Elbow tendinopathy

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Summary

Lateral epicondylosis is a common pathology of the upper extremity. The origin of the ECRB is the most commonly cited anatomic location of lateral epicondylosis pathology. Histologic examination shows the features of a *failed healing response*, with absence of acute inflammatory cells. The typical patient with lateral epicondylosis is an adult in the fourth or fifth decade of life, with no difference about the sex. Diagnosis is based on history and physical examination. The role of imaging is to confirm the diagnosis. The most consistent symptom of lateral epicondylosis is pain over the lateral aspect of the elbow. Therapeutic modalities for lateral epicondylosis vary widely and lack definitive evidence. Open, percutaneous or arthroscopic surgery is recommended when functional disability and pain persist after 6 to 12 months of nonoperative management. Future studies using validated clinical measures and imaging are needed to determine the best management for patients with lateral epicondylosis.

Key words: arthroscopy, elbow tendinopathy, injections, platelet rich plasma, surgery.

Introduction

Elbow tendinopathy (ET) ("tennis elbow") is a common pathology of the upper extremity, with an incidence up to 4-7/1000 patients per year[1, 2]. ET has a substantial impact both on athletes and workplace. Traditionally, ET has been attributed to a *failed healing response* of the extensor carpi radialis brevis (ECRB) origin, although the underlying collateral ligamentous complex and joint capsule also have been implicated[3]. The condition was first described by Runge[4] in 1873 and successively named "lawn-tennis arm" by Major[5] in 1883 because of its association with the above sport.
The origin of the ECRB is the most commonly cited anatomic location of ET pathology[1, 2]. Histologic examination shows the features of a *failed healing response*, with absence of acute inflammatory cells. These characteristics are consistent with them found in other tendinopathic tendons[6-8]. The overall presentation is consistent with a pattern of repetitive microinjury and healing attempts[9]. Nirschl et al.[10] noted that degeneration within the extensor *digitorum communis* (EDC) involved up to 50% of patients.

Epidemiology

The typical patient with ET is an adult in the fourth or fifth decade of life[1, 2]. Incidence is similar in men and women, with symptoms more commonly seen in the dominant arm. Causes and mechanism of ET are still unclear. Probably the condition arises from a combination of mechanical overloading[11] and abnormal microvascular responses[12]. The onset of symptoms has been attributed to an overexertion of the extremity with repetitive wrist extension and alternating forearm pronation/supination. A history of manual labor with heavy tools and significant strain while performing repetitive tasks could also be considered a causative factor of the condition. ET is likely to be a self-limiting pathology, and symptoms will improve over time. Approximately 80% of patients with newly diagnosed ET report symptomatic improvement at 1 year[1, 13]. Although most patients may experience mild residual symptoms, only 4% to 25% of patients non responding to conservative management will require surgery[1, 2]. Poor prognostic factors for successful non-operative management options include manual labor, dominant arm involvement, long duration of symptoms with high baseline pain levels, and poor coping mechanisms[13].

Diagnosis

The diagnosis is mainly based on history and physical examination. The role of imaging is to confirm the diagnosis. The most consistent symptom of ET is pain over the lateral aspect of the elbow. The pain is usually sharp and exacerbated by activities involving active wrist extension or passive wrist flexion with the elbow extended. Inability to hold items (e.g., a coffee cup) because of pain in the lateral aspect of the elbow is also a characteristic complaint[3].
Although there is often a history of repetitive activity, symptom can be insidious, with no clear inciting event. Typically, the patient complains of maximal tenderness slightly anterior and distal to the lateral epicondyle over the origin of the ECRB and the EDC muscles. Less frequently, localized tenderness is present at the apex of the bony lateral epicondyle[3], rarely accompany by swelling, erythema, or warmth. Pain localized to the lateral epicondyle or just slightly distal

