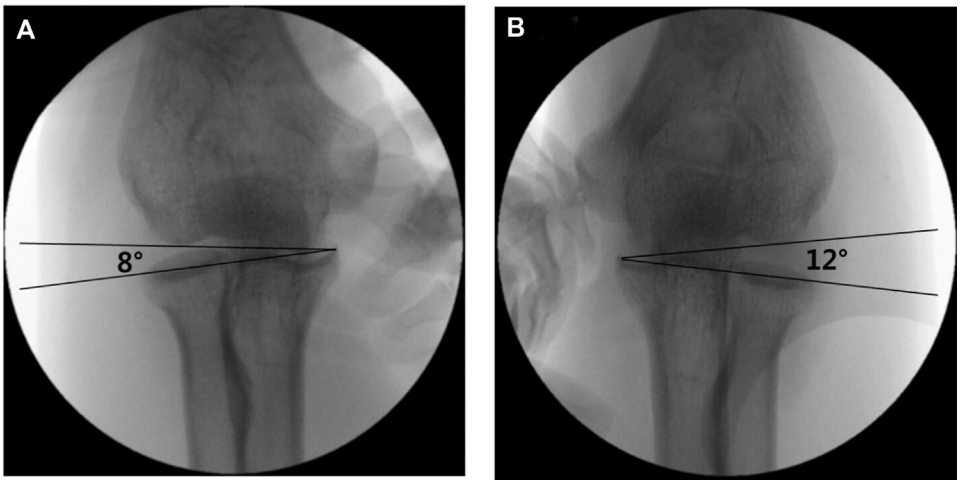


Figure 2 The captellum-sublime tubercle-radial head (CSR) angle in the normal (**A**) vs. affected (**B**) elbow in a 46-year-old man diagnosed with the lateral epicondylitis from the study group.

subjective measure influenced by the individual or cultural background, so the number expressed in VAS is inevitably relative. We asked the patient, "what is the current joint pain compared to the worst pain experienced over the entire lifetime" and recorded the changes in this pain level before and after treatment, so the degree expressed in numbers in the present study might differ from that of other researchers.

The Mayo elbow performance score (MEPS)[9] was surveyed at the same time. For MEPS, in the evaluation of lateral epicondylitis, we believe that it is not suitable to evaluate the change of fine symptoms as the evaluation shows changes only in the pain issue with a large increment between grades, which makes it difficult to analyze the statistical difference. However, months after the surgery, the patients showed a pattern of improvement from poor/fair to good/excellent. If the case corresponded to the “poor” in the final follow-up, it was considered as a failed operation.

Lateral joint opening: capitellum-sublime tubercle-radial head angle

Under general anesthesia, varus stress radiography was taken on both sides with the elbow flexed 15° and the forearm fully supinated under C-arm fluoroscopy by manipulation[20,24] to measure the capitellum-sublime tubercle-radial head (CSR) angle representing lateral opening of the elbow joint. The CSR angle was defined as the angle at the sublime tubercle of the coronoid process between the line that is tangential to the distal margin of the capitellar joint surface and the line that bisect the articular dish of the radial head (Fig. 2).

The lateral joint opening in millimeters could be used as an indicator of subtle varus instability[24], but it is a relative measure that can be affected by the patient's physique. For example, even in the cases of identical CSR angle, in a patient with large mediolateral width of the joint, the lateral opening may be exaggerated compared with a patient with small joint. Our method has the disadvantage that the degree of opening of the joint may vary depending on the load of the examiner because it is not performed with a predetermined force using an instrument such as Telos or dynamometer. However, such a drawback can be overcome by comparing it with the normal side and performing by the same examiner. We believe that it is possible to provide the sensitivity to detect soft tissue attenuation only by checking the lateral opening of the joint that is clearly distinguished from the normal side under a physiological load such as manual manipulation.

Figure 3 Moving varus stress test (MVARST) of the elbow.

Physical examination: moving varus stress test

The patient was seated on a stool and the hand is turned back to the spine. The surgeon grabbed the patient's thumb with his or her hand and pulled so that varus force could be exerted on the elbow joint. The forearm should be placed in pronation and the wrist should be in neutral or slight extension to avoid unintentional ECRB tension (Fig. 3). The elbow joint alternated between flexion and extension ranging from 45 to 90 degrees, and the patient was assessed as positive when the patient complained of pain at a specific flexion position.

This method is a mirror image test of a moving valgus stress test[30], which is a widely known test to confirm the attenuation injury of the medial collateral ligament. This can be understood as a type of modified varus stress test, which is a method designed to provoke pain by selectively and stably applying the varus stress to the lateral collateral ligament complex.

