Posterolateral Rotatory Instability of the Elbow in Association with Lateral Epicondylitis

A Report of Three Cases

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Lateral stability of the elbow depends on the integrity of the lateral collateral ligament and secondary soft-tissue restraints[1-4]. Posterolateral rotatory subluxation of the elbow has been recognized in association with elbow trauma, surgery for epicondylitis, and congenital cubitus varus deformity[4-9]. While abnormal signal changes on magnetic resonance imaging have been shown to include the lateral collateral ligament in patients with tennis elbow[10], we are aware of no reports of elbow instability detected in patients in whom epicondylitis had been managed nonoperatively.

We describe the cases of three middle-aged women who presented with atraumatic lateral epicondylitis and subsequently had clinical findings consistent with posterolateral rotatory instability of the elbow. Two of the patients had a visible synovial fistula and magnetic resonance imaging studies indicative of insufficiency of the lateral collateral ligament. All three patients eventually underwent an examination of the elbow under anesthesia, which confirmed the posterolateral rotatory subluxation. All were treated with débridement and reconstruction of the common extensor tendon origin and the lateral collateral ligament. The potential role of corticosteroid injections in the pathogenesis of combined lateral epicondylitis and rotatory instability of the elbow is discussed. Each patient was informed that data concerning her case would be submitted for publication.

Case Reports

Case 1. A fifty-one-year-old, healthy, right-hand-dominant female administrative assistant presented with pain in the lateral aspect of the left elbow of unknown cause. She reported no elbow trauma, unusual musculoskeletal pain, or prior use of fluoroquinolone antibiotics (which has been reported to be associated with degenerative tendon conditions[11]). A diagnosis of tennis elbow was made by the treating hand surgeon on the basis of tenderness over the lateral epicondyle that was exacerbated by wrist extension against resistance. Conservative treatment measures, including the use of a forearm counterforce strap and a wrist splint, were initiated. Two corticosteroid injections, consisting of approximately 40 mg of triamcinolone acetonide, were administered three months apart into the area of maximum point tenderness. Each injection provided nearly complete relief of symptoms for four to six weeks.

The patient sought a second opinion one year following the development of the symptoms. At this time, she described pain and swelling localized to the lateral aspect of the left elbow in addition to a sensation of elbow joint laxity with loading. Examination revealed a tender fluctuant mass extending for a distance of two fingerbreadths distal to the lateral epicondyle. The overlying tissue appeared to be mildly atrophic and less pigmented than the surrounding skin. Subtle posterior subluxation of the radial head was palpable with a lateral pivot-shift maneuver, which was performed by applying a valgus stress and axial load to the partially flexed elbow with the forearm fully supinated[4,5].

The findings on plain radiographs of the left elbow were unremarkable. However, magnetic resonance imaging revealed rupture of the lateral collateral ligament with retraction of a large portion of the common extensor tendon origin and a 2 by 2.5-cm synovial fistula projecting into the subcutaneous tissues (Fig. 1-A).

The patient elected to proceed with surgical treatment. The results of preoperative laboratory studies, including a complete blood-cell count and measurements of the serum uric acid level, erythrocyte sedimentation rate, and C-reactive protein level, were normal. A joint fluid analysis demonstrated features of a noninflammatory synovial effusion. Cultures were negative.

With the patient under axillary block anesthesia, the lateral epicondyle and the common extensor tendon origin were exposed through a curvilinear incision. A large defect in the common extensor tendon origin was appreciated distal to the lateral epicondyle (Fig. 1-B). Gross posterolateral and varus

instability was demonstrated with application of stress to the joint. A capsulotomy anterior to the lateral collateral ligament was performed, revealing a bare epicondyle anteriorly and attenuated tissue posteriorly. The capsular tissue was thinned and expanded.

The remaining attachments of the common extensor tendon origin and the lateral collateral ligament were elevated sharply from the lateral epicondyle and débrided back to healthy-appearing tissue. The lateral collateral ligament was reconstructed with a free palmaris longus tendon graft harvested from the ipsilateral forearm[6] (Fig. 1-C). The joint capsule was closed beneath the graft as well as possible, and remnants of the endogenous collateral ligament were sewn to the tendon graft construct proximally. Extensions of the common extensor origin were then reattached to the lateral epicondyle with use of a bone anchor and number-2 braided polyester suture. Because of the poor quality of the capsular tissue and the partially deficient extensor origin, an anconeus muscle pedicle flap was rotated for coverage of the tendon graft and the inferior margin of the radiohumeral joint[12] to prevent recurrence of the synovial fistula. Restoration of elbow joint stability was confirmed fluoroscopically with varus and lateral pivot-shift testing.