to the extensor origin is often elicited.
Although ET remains primarily a clinical diagnosis, several imaging modalities have been proposed to provide ancillary information[14]. Radiographs occasionally show calcifications within the extensor mass origin or intra-articular pathology[14]. Such calcifications can be associated with persistent disease and are present in approximately 20% of patients requiring surgery[14].
Magnetic resonance imaging (MRI) may be used to evaluate a suspected intra-articular process, to assess the competency of the radial collateral ligament complex, and to define the extent of tearing of the extensor origin. MRI may also show oedema and thickening of the extensor origin in 90% of symptomatic patients[15, 16]. With high resolution and fine-cut imaging, the ECRB origin can be classified as separated away from the radial collateral complex, thinned, or partially versus completely torn[17]. Although an increased T2 signal at the ECRB origin does not correlate with symptom severity, the extent of tendon involvement has proved to be accurate at the time of debridement [18]. MRI has not been shown to provide useful information in determining response to management because increased T2 signal may persist weeks after symptom resolution[17]. MRI findings must be correlated with clinical examination since 14% to 54% of asymptomatic elbows will show oedema in the common extensor origin[16, 17]. Ultrasound in ET identifies focal hypoechoic areas, intrasubstance tears, peritendinous fluid, and thickening of the common extensor origin. Ultrasound provides moderate sensitivity (64% to 88%) when evaluating the extensor origin architecture. However it has a variable specificity (36% to 100%)[19, 20]. Given the significant level of operator dependence, ultrasound is most useful when performed and interpreted by experienced individuals[19, 20].

Differential Diagnosis

Diagnosis of ET should exclude several conditions that determine similar symptoms. Radial tunnel syndrome, or compression of the posterior interosseous nerve, may be difficult to differentiate from ET.
Maximal tenderness in radial tunnel syndrome is typically noted 3 to 4 cm distal and anterior to the epicondyle over the mobile wad[21]. Resisted wrist extension may not be painful in radial tunnel syndrome, but it is painful in ET [21].
Resisted thumb and index finger extension may be painful in radial tunnel syndrome but not with ET[21]. Resisted forearm supination may be painful in radial tunnel syndrome because of compression of the radial nerve (PIN) within the supinator muscle. ET and radial tunnel syndrome may coexist in up to 5% of patients[21]. In each patient, the cervical spine and shoulder also should be examined to identify proximal radicular symptoms or pathologic findings. A careful examination also is warranted to identify the patient with intra-articular pathology, such as radiocapitellar chondral lesions. Estimates of concurrent intra-articular pathology range from 11% to 69%[10, 22]. Ruch et al.[23] reported on 10 patients with a posterolateral plica causing refractory lateral elbow pain. The most suggestive physical examination findings included a painful clicking at terminal extension and forearm supination, as well as maximal tenderness over the posterior radiocapitellar joint. Some patients initially diagnosed and conservatively managed for ET have had unrecognized posterolateral rotatory elbow instability[24]. Limited shoulder internal rotation was reported as co-morbidity in a series of patients with ET[25]. Such restriction of motion during a tennis serve may require compensation via excessive wrist flexion, which would strain the ECRB and predispose these individuals to ET[25].

Management

Therapeutic modalities for ET vary widely and lack definitive evidence. A variety of management options has been proposed. The basic premise behind each approach is the desire to aid or enhance natural healing. Nirschl and Ashman[26] suggested an ordered management progression parallel to the healing response. This approach begins with initial control of exudation and hemorrhage, followed by the promotion of tissue healing, encouragement of general fitness, and the control of force loading. The final step, necessary in only a minority of patients, is removal of the pathologic tissue.

Conservative therapy

Non-steroidal anti-inflammatory drugs and corticosteroid injections have traditionally been used for the management of patients with ET. However, they have not been shown to be more effective than watchful waiting in the long-term[27, 28].
Eccentric exercise regimens have shown some efficacy compared to age-gender-activity matched controls, though a sub-cohort of patients remains refractory[29, 30].
Polidocanol (Aetoxisclerol®, Kreussler, Germany), prolotherapy, autologous whole blood and platelet rich plasma (PRP) injection therapies have reported promising outcomes for ET. Polidocanol is a vascular sclerosant. It is used for the management of tendinopathy to sclerose areas of high intra-tendinous blood flow, sometimes termed “neovessels”, which are seen histopathologically[9] and *in vivo* under high resolution ultrasound with color Doppler. Neovascularity is thought to be associated with the underlying mechanism of ET and other overuse tendinopathies, though whether it is a causal agent in the pathophysiology of tendinopathy is not clear[31]. Another study reported that sustained sclerosis of neovascularity in ET was a good predictor of positive clinical effect at 2 years[32].
Several RCTs and prospective case series have reported positive effects of polidocanol therapy for patellar, epicondylar and Achilles tendinopathies[32, 33]. The use of prolotherapy dates to the 1930s[34], when it was developed for pain associated with presumed ligament laxity. Although several injection agents have been used, hyperosmolar dextrose and morrhuate sodium (also a vascular sclerosant) are the most popular and best studied agents[35, 36]. Prolotherapy has also been used to manage ET[37].
Autologous whole blood and platet-rich plasma (PRP) have been used as injectants for tendinopathy with the aim of providing cellular and humoral mediators to induce healing in areas of degeneration. Autologous whole blood injections have been used for medial[38] and lateral epicondylosis[39].
PRP is prepared from autologous whole blood, which is centrifuged to concentrate platelets in plasma. The aim is to augment the native healing process at the site of pain through the action of platelet-derived growth factors (PDGF).

