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## CASE REPORT

### Idiopathic Degenerative Elongation of Extensor Pollicis Longus Tendon: Case Series

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#### ABSTRACT

Three cases of elongation and attrition of the extensor pollicis longus without detectable fracture after wrist injury or dislocations of carpal bones or radio carpal joint and any connective tissue disorders are reported. The average age of all patients was 20 years. With palmar flexion of wrist, patients were able to completely extend their thumb finger, but with dorsiflexion of wrist, the ability to extend the thumb was lost. Elongation and thinning of the extensor pollicis longus tendon have not been reported in the literature. No mechanisms have been proposed to account for this problem. The following cases, the first of its kind of which we are aware, may help to clarify the relationships between degeneration and tendon rupturing.

**Key words:** Case series, extensor pollicis longus, degenerative elongation

#### Introduction

Of the many idiopathic abnormalities of the hand, those involving the extensor mechanism are very rare. Zadek [1] reported the first case on 20 October 1933, with the complaint of inability to extend his thumb. This was first described in relationship to a clasped thumb [1],[2]. It is extremely rare, however, to find cases of spontaneous elongated and narrowed pollicis longus, especially near lister tubercle. Review of the literature has revealed that simply closed ruptures of the extensor pollicis longus tendon usually appear as a consequence of fractures of the wrist joint or the carpal bones or ensue from polyarthritic changes or result from a process of degeneration [3].

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Mechanical injury of the tendon is quite rare but can be observed after direct trauma or operative treatment of a distal fracture of the radius. Closed traumatic ruptures of the extensor pollicis longus tendon in the absence of pathological changes are – in spite of the frequency of rotation injuries of the forearm – very rare [3]. Here, we report three patients with a history of no extension of thumb during the dorsiflexion of wrist and without any limitation with palmar flexion, without any specific risk factor.

#### Case Report

A 20-year-old man was first seen in 1999 with the complaint of inability to extend his fingers during dorsiflexion of wrist. He was able to extend his thumb by flexion of his wrist but unable to extend it during dorsiflexion. Interestingly, up to 2002, a 25-year-old woman and a 35-year-old man complained of the same problem. Their problem has approximately been started for 1 year. Apart from this deficit, history and physical examination did not reveal any deformity or other signs and symptoms. No previous fracture of distal third of radius or

dislocations of carpal bones or radio carpal joint was detected. They also did not have any signs or symptoms of connective tissue disorders such as rheumatoid arthritis. Complete serum examination for chemical and immunologic diagnostic factors was negative. There was no history of any systemic illness or no family history of a similar condition.

They underwent tendon transfer for correction of this extensor deficiency. At surgery, on surgical approach between second and third dorsal compartment, tendons of extensor pollicis longus present on the ulnar side of lister's tubercle was elongated and narrowed and some degrees of attrition were seen. Because of elongation, the extensor tendon was rerouted to radial side of lister's tubercle; the tendon imbricated on itself and without cutting the layers of tendon was sutured. With routine closure of subcutaneous tissue and skin, a short arm splint was applied from distal palmar crease and tip of thumb to elbow in wrist dorsiflexion and abduction of extension of thumb. Active motion was initiated at week 4. Passive, but restricted, flexion began at 7 weeks after operation. On physical examination at 3 and 12 months after surgery, the appearance and function of thumb finger were normal.

## Discussion

The most challenging aspect of this abnormality is to account for its pathogenesis. Jozsa et al [4] proposed that in the degenerative tendinopathies, an increase of the dry mass content was observed, being especially marked in calcifying tendinopathy. In the degenerated tendons, the average collagen fibre diameter had decreased, and two clearly distinct populations of thick and thin fibres were observed [4]. Although we were not able to take some specimens from our patients, we were convinced that due to some degrees of attrition, these tendons were degenerative, especially near the lister's tubercle. The features of this condition did not differ morphologically from those of rupture occurring due to degenerative changes, which were approved by microscopic investigation [5],[6]. Interestingly, unlike other studies for rupture tendon [1],[7], the underlying cause for this attrition was not found, and these tendons were not ruptured. Whether these tendons are exposed to be ruptured is debated. Tallon et al [8] reported that the general pattern of degeneration was common to the ruptured and tendinopathic tendons, but there was a statistically significantly greater

degree of degeneration in the ruptured tendons. It is therefore possible that there is a common, as yet unidentified, pathological mechanism that has acted on tendon populations [8]. From our review of the literature, it appears that this is only the first report in the English literature of attrition and elongation of thumb extensors without any rupture. But we declare to this error of our study that any hypothesis from our study was not acceptable due to lack of a photograph from the tendon as a minimum proof of evidence.

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