Postoperatively, the elbow was immobilized in 60° of flexion with the forearm fully pronated[2,4]. Active elbow-motion exercises with a 30° extension-block splint were encouraged at approximately two weeks postoperatively. Forearm-rotation exercises were initiated at five weeks, and the use of the splint was discontinued at six weeks. Unrestricted use of the extremity was permitted at four months following the surgery. When she was last examined, two years postoperatively, the patient reported no residual elbow pain or sensation of joint laxity. With the exception of a loss of 5° of terminal extension, the patient had full elbow and forearm motion and no clinical or radiographic evidence of recurrent joint instability.

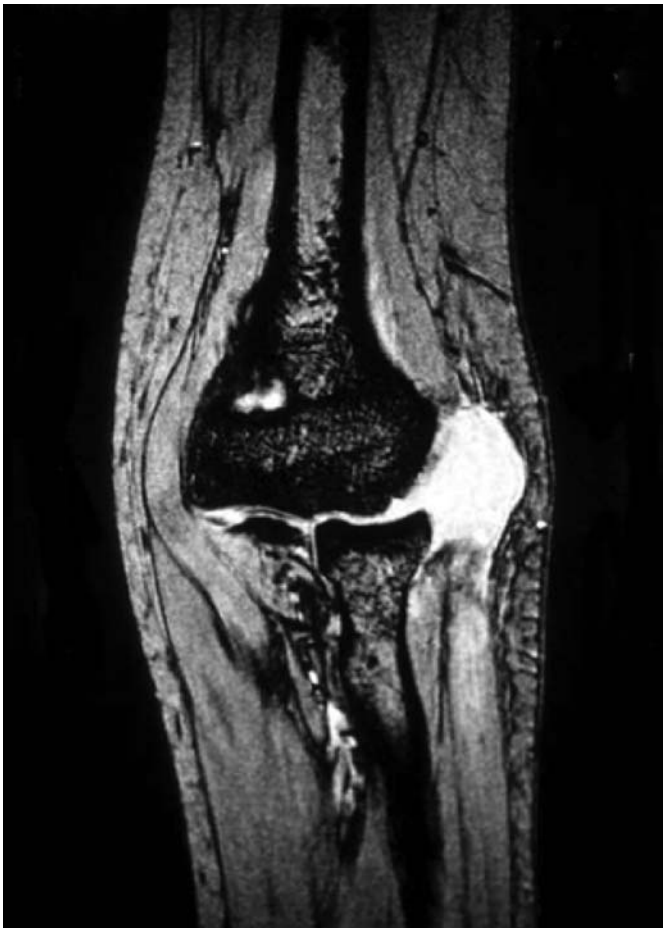

Fig. 1-A

Figs. 1-A, 1-B, and 1-C Case 1. **Fig. 1-A** T2-weighted coronal magnetic resonance image revealing rupture of the lateral collateral ligament and the extensor tendon origin at the epicondyle with a synovial fistula.

CASE 2. A forty-nine-year-old, healthy, right-hand-dominant female nurse presented with pain in the lateral aspect of the right elbow, which she related to pushing up from the ground with her hands to stand. She reported that she had not had previous elbow surgery or relevant trauma and that she had not used fluoroquinolone antibiotics preceding the onset of the symptoms. A diagnosis of tennis elbow was made by her treating physician on the basis of the history and the pain localized to the lateral epicondyle. Physical therapy was prescribed, and over a fifteen-month interval four separate corticosteroid injections were administered into the region of maximum point tenderness. Each injection, consisting of approximately 40 mg of triamcinolone acetonide, markedly but only temporarily relieved the symptoms.

Shortly following the fourth injection, prominent swelling with more pronounced discomfort developed over the lateral aspect of the right elbow. The patient subsequently sought a second opinion, at which time she reported pain and a clicking sensation in the elbow with active extension. The skin over the lateral epicondyle was thinned, and there was a tender area of swelling corresponding to the common extensor origin. The lateral pivot-shift test elicited sensations of both pain and apprehension. Baseline radiographs of the elbow revealed unremarkable findings, whereas magnetic resonance imaging demonstrated disruption of the lateral collateral ligament and the common extensor tendon origin with a synovial fistula extending through the soft-tissue defect.

The elbow joint was aspirated, and the fluid analysis revealed features of a noninflammatory synovial effusion. Cultures were negative. The results of preoperative serological studies, including a complete blood-cell count and measurements of the uric acid level, erythrocyte sedimentation rate, and C-reactive protein level, were normal.

