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Case Report

# Spontaneous Midsubstance Flexor Tendon Ruptures of the Hand

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Spontaneous midsubstance ruptures of the flexor digitorum profundus (FDP) tendon are rare and often overlooked because of their subtle presentation and lack of preceding trauma. These injuries most often involve the little finger. We report two cases of spontaneous midsubstance FDP rupture in otherwise healthy males who presented with loss of active distal interphalangeal and proximal interphalangeal joint flexion and benign examination findings. Magnetic resonance imaging in both cases demonstrated complete midsubstance rupture of the FDP tendon in the palm with an atrophic flexor digitorum superficialis. Each patient underwent primary four-strand core repair with an epitendinous running suture. One required carpal tunnel and Guyon canal release after a five-week delay to surgery. Both patients achieved excellent functional recovery with no pain and near-full range of motion at 5 months after surgery. As demonstrated by these cases, finger flexion deficits without edema or pain should prompt consideration of midsubstance FDP rupture, for which timely direct repair can yield excellent outcomes.



Spontaneous flexor tendon ruptures in the forearm and hand are defined as ruptures of the tendon substance that occur without preceding trauma or identifiable local pathology.[1] These injuries are uncommon, and midsubstance ruptures represent only a small proportion of flexor tendon tears.[2] Spontaneous midsubstance ruptures most often involve the flexor digitorum profundus (FDP) tendon of the little finger and occur in zone III.[2,3]

The etiology of these injuries remains controversial. Proposed mechanisms include degenerative tendon changes, relative hypovascularity in watershed zones near the lumbrical origin, and anatomic variations that increase stress on the little finger FDP.[2,4,5] Unlike traumatic FDP avulsions, which typically present with an edematous digit, loss of flexion, and pain along the synovial sheath, midsubstance ruptures often present more subtly, with some patients reporting only a “pop” and minimal pain or edema, which can delay diagnosis.[2,6]

We present two instances of spontaneous midsubstance rupture of the flexor tendons in the palm. Both patients consented to the use of their cases and associated images in this article.

## Case Series

### *Case 1*

A 63-year-old left-handed retired man presented with sudden inability to flex the left little finger at the distal interphalangeal (DIP) joint after extending his finger while carrying a heavy bag. He felt a pop but reported no persistent pain. There was no history of inflammatory disease, renal disease, diabetes, thyroid disease, wrist arthritis, or prior hand or wrist fracture including hook of hamate. He denied having ever used corticosteroids or other medications predisposing to tendon rupture and was not involved in any activities requiring repetitive hand use or forceful gripping prior to symptom onset.

Examination showed full, painless passive motion at the proximal interphalangeal (PIP) and DIP joints, intact flexion at the metacarpophalangeal (MCP) joint, weak active PIP flexion, and absent active DIP flexion, suggesting an atrophic, minimally functional flexor digitorum superficialis (FDS) and FDP rupture. There was no edema or bruising. Initial radiographs were negative for fracture or bony abnormality. Magnetic resonance imaging (MRI) demonstrated an intact, but atrophic, FDS and complete midsubstance FDP rupture at the level of the middle metacarpal shaft (Fig. 1).

Two weeks postinjury, the patient underwent tension-free end-to-end surgical repair of the FDP tendon. A four-strand core

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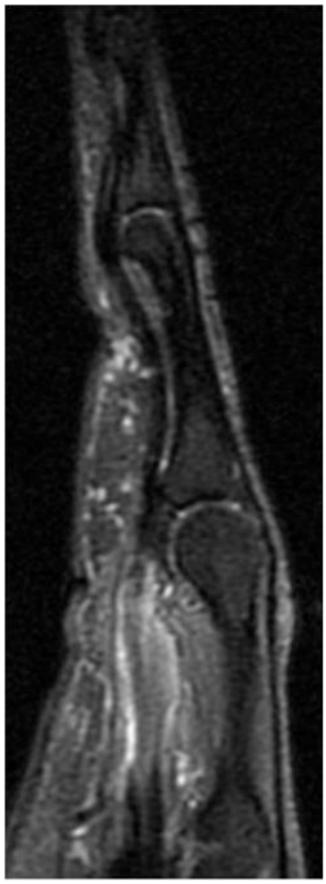