Generally, applying the varus stress or moving shear stress while facing a patient without fixing their shoulder makes it difficult to perform a consistent and stable test, as there is no stable fulcrum due to internal rotation of the shoulder joint. This method enables the stable and consistent application of load to the lateral collateral ligament by applying moving shear stress to the elbow joint with the patient’s arm placed on the back and shoulder joint

fixed at the end point of the internal rotation. The purpose of this test is not to detect rotational instability such as PLRI, but to load operable varus tension on the lateral collateral ligament complex including LUCL, so it is not necessary to hold the forearm in supination like that of the lateral pivot shift test.[29]

Operative procedures and postoperative rehabilitation

On the lateral aspect of the elbow, along the line assumed to be the path of the anconeous tendon, a 5-cm oblique incision was made from the top of the lateral epicondyle, and the fascia of the common extensor was exposed. The locations of ECRB, extensor digitorum communis (EDC), and extensor carpi ulnaris (ECU) were confirmed by palpating the apex of the lateral epicondyle, and the anconeus tendon was confirmed. In the case of the débridement, using the Kaplan approach,[7] an incision was made along the fiber of tendon between the ECRB and the EDC, and the ECRB attachment was selectively elevated off the bone surface to remove the disarrayed tissues. Care was taken not to damage EDC / ECU complex which is confluent to radial collateral ligament (RCL)[39] to avoid iatrogenic injury of the LCLC. The bone surface was curetted, and 6-7 perforations were made using a 1.6-mm K-wire. The ECRB was repaired to the EDC and ECU complex with a side-to-side fashion with absorbable suture material.[28]

In the case of reconstruction, in addition to the débridement procedures, using the Kocher approach, [7] the interval between the anconeus muscle and LUCL was developed to reach the supinator crest of the ulna taking care not to damage the anconeus tendon. On the supinator crest, two 3.2mm drill holes were drilled by forming a bone bridge of at least 1cm along with the footprint of the LUCL. Without opening of the joint capsule or LCLC, the capitellum and radiocapitellar joint were identified and imaginary circumference of the capitellar hemi-sphere was drawn by palpation on the posterolateral joint capsule to determine the geometric center of the rotation of the elbow. A 4.5mm bone tunnel was formed at the center of the capitellar circumference below the lateral epicondyle of the humerus that matches the center of the rotation. If the humeral tunnel is drilled medially adjacent to the humeral insertion of the anconeus tendon, the graft can be easily positioned in the isometric point. The anconeus muscle has a consistent anatomical relationship with LUCL and is well known as a dynamic stabilizer, “the dynamic LUCL” that contributes to posterolateral stability.[1,22,33] Therefore, in the posture where the elbow is bent about 90 degrees, using the anconeus tendon as a guide leading to the humeral insertion of the LUCL and probing the posterolateral joint capsule through the Kocher interval to refer to the imaginary circumference of the capitellar hemicircle and the location of the radiocapitellar joint, the isometric point of the humeral tunnel can be determined without difficulty.[6,46] Additional two small holes were formed in the posterior cortex of the humerus using a 2.0-mm K-wire in a Y-shape toward the humeral tunnel. The semitendinosus allograft tendon was split to make it to a thickness sufficient to barely pass through a 3.5 mm hole and pulling threads were made via whipstitches on one end so that a loop was formed by first passing the bone tunnel of the ulna. The length of the tendon was adjusted with reference to the depth of the humeral tunnel, and another pulling thread was made on the other side in the same manner. The LUCL was reconstructed by docking both ends of the graft into the humerus,[2,16,42] and 2 or 3 yoke stitches were formed around the graft to adjacent soft tissues including the anconeus tendon and the ECU origin (Fig. 4).

All patients in both groups maintained a long arm splint at 45 degrees of elbow flexion to avoid tension on the graft[15] for 1 week after surgery until the swelling and pain subsided. For the next 2 weeks, in a hinged brace, the range of motion was gradually increased by the turn-buckling method[24] while limiting not to bend over 90 degrees of elbow flexion. Full range of motion was allowed for the following 3 weeks in the brace. After 4-6 months, patients were allowed to return to the complete level of daily living, work, and exercise.

Results

Captellum-sublime tubercle-radial head angle

Under general anesthesia, the CSR angle (in °, degrees) was measured in the débridement group with a mean of 3.5 ± 1.2 (range 1 ~ 6) on the normal side and 5.4 ± 1.9 (range 2~10) on the affected side; and in the reconstruction group with a mean of 3.6 ± 1.5 (range 2~8) on the normal side and 7.2 ± 1.9 (range 3~12) on the affected side. The normal side measurement values for patients with both sides affected were replaced by the mean values of patients with one side affected from both groups for statistical analysis. In the débridement group, there was no statistical difference between the normal side and the affected side (paired t-test, $P = .07$); whereas in the reconstruction group, the affected side showed a statistically significant larger angle compared with the normal side (paired t-test, $P = .01$).

The accuracy of the CSR angle as a predictive indicator for concomitant ligament insufficiency in all patients in both groups was investigated by calculating the area under curve value using the receiver operating characteristic curve (Fig. 5). As a result, when CSR values were divided into two based on 5 degrees, it was found to be significant as a predictive indicator for ligament insufficiency (area under curve = 0.88, $P < .001$) by dividing the patient group into two distinct normal distributions. Sensitivity, specificity, positive predictive value, and negative predictive value are 80.9%, 82.1%, 84.4%, and 78.0%, respectively, when the optimal cutoff value of CSR is $\geq$ 5 degrees.