At surgery, the common extensor origin was found to be torn and retracted with the tendon edge positioned approximately 3 cm distal to the epicondyle. Pathological laxity of the elbow was demonstrated with application of posterolateral rotatory stress under fluoroscopy. The synovial fistula was entered, and the lateral collateral ligament was found to be partially disrupted and attenuated. Both varus laxity and pos-

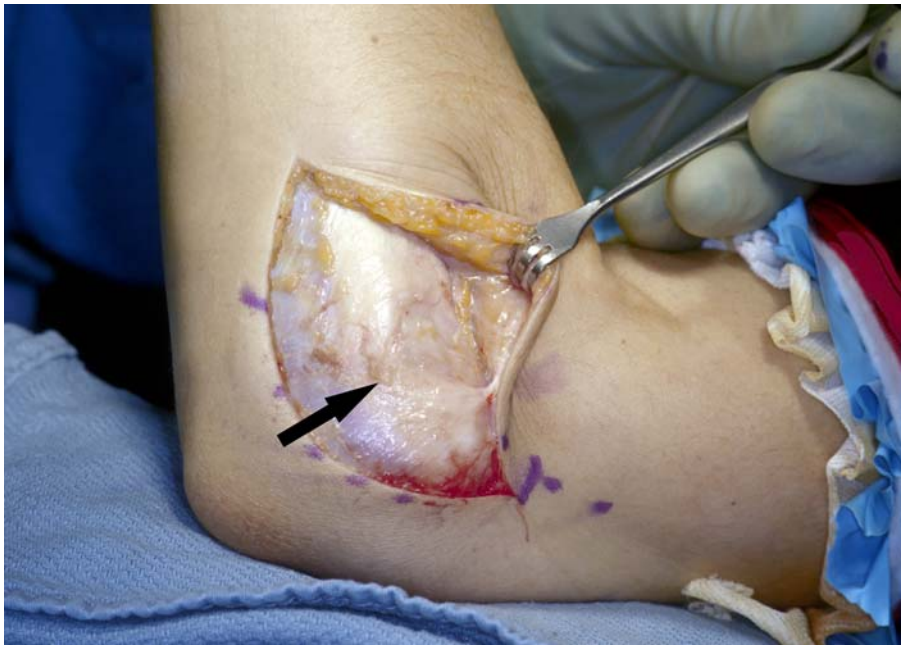

Fig. 1-B

Intraoperative photograph depicting a torn and retracted common extensor tendon origin (arrow). This was visible on incision of the skin.

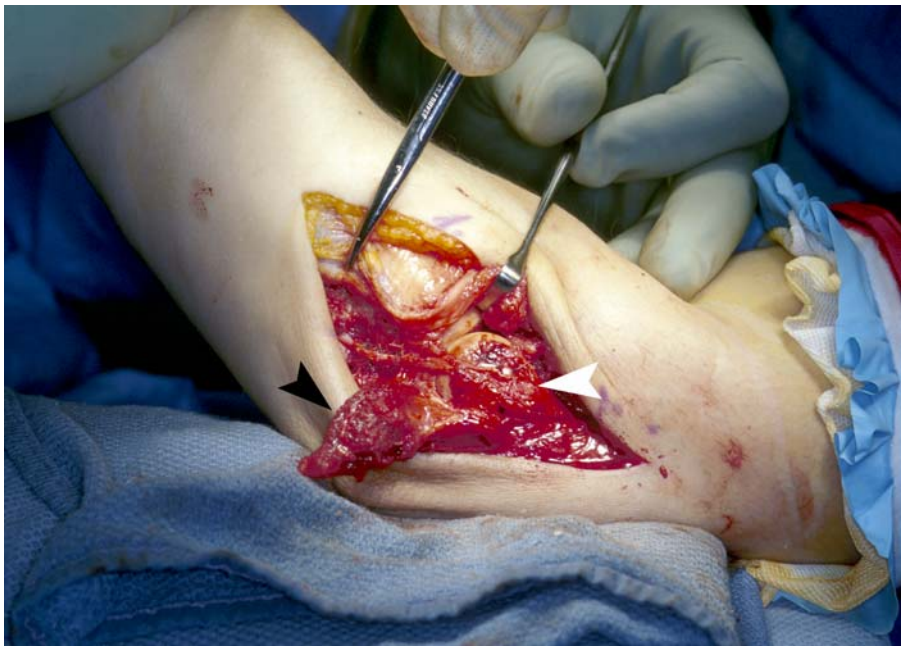

Fig. 1-C

Intraoperative photograph showing reconstruction of the lateral collateral ligament (white arrowhead at the humeral tunnels) and the reflected anconeus muscle (black arrowhead).

terolateral rotatory subluxation of the elbow were confirmed by direct visualization. Small chondral defects were detected in the radial head and capitellum, but joint congruity had been preserved.