**Figure 1.** MRI demonstrating a complete rupture of the FDP tendon of the little finger.

suture repair with 2-0 FiberWire (Arthrex, Naples, FL) was performed along with a 6-0 Ethilon epitendinous running suture. The four-strand repair consisted of a modified Kessler two-strand repair with a buried knot plus another two-strand horizontal mattress repair. A carpal tunnel release was not required. The rupture was proximal to the lumbrical origin with approximately 3 cm of tendon retraction.

He was placed in a flexor tendon splint for 5 weeks and instructed to begin hand therapy with an early active range of motion protocol 3 days after surgery. At approximately 11 weeks after surgery, the patient had no pain or soreness, full finger extension, and lacked approximately 5 mm of terminal flexion to the palmar crease. At the 5-month follow-up, the patient could actively flex the DIP joint 70° and the PIP joint 95°, and he returned to his light duty job. At the final follow-up, there was no evidence of adhesions or rupture of the repair. The patient's Quick Disabilities of the Arm, Shoulder, and Hand (*Quick*DASH) score was 0 at 9 months.

*Case 2*

A 65-year-old right-handed man presented with an inability to flex the right little finger at both the DIP and PIP joints after his finger caught on plastic packaging. He denied pain, swelling, or bruising. Medical history was negative for inflammatory disease, renal disease, diabetes, thyroid disease, wrist arthritis, or prior hand or wrist fracture including hook of hamate. He denied having ever used corticosteroids or other medications predisposing to tendon rupture and was not involved in any activities requiring repetitive hand use or forceful gripping prior to symptom onset.

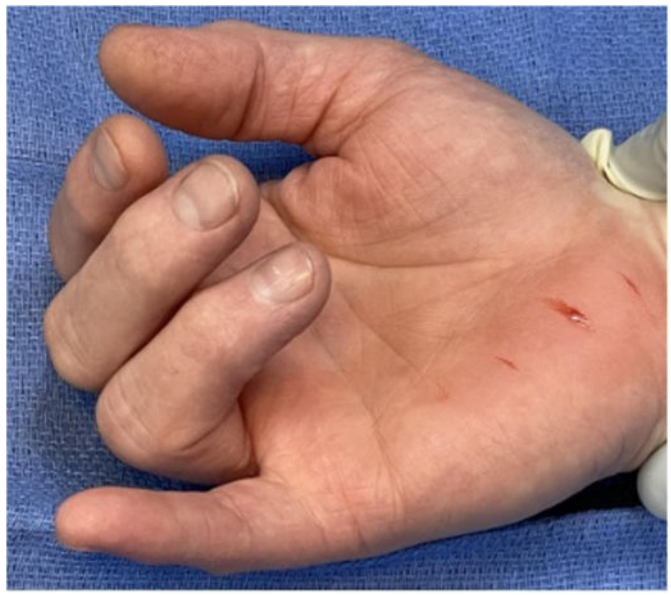

**Figure 2.** Preoperative demonstration of lack of flexion in the DIP joint. Limited flexion in the PIP joint because of atrophic FDS.

Physical examination revealed full, painless, passive motion and an absence of active PIP and DIP flexion (Fig. 2). Initial radiographs were negative for fracture and osseous abnormalities. The MRI demonstrated an intact, but atrophic FDS, which was confirmed intraoperatively, and a full-thickness disruption of the FDP tendon at the middle third of the little finger metacarpal diaphysis (Fig. 3).

