CASE REPORT

Bilateral closed flexor pollicis longus musculotendinous junction ruptures

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SUMMARY

We present a case of bilateral closed flexor pollicis longus musculotendinous junction ruptures. Our case suggests multifactorial aetiology and provides further evidence for genetic influences in musculotendinous junction injuries.

BACKGROUND

Closed flexor tendon ruptures of the hand are uncommon. They usually occur at the flexor digitorum profundus (FDP) bony insertion¹ and less commonly at the musculotendinous junction.

Closed musculotendinous junction ruptures in the absence of underlying disease or direct trauma are very rare with limited literature on the subject.² Furthermore, the pathogenesis of such injuries is incompletely understood.

CASE PRESENTATION

A fit and well 34-year-old male laundry worker presented with a 2-week history of inability to flex his non-dominant left thumb at the interphalangeal joint. He was a non-smoker with no previous steroid use or drug abuse. There was no history of injury or previous fractures, and he did not participate in regular sporting activities.

INVESTIGATIONS

Ultrasound scan showed a flexor pollicis longus (FPL) musculotendinous junction rupture. When the interphalangeal joint of the thumb was moved the tendon could be seen moving in the forearm confirming continuity (figures 1 and 2). The FPL muscle was, however, atrophied, suggesting a rupture at the musculotendinous junction. Doppler ultrasound scan and nerve conduction studies were normal.

TREATMENT

Intraoperatively, there was complete rupture at the FPL musculotendinous junction. On gross examination there was no evidence of attrition or other pathology. The patient was surgically treated with a ring finger flexor digitorum superficialis (FDS) tendon transfer. His thumb was placed in a resting splint postoperatively and he was started on an early active mobilisation regime a week later.

OUTCOME AND FOLLOW-UP

The patient had full return of thumb flexion and hand function. He returned to work, but 2.5 years later presented with a similar history affecting his dominant thumb, again with no obvious aetiology.

Ultrasound scan confirmed another FPL musculotendinous junction rupture and he was offered another tendon transfer but declined surgery due to personal reasons.

DISCUSSION

The weakest points along the muscle-tendon unit are known to be the bony insertion of the tendon and, less commonly, the musculotendinous junction.³ Clinically, ruptures are uncommon and typically seen in the context of trauma or underlying pathology.

Closed musculotendinous junction ruptures are proposed to occur from longitudinal traction on contracted muscles⁴ or attrition of the musculotendinous junction by nearby structures from pre-existing pathology or following trauma.⁵ In the upper limb, closed musculotendinous injuries frequently involve the rotator cuff, long extensor and long flexor muscles.⁶ In the lower limb, the Achilles tendon is the most frequently injured tendon, especially in athletic individuals or during recreational activity. The incidence of Achilles tendon rupture in the general population is known to be around 7/100 000.⁷ Several case series have reported under 10% of these ruptures to occur at the musculotendinous junction.^{7 8}

Closed flexor tendon ruptures usually occur at their bony insertion secondary to trauma or underlying pathology, such as rheumatoid arthritis.² In the upper limb, the FDP tendon is most commonly involved in closed flexor tendon ruptures. FDP tendon avulsion injuries are relatively common due to stress concentration at the hard-soft tissue interface.⁹ These injuries are also called 'jersey fingers', due to their association with sports, where the flexed distal interphalangeal joint is hyperextended or forcefully flexed against passive extension.¹⁰ The ring finger accounts for 75% of FDP tendon avulsions. FDS tendon ruptures are less common and most commonly affect the ring and middle fingers.¹¹

Closed FPL tendon ruptures are rare with only around 10 reported cases in the English literature.^{1 2} All these cases were as a result of trauma or underlying pathology.¹² Reported mechanisms included crush injuries, hyperextension injuries and after treatment of other pathologies, for example, Bennett's fracture¹³ or distal radius fractures.¹⁴

Spontaneous tendon ruptures describe a group of rare closed injuries where 'no intrinsic or extrinsic pathological processes can be macroscopically identified'.¹ A review of the current English literature shows around 60 spontaneous flexor tendon

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Figure 1 Ultrasound scan showing continuity of left flexor pollicis longus tendon at the first metacarpal. Right to left is distal to proximal.

ruptures to date.² These are most commonly reported to involve the FDP tendon with no predisposing factors, but have occasionally been linked to variations in tendon anatomy such as fusion of the ring and little finger FDP tendons side to side until the mid-palmar level.⁶ The mechanisms involved in spontaneous flexor tendon injuries are incompletely understood. They have been speculated to involve repetitive movements with microtrauma, or increased stress loading, especially when carrying out tasks involving flexion against resistance.¹⁵

We present a case of bilateral closed FPL musculotendinous junction ruptures in the absence of any obvious underlying pathological process or direct trauma. Our patient's work predominantly required repeated folding of clothes and manual transfer with no excessive force. Imbriglia and Goldstein¹⁵ have reported 10 cases of spontaneous FDP tendon ruptures of the little finger with all patients having jobs requiring repetitive motion with power grip on a routine basis. Boyes *et al*¹ found that the force producing the flexor tendon ruptures was often slight, and suggested that some underlying factors may already predispose the muscle tendon unit to rupture. Although our patient had no relevant family history and declined genetic tests, the bilateral nature of our case suggests a role for genetic influences.

It is known that there is a spectrum of genetic influences on the strength of connective tissue. Disorders of connective tissue, such as Ehlers-Danlos syndrome (EDS), have highlighted genetic mutations in two of the three chains constituting ubiquitous collagen type V α 1 (COL5A1)¹⁶ and collagen type V α 2 (COL5A2).¹⁷ Numerous mutations have been identified in rarer

variants of EDS, of particular note is the tenascin-X deficient EDS caused by mutations in the TNXB gene¹⁸ coding for tenascin glycoprotein. This decreases the messenger RNA expression of several collagen type VI genes such as COL6A1, COL6A2 and COL6A3.¹⁹

Studies suggest that various genes, such as tenascin-C (TNC), COL5A1 and matrix metalloproteinase 3 (MMP3), may be involved with musculotendinous injuries. The TNC gene encodes a glycoprotein found in tendons thought to be concerned with cell-matrix interactions.²⁰ TNC expression is regulated at the musculotendinous junction in a dose-dependent manner based on mechanical loading in tendons.²¹ The COL5A1 gene encodes the α 1 chain of the low abundance type V fibrillar collagen found in tendons. It has been widely implicated as a polymorphic association with tendon integrity in white individuals.²² The MMP3 gene codes for a protein involved in the homeostatic processes in tendons and regulates their mechanical properties. An interaction of MMP3 with COL5A1 has been proposed with regard to tendinopathy.²³

Our case presentation suggests multifactorial aetiology including repetitive microtrauma and genetic influences. Musculotendinous junction ruptures are difficult to repair directly and tendon transfer remains the best surgical option.

Learning points

- ▶ Closed musculotendinous junction ruptures in the absence of underlying pathology or direct trauma are rare and difficult to repair. Tendon transfer remains the best surgical option to restore function.
- ▶ Musculotendinous junction injuries may have multifactorial aetiology.
- ▶ Genes such as TNC, COL5A1 and MMP3, may contribute to musculotendinous junction injury susceptibility.

Contributions AQ and KYW contributed equally.

Competing interests None declared.

Patient consent Obtained.

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Figure 2 Ultrasound scan showing continuity of left flexor pollicis longus tendon in the distal forearm. Right to left is distal to proximal.

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