The deficient lateral collateral ligament was reconstructed with a palmaris longus tendon autograft. Capsular tissue was then attached to the undersurface of the tendon graft, and the remaining collateral ligament origin was sewn to the graft construct proximally. The extensor tendon origin was débrided and was repaired to the lateral epicondyle with one suture

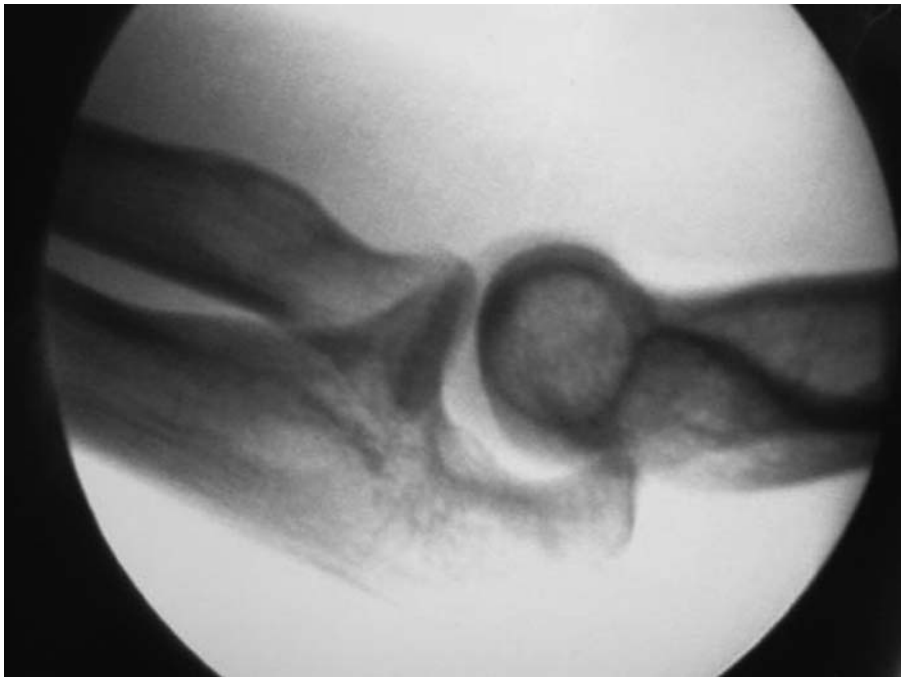

Fig. 2

Case 3. Fluoroscopic image made during performance of the lateral pivot-shift test with the patient under anesthesia. Posterior subluxation of the radial head and widening of the ulnohumeral joint are seen with elbow extension.

anchor. The anconeus muscle was advanced proximally and anteriorly to cover the graft and the inferior part of the radiohumeral joint. A rehabilitation program identical to the program described for Case 1 was prescribed.

Histological analysis revealed features of myxoid alteration, fibroblast proliferation, and neovascularization in the torn tendon edge[13-15]. At the latest clinical examination, nine months after the surgery, the patient reported that she had no residual elbow pain. She had full elbow extension as well as elbow flexion and forearm rotation that were symmetrical with those on the contralateral side. There was a painless palpable click directly over the ligament reconstruction site with elbow flexion but no clinical or radiographic evidence of recurrent elbow joint instability.

CASE 3. A forty-five-year-old, right-hand-dominant female administrator presented with pain in the lateral aspect of the left elbow that had developed while she was constructing a shed in her yard. Conservative measures for the treatment of tennis elbow, including nonsteroidal anti-inflammatory medication and a counterforce strap, were prescribed by her primary care physician. Initial records documented tenderness over the lateral epicondyle.

The patient was referred for orthopaedic evaluation two months later. She reported no medical problems, previous use of fluoroquinolone antibiotics, or previously recognized elbow injury. On physical examination, point tenderness over the lateral epicondyle was elicited in addition to lateral elbow pain with resisted wrist extension. There was no appreciable joint instability with lateral pivot-shift testing. Two separate corticosteroid injections, consisting of approximately 15 mg of triamcinolone acetonide, were administered over a six-month interval; each injection resulted in partial pain relief for several months.