Five weeks postinjury, tension-free end-to-end surgical repair of the FDP to the little finger (Fig. 4) was achieved using a similar four-strand core suture repair with 2-0 FiberWire (Arthrex, Naples, FL) along with an epitendinous running suture using 6-0 Ethilon. The proximal portion of the rupture was located at the distal carpal tunnel, necessitating release of the carpal tunnel and Guyon canal. The rupture was proximal to the lumbrical origin with 4 cm of retraction (Fig. 5).

After surgery, the patient was placed in a flexor tendon splint for 6 weeks with the MCP joints in flexion and the wrist in slight flexion. Home therapy started a few days after surgery, and formal hand therapy was started 11 days after surgery with an early active range of motion protocol. At the 2-month postoperative visit, the patient had minimal edema and was able to actively flex the little finger DIP joint 70° and PIP joint 90°, with full finger extension and no pain. Active motion was unchanged at the 5-month postoperative visit (Fig. 6), and there was no evidence of adhesions or rupture of the repair. The patient's *Quick*DASH score at 6 months was 0.

## Discussion

These two cases illustrate key features of spontaneous midsubstance FDP rupture and highlight principles that may guide diagnosis and treatment. Although flexor tendon ruptures are classically associated with forceful hyperextension, spontaneous midsubstance ruptures may occur during routine, low-energy activities such as grasping or carrying objects.[2,6] Both of our patients described only a minor incident and a popping sensation.

The little finger FDP may be predisposed to midsubstance rupture because of relatively high force transmission despite smaller cross-sectional area compared to other fingers and frequent absence or functional limitation of the FDS.[3] Anatomic

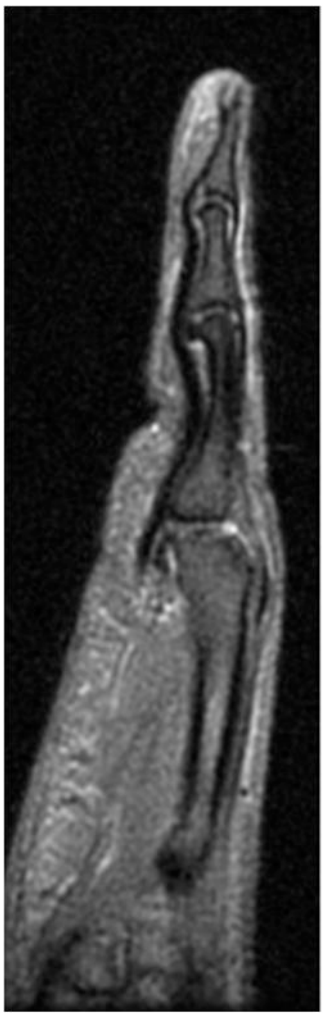

**Figure 3.** MRI demonstrating a complete rupture of FDP tendon of the little finger.

studies also suggest a hypovascular watershed zone near the lumbrical origin, which may increase susceptibility to degenerative change.[2,5] A histopathologic study of spontaneous tendon rupture frequently demonstrate hypoxic tendinopathy and mucoid degeneration, even when gross inspection appears normal, supporting a degenerative mechanism.[4]

The true spontaneity of these ruptures remains controversial. A prior literature review by Lee and McGrouther[7] suggests that many reported cases of spontaneous midsubstance flexor tendon ruptures had either an underlying disease process or undocumented trauma. In both cases presented here, systemic disease, medication exposure, prior finger triggering and/or pain in the hand or wrist, arthritis, and fracture nonunion were excluded by history, imaging, and intraoperative inspection. Intraoperatively, there were no unusual findings other than FDP tendon rupture. MRI played a critical role in both cases by localizing the rupture, assessing degree of tendon retraction, confirming FDS atrophy, and evaluating for hook of hamate nonunion, occult osteophytes, and generalized synovitis. This information allowed targeted incision planning and facilitated primary end-to-end repair, including in the delayed presentation. We found MRI to be a highly valuable adjunct. However, surgical exploration remains the gold standard.

