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Surgical management of lateral epicondylitis combined with ligament insufficiency

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Background: Lateral collateral ligament (LCL) insufficiency may occur in patients with chronic lateral epicondylitis (LE). We report on 14 consecutive patients with chronic LE and LCL insufficiency. **Methods:** We performed a retrospective review of 14 patients with LE and LCL insufficiency diagnosed between 2006 and 2015. The patients had undergone débridement for LE and ligament reconstruction for LCL insufficiency. The study included 9 men and 5 women with an average age of 53 years (range, 41-69 years). The mean follow-up period was 36 months (range, 24-97 months). We analyzed the pain visual analog scale score; Mayo Elbow Performance Score; Disabilities of the Arm, Shoulder and Hand score; range of motion; and posterolateral rotatory drawer test. We compared histories of steroid injection, trauma, and surgery.

Results: The pain visual analog scale score, Mayo Elbow Performance Score, and Disabilities of the Arm, Shoulder and Hand score were significantly improved postoperatively and improved in all patients. Three patients had mild instability on the stress test at final follow-up. All patients had a history of steroid injection, 2 had a history of trauma, and 3 had a history of surgery. The number of steroid injections and the number of cases receiving steroid injections more than 3 times were significantly higher in patients with LCL insufficiency.

Conclusions: Assessment of stability is important in patients with chronic LE and risk factors such as multiple steroid injections. Simultaneous surgical treatment including open débridement and ligament reconstruction provides satisfactory pain relief and functional improvement in patients with LE and LCL insufficiency.

Level of evidence: Level IV; Case Series; Treatment Study

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Lateral epicondylitis (LE) is caused by the degeneration of the common extensor tendon and is a common cause of elbow pain.³⁰ Most LE conditions are self-limiting and respond to conservative treatment, including physical therapy, local injections, and bracing.³⁰ Steroid injections are very effective for acute pain in patients with LE. However, long-term

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follow-up results of using steroid injections have shown poor patient outcomes.^{9,27} Patients who are refractory to conservative treatment may require surgical intervention.^{5,28} Although the surgical treatment for patients with LE is very effective, a few patients may need revision surgery.^{12,22} Patients with refractory LE should be evaluated for alternative diagnoses. Several conditions, such as radial tunnel syndrome, cervical radiculopathy, osteochondral radiocapitellar lesion, posterolateral elbow plica, and posterolateral rotatory instability (PLRI) of the elbow, trigger symptoms similar to those of LE. These differential diagnoses may coexist with LE.⁵

The origins of the lateral collateral ligament (LCL) and extensor carpi radialis brevis (ECRB) are located close to each other anatomically.^{10,24} Therefore, iatrogenic injury during surgery for LE as well as sequelae associated with nonoperative treatment can lead to PLRI of the elbow.^{8,17,21} Steroid injections may weaken the ligament and exacerbate ligament insufficiency during the relatively painless period after the injection.^{20,27} PLRI may also be induced by trauma in patients with chronic LE.¹³ Unrecognized accompanying instability may lead to poor treatment outcomes.¹⁸ Therefore, it is important to identify and treat PLRI in patients with chronic LE, although to our knowledge, few studies have investigated the condition. Kalainov and Cohen¹⁷ reported 3 cases of PLRI with LE. Dzugan et al¹³ reported 7 patients with chronic LE with acute LCL injury. Chanlalit and Limsricharoen⁸ also reported a case of ligament insufficiency due to steroid injections for chronic LE.

We hypothesized that (1) steroid injections are associated with LCL insufficiency and (2) if LCL insufficiency is associated with chronic LE, LCL insufficiency should be treated concomitantly to obtain good results. In this study, we analyzed the causes of LCL insufficiency and reported 14 consecutive patients who were treated for chronic LE and LCL insufficiency.