Visual analog scale and Mayo elbow performance score

The mean preoperative VAS was 4.5 ± 0.8 (range 3 ~ 7) in the débridement group and 6.5 ± 1.1 (range 5 ~ 9) in the reconstruction group, showing a statistically significant higher value in the reconstruction group ($P = .01$, independent t-test). This statistical difference is thought to be due to the result of deliberately assigning patients with VAS scores greater than 5 to the reconstruction group when dividing the patient group. The VAS significantly changed during the five assessment periods ($P < .001$); however, this change was not affected by group ($P = .759$). The interaction between group and time was also statistically significant ($P < .001$). Post hoc analyses revealed that this significant improvement in VAS stemmed from a significant improvement in both groups. The VAS continuously decreased in both groups and shows more rapid recovery in the reconstruction group than the débridement group (Fig. 6).

The MEPS had a mean value of 68.8 ± 9.2 (range 55-85) points in the débridement group with 5 poor elbows, 15 fair elbows, and 5 good elbows. In the reconstruction group, there were 8 poor elbows, 11 fair elbows, and 3 good elbows, with a mean value of 68.2 ± 9.9 (range 50-85).

In the case of the débridement group, 3 elbows of two patients experienced operation failure indicating a 13% failure rate, and all of them were included in the reconstruction group and ultimately underwent LUCL reconstruction. However, in the reconstruction group, no cases of operation failure were observed. The MEPS significantly changed during the five assessment periods ($P < .001$), and this change was affected by group ($P = .048$). The interaction between group and time was also statistically significant ($P = .031$).