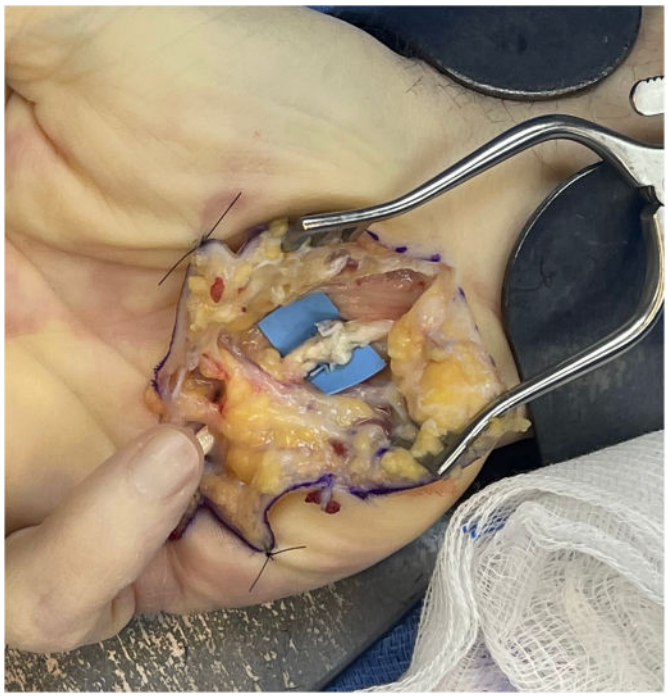

**Figure 4.** Surgical repair of the ruptured FDP tendon.

From a surgical perspective, early recognition is critical. With delay, tendon ends may retract and scar, making primary repair difficult and sometimes necessitating tendon grafting or staged reconstruction.[2,6] Prior reports describe successful management of midsubstance FDP ruptures with both tendon grafting and primary repair, though superior motion is generally observed when primary repair is possible.[2,8–10] In our series, tension-free end-to-end repair was achieved in both cases, including one repaired 5 weeks after injury, with excellent functional outcomes.

When the rupture lies near the carpal tunnel, as in our second case, concomitant carpal tunnel release—and in some cases, Guyon canal release—may be required to provide exposure and allow retraction of other vital structures such as the ring-little common digital nerve and artery and the superficial arch. In patients with a well-functioning FDS, some authors have advocated nonsurgical management of isolated FDP rupture. However, when the FDS is absent or markedly atrophic, as in both of our patients, FDP repair is important to restore finger flexion strength and avoid functional deficit.[2,9,10] At a minimum, a four-strand repair is required to initiate early active motion, with epitendinous repair and larger suture size also adding to fixation strength.

The fourth lumbrical has bipennate muscle bellies originating from adjacent radial and ulnar sides of the FDP tendons to the ring and little fingers, and the average length of origin is 13–22 mm.[11] Despite its proximity to the rupture, there are no data supporting specific treatment of injured muscle. This is likely because some fibers remain intact and the adjacent origin is preserved.

*Clinical lesson and conclusion*

In the absence of identifiable systemic, local, or medication-related risk factors, we believe these cases represent spontaneous midsubstance ruptures of the FDP tendon in the palm.