Materials and methods

Subjects

This study is a retrospective case series of chronic LE with LCL insufficiency. We performed a retrospective review of patients who underwent surgery for chronic LE. Chronic LE was defined as the absence of response to conservative treatment for more than 1 year. A total of 116 operations in 112 patients with chronic LE were performed from January 2006 to December 2015 by a single surgeon. Patients who required ligament reconstruction for LCL insufficiency were selected from among these patients. LCL insufficiency was defined as significant varus or PLRI on physical examination. We performed the varus stress test and posterolateral rotatory drawer test.^{6,7} We defined mild instability as laxity with a firm endpoint and defined severe or significant instability as apparent widening or subluxation without an endpoint. Overall, preoperative instability was not found in 92 surgical procedures; they were thus excluded. In 24 patients, preoperative instability was suspected. Seven patients had mild laxity with a firm endpoint under anesthesia, did not undergo ligament reconstruction, and were excluded from the study. Instability due to bony lesions such as cubitus varus or fracture was excluded. LCL insufficiency due to apparent trauma such as dislocation was also excluded. One patient had an apparent history of trauma and was excluded. Two patients were lost to follow-up and were excluded. The patients who underwent 14 of the 116 surgical procedures were diagnosed with chronic LE and LCL insufficiency and were followed up for more than 2 years. This group of 14 patients is the focus of our study.

The patients included 9 men and 5 women with an average age of 53 years (range, 41-69 years). Eight patients underwent surgery on the dominant side. The mean duration of conservative treatment was 24 months (range, 12-48 months).

The history, physical examination, radiographs, and magnetic resonance imaging (MRI) were reviewed. The radiographs showed no abnormalities such as cubitus varus or osteoarthritis. LE and LCL insufficiency were diagnosed based on physical examination and MRI findings. All patients reported pain on resisted wrist extension and had tenderness to the lateral epicondyle. Eight patients complained about unstable symptoms, and all patients showed positive findings on the varus stress test and posterolateral rotatory drawer test. Instability was confirmed by fluoroscopic findings, such as radiocapitellar joint widening and subluxation during the varus stress test and posterolateral rotatory drawer test under anesthesia. MRI performed in 12 patients showed high signal intensity and discontinuity on the common extensor tendon. Typical findings including ligament discontinuity, joint widening, and radiocapitellar incongruity were observed in 10 patients (Fig. 1, A); 2 patients showed only altered ligament signals.15,23

Surgery

With the patient under general anesthesia, the stress test was performed before surgery. LCL insufficiency was confirmed under anesthesia using a lateral pivot-shift test and posterolateral rotatory drawer test. A clunk that occurred with reduction was identified. Fluoroscopy-assisted stress testing was conducted to confirm LCL insufficiency by widening and subluxation of the radiocapitellar joint (Fig. 1, *B*). All patients were confirmed to have LCL insufficiency.

A curvilinear incision was made on the lateral epicondyle. Débridement of tendinosis associated with the common extensor tendon in LE was performed. We did not perform any repair procedure after débridement. After débridement, ligament reconstruction was performed. All patients showed an attenuated LCL and joint capsule (Fig. 1, C). Elevation of the common extensor origin exposed the LCL at the humerus. The LCL and supinator crest were exposed distally between the anconeus and extensor carpi ulnaris. Reconstruction was performed using a free palmaris longus tendon graft harvested from the ipsilateral forearm. In the absence of the palmaris longus tendon, a half-slip of the flexor carpi radialis tendon was used. An osseous tunnel of the ulna was created at the supinator crest and 1 cm posterior to it. The isometric point of the LCL on the humerus was found, and a humeral tunnel was created at the isometric point. The graft was passed through the ulnar and humeral tunnels over the attenuated LCL complex. The graft was securely sutured to itself with the elbow at 30° of flexion and full pronation (Fig. 1, D).

Patients were diagnosed with severe varus instability when there was apparent joint widening without an endpoint under varus stress. Under conditions of severe varus instability and a satisfactory annular ligament, a dual reconstruction of the lateral ulnar collateral ligament Download English Version:

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