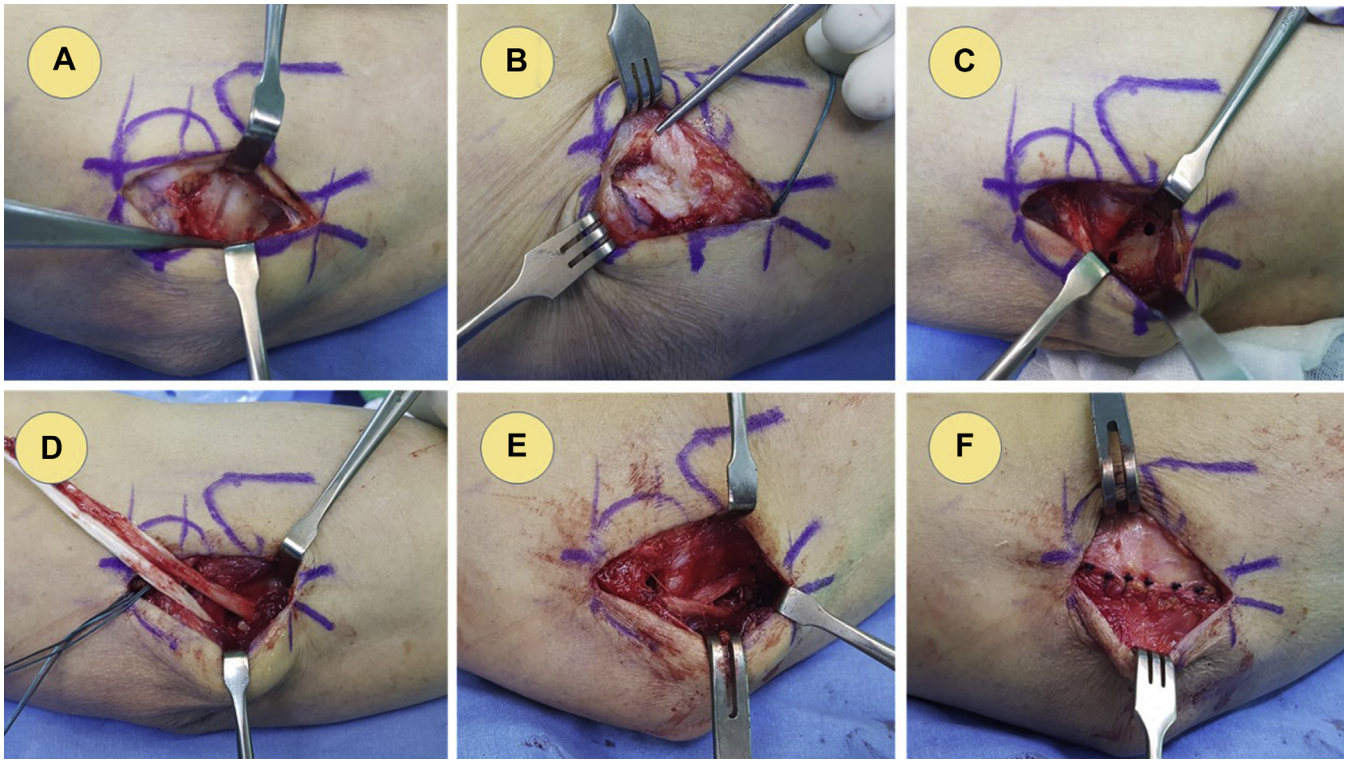
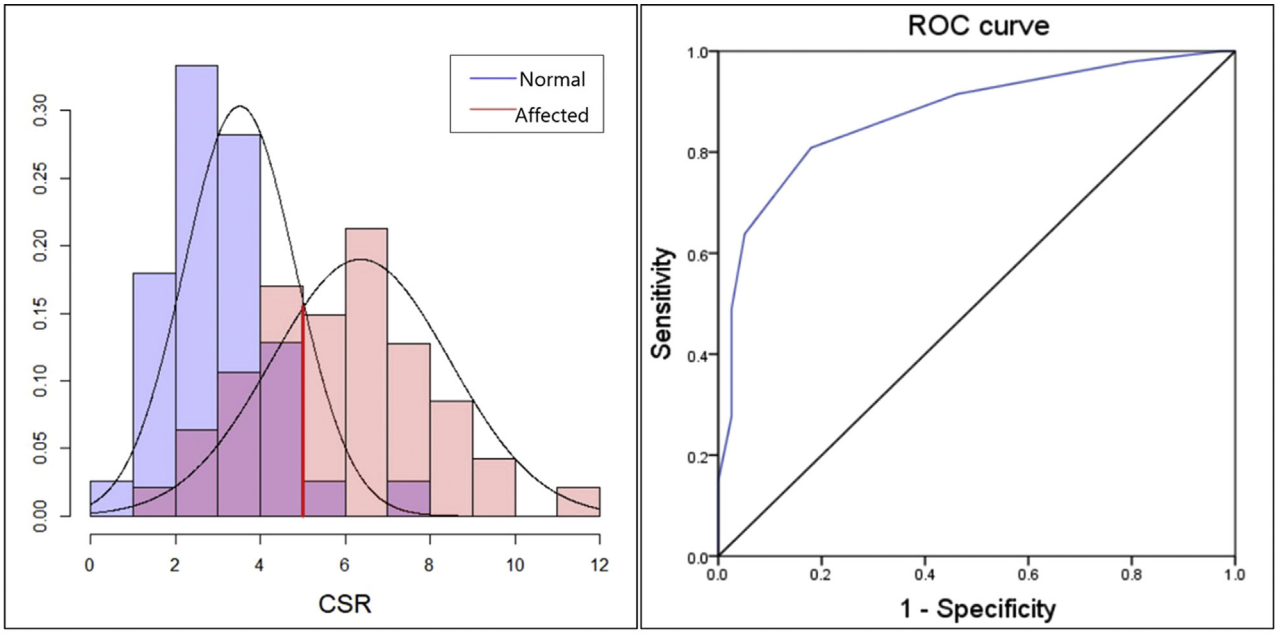
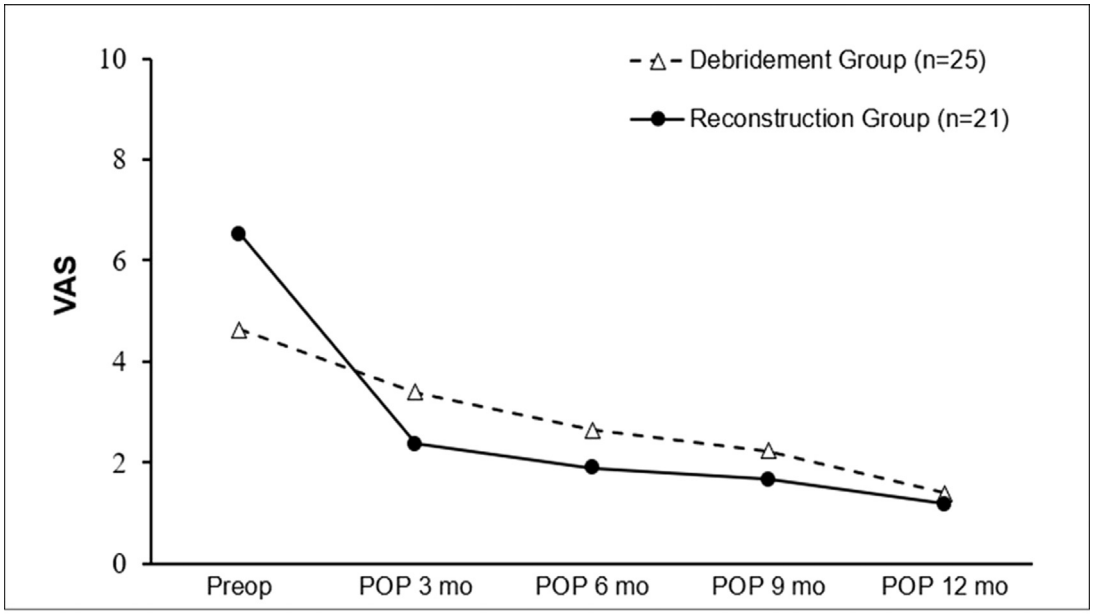


Figure 4 Surgical procedure: (**A**) development of the Kocher interval, (**B**) débridement of ECRB origin via the incision of Kaplan approach, (**C**) preparation of the ulnar tunnels through Kocher interval, (**D**) placement of a graft looping through the ulnar tunnels, (**E**) docking both ends of the graft into the humeral tunnel, (**F**) repair of the ECRB to the EDC in the side-to-side fashion. *ECRB*, extensor carpi radialis brevis; *EDC*, extensor digitorum communis.