Ten months following the initial onset of symptoms, the patient reported recurrent and more pronounced pain in the left elbow. The pain was localized laterally and was reproducible with full elbow extension, resisted wrist extension, and finger pressure over the lateral epicondyle. Radiographs of the joint revealed unremarkable findings, whereas magnetic resonance imaging demonstrated extensive partial tearing of the common extensor tendon origin, a small joint effusion (synovitis), and degenerative cysts in the capitellum. Although the lateral collateral ligament appeared intact, the entire structure was not well visualized.

The patient decided to undergo surgery to débride the common extensor tendon origin. She was informed of the possibility of detecting lateral instability intraoperatively and that this might alter the surgical plan to include ligament reconstruction. The results of laboratory studies, including a complete blood-cell count and measurement of the erythrocyte sedimentation rate, were normal.

Fluoroscopic imaging of the left elbow with the patient under general anesthesia revealed frank posterolateral rotatory instability (Fig. 2). No instability of the asymptomatic right elbow was detected. The common extensor tendon origin was

approached by elevating the inferior border of extensor carpi radialis longus muscle. Abundant amorphous tissue was visualized at the origins of the extensor carpi radialis brevis muscle and the lateral collateral ligament, and there was definite instability of the elbow joint. Degenerative tissue was débrided, and pathological laxity of the lateral collateral ligament was appreciated with application of varus and lateral pivot-shift testing. The ligament was reconstructed with use of a palmaris longus tendon autograft. Capsular tissue was then secured to the undersurface of the tendon graft, and the origins of the common extensor tendon and endogenous lateral collateral ligament were reattached to the lateral epicondyle with use of one suture anchor. As adequate soft tissue was available for joint coverage, the anconeus muscle was not transferred. Stability was confirmed by direct visualization and with fluoroscopic imaging.

The splinting and rehabilitation protocols were similar to those used for the other two patients. Tissue obtained at the time of surgery demonstrated features of fibroblast proliferation and neovascularization. At the latest clinical examination, eight months after the surgery, the patient reported resolution of the elbow pain. Full and symmetrical elbow and forearm motions were demonstrated. There was painless palpable crepitus over the radiohumeral joint with stress testing but no clinical or radiographic evidence of recurrent joint instability.

Discussion

Posterolateral rotatory instability of the elbow typically results from a traumatic event involving a combination of axial compressive, external rotatory, and valgus forces, such as occurs when a person falls on an outstretched hand[2,5]. This pattern of instability has been recognized following failed surgery for tennis elbow and has been attributed to iatrogenic injury to the lateral collateral ligament[6,9]. While lateral ligament insufficiency has also been detected by magnetic resonance imaging in patients in whom tennis elbow had been managed conservatively, and by direct visualization during primary surgery for tennis elbow, concurrent rotatory instability has not been previously reported, to our knowledge[10,16].

At present, there is little clinical information to guide the choice of corticosteroid preparation or method or the number of injections that can be safely administered for the treatment of tennis elbow[17]. Although a steroid injection may alleviate the pain associated with tennis elbow, the effects are usually transitory[18]. In addition, animal data support caution regarding the nonjudicious use of local corticosteroids. An intratendinous injection results in collagen necrosis followed by a decrease in tensile strength[19-23]. An injection into an injured ligament impairs healing and adversely affects the failure load of the tissue[24-27]. A reduction in the structural properties of severed rabbit ligaments has been shown to persist for three weeks or more following a single corticosteroid injection[24,26].

We postulate that the degenerative changes in the extensor tendon origin associated with lateral epicondylitis can also involve the underlying lateral collateral ligament. The epicondylar attachments of the extensor carpi radialis brevis, the extensor digitorum communis, and the lateral collateral ligament are confluent[2,17]. Repeated corticosteroid injections into the tendon and ligament origins may contribute to weakening and ultimate failure of these structures. Early joint-loading following an injection may further compromise the lateral soft-tissue restraints of the elbow. Seemingly minor previous trauma to the elbow could conceivably be an additional factor in tendon and ligament deficiency.

Posterolateral rotatory subluxation of the elbow should be considered in the differential diagnosis of either persistent or recurrent symptoms of tennis elbow, especially in patients with mechanical elbow symptoms and/or a recognized synovial fistula. Magnetic resonance imaging and stress radiographs may be helpful for identifying subtle cases of instability[5,6,9,28]. We currently advocate intraoperative evaluation for lateral instability of the elbow joint following surgical treatment of lateral epicondylitis. When lateral elbow instability is identified, débridement and repair of degenerative extensor tendon tissue with reconstruction of the lateral collateral ligament may be indicated. Supplemental coverage of the radiohumeral joint, when required, can be achieved with a local anconeus muscle transfer. Additional clinical series with longer follow-up are necessary to determine the efficacy of this surgical approach. ■

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