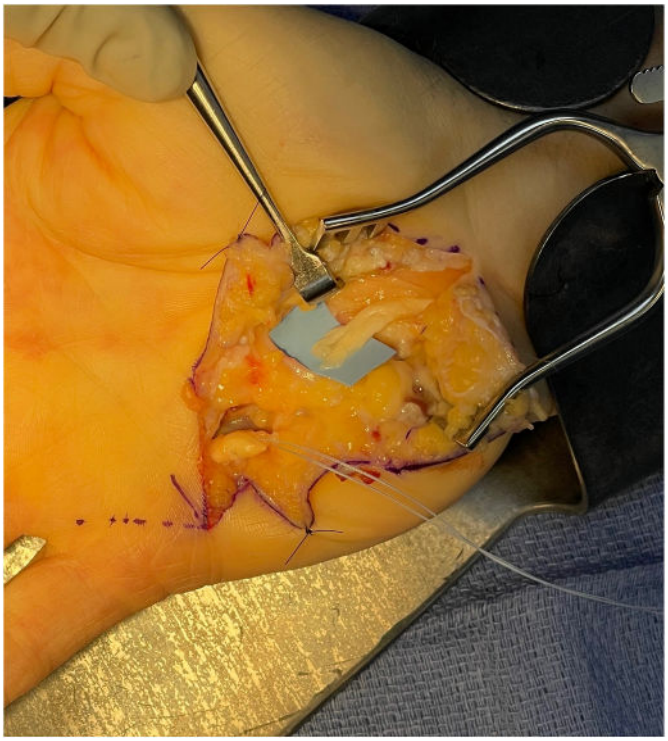

**Figure 5.** FDP rupture with 4 cm of retraction.

Histologic evaluation, which was not performed, may have provided additional support for this conclusion. Both patients presented with minimal pain, absence of edema, and preserved painless passive motion—findings that are atypical for traumatic FDP avulsion injuries. This subtle clinical presentation may contribute to delayed diagnosis and treatment, as demonstrated in case 2. Surgeons should therefore maintain a high index of suspicion for midsubstance rupture when evaluating patients with isolated finger flexion deficits, even in the absence of classic signs of tendon injury, particularly if it is the little finger in nonathletes.

If not completely sure of the injury location, an MRI can be helpful. Although others mention advanced imaging to assist in the diagnosis, we used MRI to precisely locate the pathology, minimize our incision, and optimize our ability to perform a primary repair. Although tendon grafting or reconstruction may be required with delayed presentation of more than 4–5 weeks, an attempt at primary end-to-end repair should be made whenever feasible as it can achieve excellent functional outcomes with less morbidity than reconstruction procedures. Both cases were limited by relatively short in person follow-up and formal grip strength testing was not performed. However, both patients demonstrated excellent active range of motion and *Quick*DASH scores, neither engaged in heavy physical activity, and both reported high satisfaction with their early outcomes.

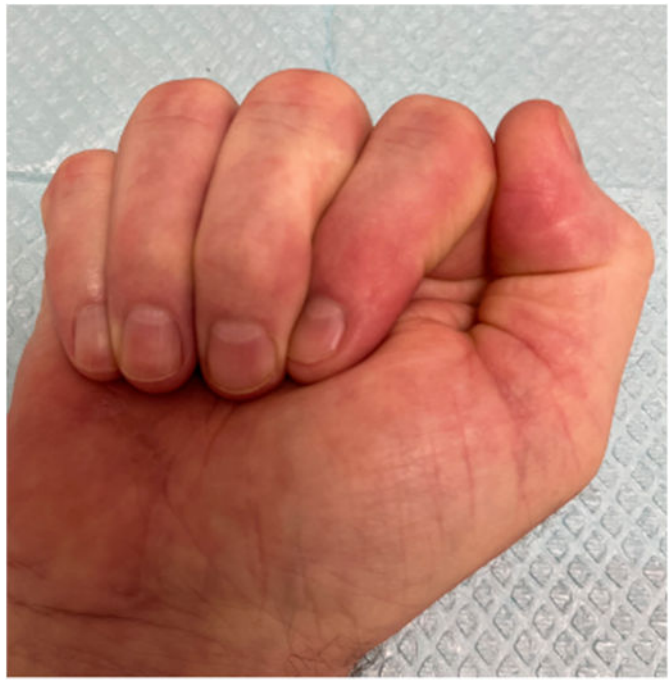

**Figure 6.** Five months postoperative demonstration of active flexion in the PIP and DIP joints.

## Conflicts of Interest

No benefits in any form have been received or will be received related directly to this article.

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