Variable	Cut-off Value	Group		Cut-off Value	AUC				Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
		Affected	Normal		AUC	95% Confidence Interval	Standard Error	p value				
CSR	≥ 5	38	7	≥ 5	0.875	0.799-0.950	0.038	<.001	80.9%	82.1%	84.4%	78.0%
	< 5	9	32									
	total	47	39									

Figure 5 Histogram of CSR angle for normal and affected side with reference value 5 (left) and receiver operating characteristic (ROC) curves of CSR angle to predict potential lateral ligament insufficiency in lateral epicondylitis patients (right). *CSR*, capitellum-sublime tubercle-radial head.



(Value: Mean ± SD)

VAS	Group			Analysis for repeated measures	
	A. Debridement (n=25)	B. Reconstruction (n=22)	p	Source	P value
Preop	4.46±0.81 a[1]	6.52±1.12 a	<.001[3]	**Group**	.759[4]
POP 3 mo	3.40±1.78 b	2.38±1.28 b	.034[2]	**Time**	<.001[4]
POP 6 mo	2.64±2.00 bc	1.90±1.04 c	.209[3]	**Group x Time**	<.001[4]
POP 9 mo	2.24±2.28 c	1.67±1.28 bcd	.563[3]		
POP 12 mo	1.40±2.33 d	1.19±1.21 d	.485[3]		

[1] Values are the mean ± SD and Bonferroni's post-hoc test was used for multiple comparisons between each time points. Means with different scripts are different from each other (P<0.05).

[2] P values were derived from independent t-test.

[3] P values were derived by Mann-Whitney's U test.

[4] P values are derived from an ANOVA with repeated measures with a Greenhouse-Geisser correction was used when sphericity was not assumed.

Shapiro-Wilk's test was employed for test of normality assumption.

Figure 6 Changes in VAS during the follow-up period. *VAS*, visual analog scale.

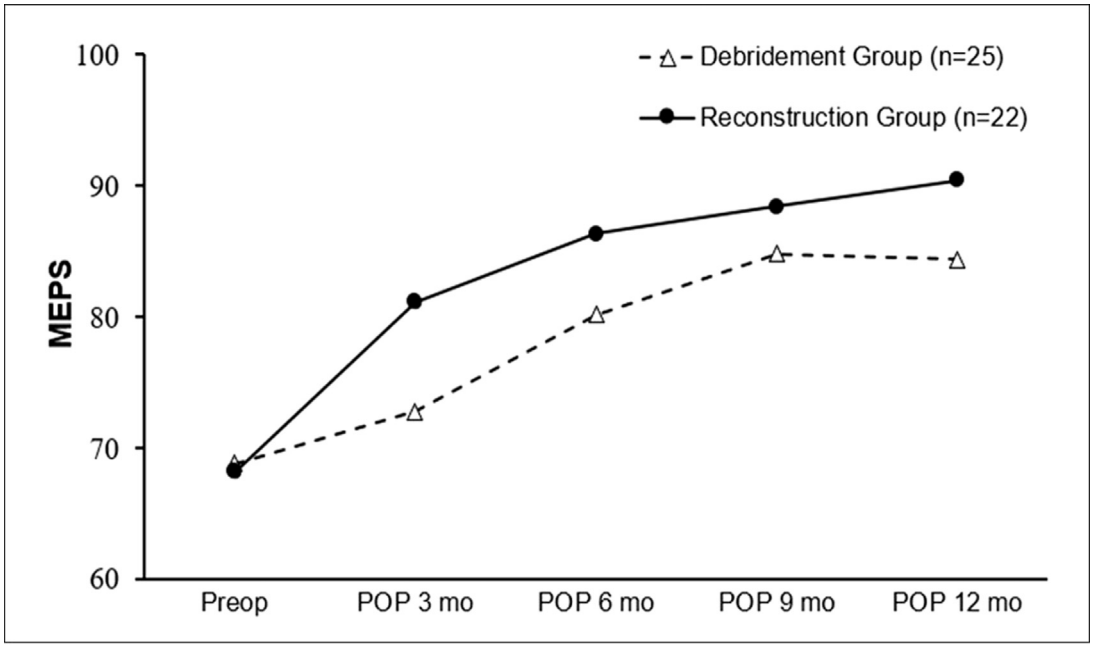
Post hoc analyses revealed that this significant increase in MEPS stemmed from a significant increase in both groups (Fig. 7).

There were no major postoperative complications in both groups. In the reconstruction group, 4 patients complained of stiffness with slight limitation in the flexion-extension arc lasting more than 2 months after surgery but improved by intra-articular steroid injection.

Discussion

Lateral epicondylitis is known to respond well to nonoperative treatment, but about 10% of patients still undergo operative treatment because they are recalcitrant to various combinations of conservative treatments.[5,14] In the case of operative treatment, 80%-90% of cases are reported to show improvement regardless of the type of the operation. However, some of these cases are even refractory to operative treatment.[23] If diseases causing lateral elbow joint pain similar to lateral epicondylitis are sufficiently excluded, those cases of lateral epicondylitis that do not respond to conservative treatment and operative treatment may have other potential etiologies apart from the tendinosis involving the ECRB attachment.