Platelets contain at least 6 PDGFs vital to bone and soft tissue healing. Since the early 1990s PRP has been used for its purported ability to improve soft tissue healing and bone regeneration. The use of PRP is being intensely studied and reports suggest that clinical use is increasing rapidly for ET, rotator cuff repair, acute and chronic muscle strain, muscle fibrosis, and ligamentous sprains.

Peerbooms et al.[40] performed a randomised prospective controlled study to determine the effectiveness of PRP compared with corticosteroid injections in patients with chronic ET. Successful treatment was defined as more than a 25% reduction in visual analog scale (VAS) pain score or DASH score without a reintervention after 1 year. The results showed that, according to the VAS pain scores, 24 of the 49 patients (49%) in the corticosteroid group and 37 of the 51 patients (73%) in the PRP group were successful, which was significantly different (P<.001). Moreover, according to the DASH scores, 25 of the 49 patients (51%) in the corticosteroid group and 37 of the 51 patients (73%) in the PRP group were successful, which was also significantly different (P 5 .005). The corticosteroid group was better initially and then declined, whereas the PRP group progressively improved.

Gosens et al.[41] performed a double-blind randomized controlled trial to determine the effectiveness of PRP compared with corticosteroid injections in patients with chronic ET. The trial was conducted on 100 patients that were randomly assigned to a leukocyte-enriched PRP group (n = 51) or to the corticosteroid group (n = 49). After the allocated treatment, they were evaluated with visual analog scale (VAS) pain scores and DASH outcome scores. The patients in the PRP group demonstrated more successful results than the patients in the corticosteroid group (P < .0001). The patients in the PRP group had a reduction of 25% on VAS pain score or DASH scores without a re-intervention after 2 years. Furthermore, the DASH scores of the corticosteroid group returned to baseline levels after 2 years, while those of the PRP group significantly improved.

Recently, Thanasas et al.[39] conducted a randomized controlled trial to evaluate the effectiveness of PRP compared with autologous whole blood in patients with chronic ET. The study was performed on 28 patients that were equally randomized into 2 groups: in group A patients were treated with a single injection of 3 mL of autologous blood and in group B patients received a 3 mL injection of PRP under ultrasound guidance. A standardized program of eccentric muscle strengthening was followed by all patients in both groups. The results evaluation was made using a VAS pain score and the Liverpool elbow score was performed at 6 weeks, 3 months, and 6 months after the injection. Patients in group B reported a larger improvement of the VAS pain score than patients in group A at every follow-up interval, but the difference was statistically significant only at 6 weeks, when mean improvement was 3.8 points (95% confidence interval [CI], 3.1-4.5) in group B (61.47% improvement) and 2.5 points (95% CI, 1.9-3.1) in group A (41.6% improvement) (P < .05). No statistically significant difference was noted between groups regarding Liverpool elbow score.

Surgical therapy

Open debridement

Surgical management of ET is recommended when functional disability and pain persist after 6 to 12 months of nonsurgical management. A 3-cm incision is made, centered just distal to the lateral epicondyle. Sharp dissection continues to the site of the common extensor origin. This enthesis is incised in line with the fibers, revealing the ECRB, deep and posterior to the ECRL. Degenerative tissue within the ECRB, which often has a gray hue, is debrided, and the underlying epicondyle is decorticated. The remaining tendon is reattached as dictated by the extent of debridement.