Similar to the mechanism of medial elbow pain due to avulsion injury of the medial ligament in throwing athletes[3,30], the lateral collateral ligament injury is thought to play a role in clinical symptoms of lateral epicondylitis. The footprint of the LUCL is adjacent to the ECRB origin which is traditionally known as the main etiology of lateral epicondylitis. Milz et al[21] reported through a histological study that owing to the characteristics of the enthesis shared by the LUCL and ECRB, pain arising from the injury of either structure could be expressed as a similar region. Previous researches on the damage of the articular capsule in the lateral epicondylitis[36,44] and the anatomical studies on the weakness of the articular capsule adjacent to the ECRB[8,25] sufficiently suggested



(Value: Mean ± SD)

MEPS	Group			Analysis for repeated measures	
	A. Debridement (n=25)	B. Reconstruction (n=22)	p	Source	P value
Preop	68.80±9.16 a[1]	68.18±9.95 a	.857[2]	**Group**	.048[3] (A<B)
POP 3 mo	72.80±9.58 a	81.14±7.06 b	.001[2]	**Time**	<.001[3]
POP 6 mo	80.20±9.63 b	86.36±7.90 c	.016[2]	**Group x Time**	.031[3]
POP 9 mo	84.80±10.15 b	88.41±10.28 c	.215[2]		
POP 12 mo	84.40±14.46 b	90.45±8.72 c	.169[2]		

[1] Values are the mean±SD and Bonferroni's post-hoc test was used for multiple comparisons between each time points. Means with different scripts are different from each other (P<0.05).

[2] P values were derived by Mann-Whitney's U test.

[3] P values are derived from an ANOVA with repeated measures with a Greenhouse-Geisser correction was used when sphericity was not assumed.

Shapiro-Wilk's test was employed for test of normality assumption.

Figure 7 Changes in MEPS during the follow-up period. *MEPS*, Mayo elbow performance score.

that the ligament adjacent to the ECRB could be affected. In several imaging studies using MRI, Bredella et al[4] reported that LUCL abnormalities were observed in more than 60% of patients diagnosed with lateral epicondylitis. Liang et al[36] reported that the most common lesion associated with lateral epicondylitis was LUCL and presented a question of whether the ECRB was the only lesion of the lateral epicondylitis. Based on these results from the previous studies and a finding from the direct cadaveric dissection that degenerative tears of various degrees are found along with the humeral attachment or parenchyma of the LCLC (Fig. 8), the authors assumed that degeneration[38] or attenuation injury of the LCLC including the LUCL and RCL may be potential underlying causes for recalcitrant lateral epicondylitis. Such accumulated microdamage or degeneration of the lateral ligament is in the form of subclinical microinstability and it is assumed that it will present similar symptoms to conventional lateral epicondylitis with pain from valgus load as the main symptom rather than macroscopic instability. Abrupt injury on the degenerated ligament complex also can develop into macroscopic instability such as varus instability or posterolateral rotatory instability.[13]

Chronic pain and instability due to medial ligament injury from repetitive valgus load have been studied extensively in throwing athletes.[30,37,45] Similarly, the authors suggest that in the case of lateral ligaments, symmetrical lesions of such an injury can develop under repetitive varus load. As is well known, the main pathogenesis of lateral epicondylitis is a varus torque injury caused by eccentric contraction of the ECRB, as in the backhand stroke of tennis.[40,41] It is natural to assume that under such circumstances, damage to the lateral ligament, which resists the varus load of the elbow, as well as the ECRB supporting the extension of the wrist. Even in the nonathletic general population, the prevalence of lateral epicondylitis could be explained by considering the fact that ECRB and lateral ligament tension are exerted in repetitive hammering or daily activities with the weight of objects in the hand against gravitational varus torque.[31] Haahr et al reported manual labor, the dominant arm, and a high level of pain as poor prognoses