Overall results are encouraging. Nirschl and Pettrone[10] reported on 88 patients with ET managed by an open release and arthrotomy. They report excellent results in 66 of 88 patients. An 11% incidence of intra-articular pathology was noted. Most surgical series confirm the results of Nirschl and Pettrone[10], reporting predominantly good to excellent outcomes. However, a significant percentage of patients report persistence of mild, intermittent symptoms. In a series of 19 patients managed with open extensor release and origin reattachment, 18 of 19 patients were "better"[42]. In a long-term prospective study, forty-seven (76 per cent) of the sixty-two patients who were evaluated at one year had no pain or only slight pain[43].

Postoperative regimen

Splint or sling immobilization for 10 days following open release and extensor origin repair is common. Range-of-motion exercises are then commenced, and strengthening is started after 6 weeks. Most surgeons use post-operative splinting protocols until strength is regained. A wrist support splint for 10 to 14 days and a gradual return to activities may be recommended.

Complications

Excessive debridement may compromise the lateral stability of the elbow, resulting in iatrogenic posterolateral rotatory instability. Neuroma of the posterior cutaneous nerve of the forearm is a potential source of persistent postoperative pain. This cutaneous branch of the radial nerve crosses 1.5 cm anterior to the lateral epicondyle on the brachioradialis fascia. Evidence of nerve injury includes paresthesia and dysesthesia distal to the incision. The diagnosis can be confirmed with symptomatic relief as a result of local anesthetic block. This complication may be reliably treated by neuroma excision with intramuscular implantation of the proximal nerve end. Reactive bone formation following open release has been reported; this may require surgical excision[44].

Arthroscopic debridement

The arthroscopic approach to ET varies among surgeons. Some prefer to debride the lateral capsule and infolded tissue that may impinge within the radiocapitellar joint, while others focus debridement on the extensor origin. The arthroscopic appearance of the lateral capsule has been classified by Baker et al.[45]. Elbow arthroscopy can be performed with the patient in the prone, lateral, or supine decubitus. We prefer the supine position with an armholder.

The joint is first injected with 30 mL of saline to displace neurovascular structures away from portal sites and to facilitate arthroscope insertion. A proximal-medial portal is then established, inserting the cannulated trocar along the anterior humeral cortex. Return of saline confirms intra-articular placement. The radial head, capitellum, distal humerus, and anterolateral capsule are visualized through this medial portal. A superior-lateral portal is created as the working portal for a motorized shaver. The degenerative capsule and undersurface of the ECRB are then released off the lateral epicondyle.

The distal extent of debridement remains parallel to the superior half of the radial head, and the proximal debridement ends at the muscular ECRL insertion. Respecting this distal margin reliably protects the origin of the lateral collateral complex[46]. The exposed epicondyle can be decorticated using a high-speed burr.

In three arthroscopic series, 93% to 100% of patients were "better" or "much better" at an average of 2 years postoperatively[22, 44, 45]. However, only 62% to 80% of patients experienced complete elimination of lateral elbow pain[22, 44, 45]. For these patients, the average time to return to work was 11 days (range, 0 to 42 days). Szabo et al.[47] published the largest comparative series to date of surgical debridement techniques. Patients were followed for a minimum of 2 years after arthroscopic (n = 41), open (n = 38), or percutaneous (n = 23) lateral epicondyle release. No statistical difference in outcomes between the groups was identified. One major limitation in this series is that it was a nonrandomized, retrospective evaluation. Peart et al.[48] performed a retrospective, comparative study of open (46 patients) versus arthroscopic release (29 patients). At 6 months, the procedures yielded statistically identical results, with nearly 70% good or excellent outcomes with both procedures. Patients returned to work earlier following arthroscopic treatment.

Postoperative regimen

Postoperatively, the patient may be placed in soft bandages and a sling for comfort. Most authors report protocols incorporating range-of-motion exercises in the first few days postoperatively[22, 45]. Some surgeons allow early strengthening guided by comfort or the resolution of swelling, while others may postpone this phase of rehabilitation until 4 to 6 weeks postoperatively.

Complication

Complications following elbow arthroscopy include nerve injury, heterotopic ossification, and posterolateral rotatory instability following overly aggressive débridement[47, 49, 50]. Keeping débridement in line with the anterior half of the radial head prevents destabilization of the lateral elbow[49].

Conclusion

Several treatment options have been proposed for the management of ET. Prolotherapy, polidocanol, autologous whole blood and PRP injection therapies for refractory ET suggest effectiveness, but are limited by lack of large definitive trials. Future studies using validated clinical measures and imaging are needed to determine whether these techniques can play a definitive role in the management of patients with ET.

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