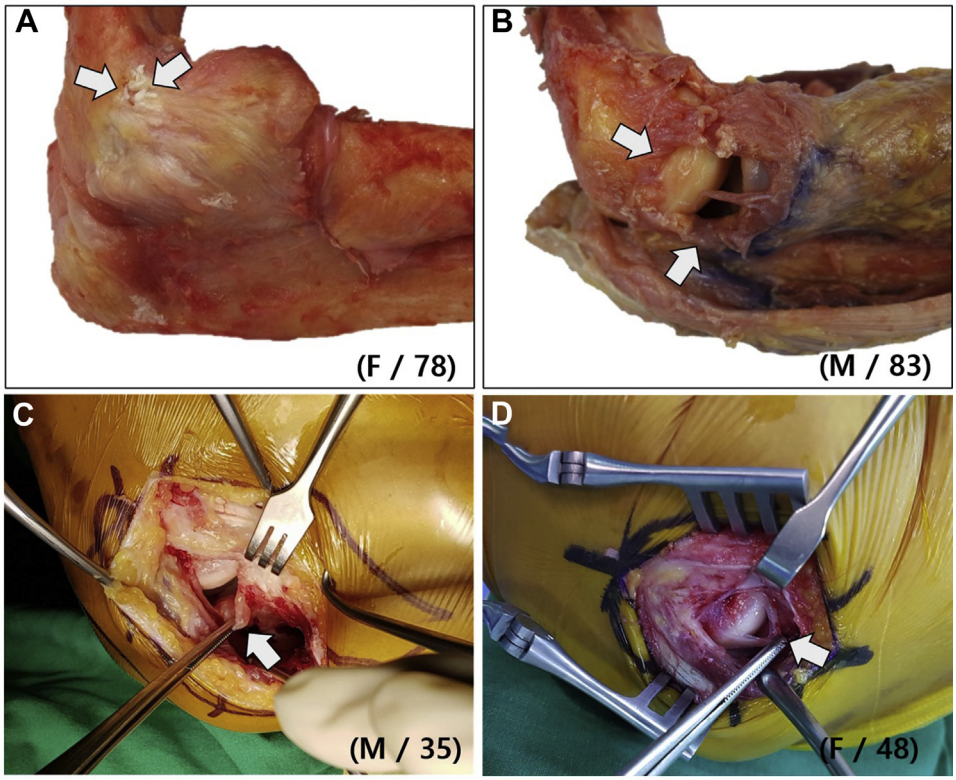


Figure 8 Degenerative lateral collateral ligament complex injuries (arrows) in cadaveric dissections (**A, B**) and in the patients (**C, D**) from the study group.

of lateral epicondylitis.[14] Although it can be assumed that the prognosis may be poor in patients with greater overuse and subsequent ECRB damage, such patients may have damage extended to their adjacent LCLC.

Therefore, by extending the existing hypothesis of the ECRB being the only lesion in the lateral epicondylitis, lateral epicondylitis could be further classified as tendinous (T) type which is limited to ECRB attachment, ligamentous (L) type limited to articular capsule and lateral collateral ligament attachment, and tendinous-ligamentous (T-L) type invading attachment of both structures, thereby providing clearer guidance on the diagnosis and the treatment of the disease (Fig. 9).

Assuming that the ligament lesions associating with lateral epicondylitis may act as a poor factor in the clinical symptoms and treatment course of patients diagnosed with tennis elbow, the authors attempted to develop a reliable and consistent physical examination to detect such ligament injury. The reasons why the authors introduced the MVARST, excluding the existing well-established test for the lateral ligament injury such as lateral pivot shift test or varus stress test, are as follows. First, lateral epicondylitis associating with subtle ligament involvement, the disease spectrum that the authors are trying to introduce, might manifest mainly the same pain as traditional lateral epicondylitis rather than instability such as PLRI or gross varus instability. Lateral elbow pain due to morbidity of LCLC injury ranging from chronic attenuation injury, degeneration to microinstability should be distinguished from the disease that are collectively diagnosed as lateral epicondylitis and subdivided from the conventional concept of lateral epicondylitis. To support such a hypothesis, a test method that evoking pain by loading a selective and stable tension on the LCLC was needed, and the methodology of the moving valgus stress test, which has already been proved to be useful for the similar purpose in the medial ulnar collateral ligament injury, was adopted and modified. The authors believe that this test will be able to distinguish whether the origin of lateral joint pain is ligament or tendon and are conducting further studies.

Second, the authors believe that exertional and tensional pain in the overt instability would be less than the incomplete ligament injury such as attenuation or degeneration injuries because the tension should be relieved by the complete rupture of the ligament. The stretching on the attenuated or degenerated ligament with inflammation causes pain when the injured ligament is maximally tensed. Hence, we expect that the MVARST in combination with stress radiography under anesthesia will provide additional information on ligament involvement.

Although this test should be validated with a large number of the patients in the future, the lesion that invaded the humeral attachment of LCLC on the MRI was found in high frequency in patients with positive MVARST in the study group. Considering that the preoperative baseline of pain was statistically significantly higher in the reconstruction group with positive results in the moving varus stress test, there is an implication that the injury in the ligament is making some contribution to the clinical feature and the prognosis. For these patients, restoration of all or part of the LCLC should be performed independently or additionally because the débridement or release of ECRB alone might be insufficient surgical treatment. We experienced a failure of conventional ECRB débridement in three cases treated with LUCL reconstruction when their symptoms had not previously improved after years of primary surgery. We also found four cases of overt PLRI with the positive pivot shift test under general anesthesia before surgery from the patients who had been diagnosed with chronic recalcitrant lateral epicondylitis over the study period. They showed satisfactory clinical results after LUCL reconstruction but were excluded from this study. In lateral epicondylitis, degenerative changes simultaneously invading the ECRB and LUCL,[4,17] steroid injection–induced

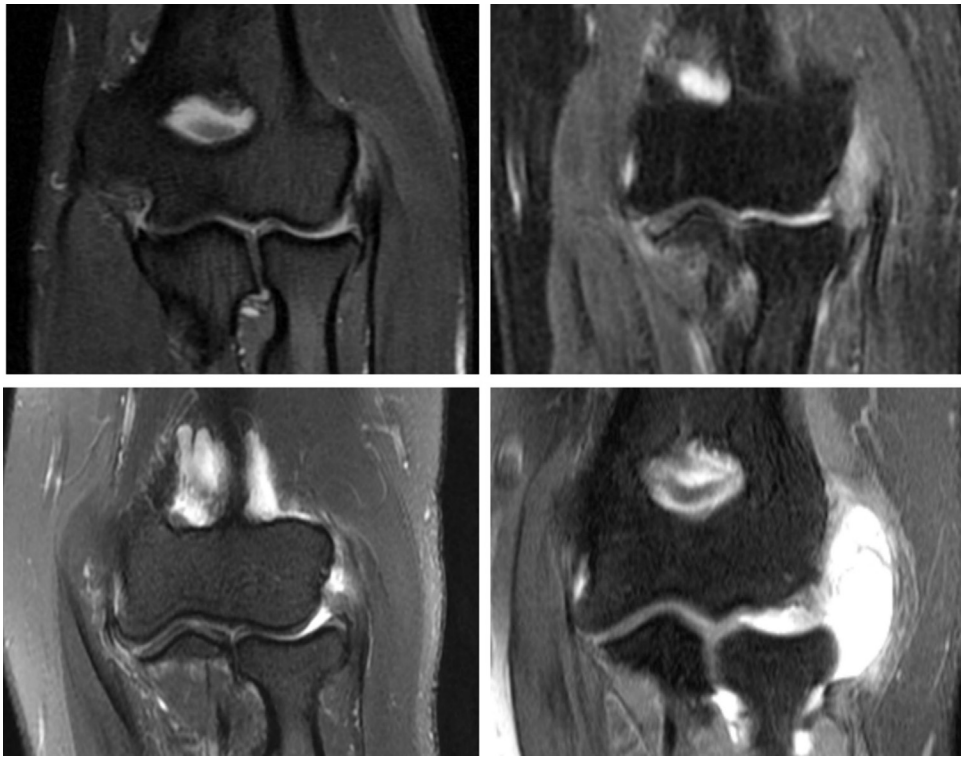


Figure 9 Different types of lesions that involve tendon (ECRB) and ligament (LCLC) in various degrees in the patients diagnosed with lateral epicondylitis of the study group. *ECRB*, extensor carpi radialis brevis; *LCLC*, lateral collateral ligament complex.

ligament injury,[8,11] development of PLRI in lateral epicondylitis due to superimposed trauma, and the weakness of the articular capsules[25] have been reported suggesting the possibility that the two lesions are linked together. Lee et al reported that significant subtle instability was accompanied in the recalcitrant lateral epicondylitis patient group by measuring radiology images under general anesthesia,[20] and the authors agree with this series of reports. Therefore, we recommend that in outpatient treatment or an operation for recalcitrant lateral epicondylitis, stress tests and instability tests should be included in a set of clinical tests to exclude the possibility of the subtle ligament injury or overlooked instability. Currently, surgery to reconstruct the lateral ligament complex is limited to LUCL, but the authors believe that the reconstruction of the RCL, if possible, is also worth considering because the CSR angle is increased in patients in the study group presenting subtle varus instability. Although additional research is required in the massive patient group in the future, 5 degrees of the CSR was found to be a meaningful critical value that could distinguish between the healthy and the affected side in this study. However, to further improve the specificity of the test, the authors propose to consider the possibility of concomitant damage to the ligament when the CSR is increased by more than 5 degrees above the healthy side through a bilateral comparison. In addition to patients with a distinguished increase in the CSR angle, ligament restoration should also be considered for patients suspected of potential ligament injury, such as high level of pain, a history of trauma, multiple steroid injections, and patients who have failed previous surgery for lateral epicondylitis. Ligament repair may also be an easier method than the reconstruction, but the ligament lesion associated with lateral epicondylitis the authors assume is a chronic degenerative change (Fig. 7), so we believe that the strength and resilience of tissues would be far less than that of acute ligament injury.

Clinical satisfaction of LUCL reconstruction in chronic LUCL injury has been reported by several authors thus far, and, the safety of reconstruction using allografts has already been confirmed.[6,42,45] LUCL reconstruction may provide in the patient with lateral epicondylitis in association with ligament insufficiency not only the support as an internal splint enhancing lateral soft tissue,[32] but also restoration of the biomechanics of the LUCL to alleviate symptoms, and may reduce the possibility of the failure of the surgery. This study has limitations of which a difference in the selection criteria between the experimental group and the control group due to the nature of the retrospective study, the small size of the patient group not enough to generalize the hypothesis, and clinical measurement by the physical examination (MVARST) and the radiological measurement (CSR angle) designed by authors that have not yet been validated for feasibility.

Conclusion

LUCL reconstruction combined with débridement of the ECRB origin for the patients with moderate to severe pain, MRI-confirmed LCLC involvement, lateral elbow pain on the varus stress test, and patients who failed primary surgical treatment showed good clinical results with no cases of surgical failure in all cases in the study group. This seems to support the assumptions that lateral ligament involvement might act as one of the underlying causes of lateral epicondylitis, and LUCL reconstruction could be considered as an option for surgical treatment of lateral epicondylitis with associating lesion in the lateral collateral ligament.

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