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Isolated Flexor Pollicis Longus Dysfunction Following a Radial Shaft Fracture: A Case Report and Review of the Literature

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Abstract

Isolated flexor pollicis longus (FPL) dysfunction following forearm trauma is rare and presents a diagnostic challenge, particularly when advanced imaging and electrodiagnostic studies fail to identify a definitive cause. We report a 39-year-old man who sustained a displaced radial shaft fracture from direct impact after an accidental fall. Loss of thumb interphalangeal flexion was recognised after initial immobilisation and persisted following open reduction and internal fixation. Clinical examination demonstrated isolated FPL dysfunction with preserved flexor digitorum profundus function to the index finger and intact thenar musculature. MRI demonstrated a partial brachioradialis tear with a large intermuscular fluid collection adjacent to the neurovascular bundle but no convincing FPL tendon rupture, compressive lesion, or denervation. Electrodiagnostic studies demonstrated severe superficial radial sensory neuropathy but were limited and did not directly assess the FPL or other anterior interosseous nerve-innervated muscles. Subsequent rehabilitation was complicated by persistent distal radioulnar joint pain, raising suspicion of an occult Galeazzi injury. Despite extensive investigation, the aetiology of the isolated FPL deficit remained unexplained. This case highlights the limitations of current imaging and electrodiagnostic testing in selective motor deficits following forearm trauma and emphasises the importance of serial clinical assessment.

Categories: Radiology, Orthopedics
Keywords: anterior interosseous nerve, case report, flexor pollicis longus, neuropathy, radial shaft fracture

Introduction

Anterior interosseous nerve (AIN) syndrome is a motor neuropathy classically characterised by weakness of the flexor pollicis longus (FPL), index flexor digitorum profundus (FDP), and pronator quadratus [1,2]. The FPL flexes the thumb at the interphalangeal (IP) joint; loss of this movement may result from impaired nerve supply to the muscle or a mechanical problem affecting the muscle-tendon unit. Isolated loss of active thumb IP joint flexion presents a more specific diagnostic challenge. Potential neurological explanations include selective involvement of the FPL motor branch of the AIN or a selective fascicular lesion within the median nerve; these are anatomically distinct and should not be equated with a complete AIN palsy. FPL tendon rupture or direct musculotendinous injury may produce a similar clinical presentation [1,2].

Traumatic and iatrogenic AIN palsies have been reported following forearm fractures, cast immobilisation, penetrating trauma, and operative fixation [3-6]. Although isolated FPL palsy has previously been described after radial shaft fractures, determining the site and mechanism of dysfunction can remain difficult when the clinical history and investigations are inconclusive. We report persistent isolated FPL dysfunction following open reduction and internal fixation of a proximal radial shaft fracture through a Henry approach. The case's educational contribution lies in the combination of uncertain symptom onset, nondiagnostic MRI, electrodiagnostic testing that did not directly assess the FPL, and persistent dysfunction on serial follow-up. These limitations prevented definitive localisation or confirmation of the underlying mechanism and highlight the importance of distinguishing a clinical pattern of isolated FPL weakness from a confirmed anatomical diagnosis.

Case Presentation

A 39-year-old right-hand-dominant man sustained a closed injury to his left forearm after tripping over a curb and falling directly onto the limb while overseas. Initial radiographs demonstrated a displaced proximal radial shaft fracture. The limb was immobilised in a sugar tong splint for his return to New Zealand (Figure *1*).



FIGURE 1: Initial (a) anteroposterior and (b) lateral radiographs demonstrating proximal radial shaft fracture.

The patient was placed in a temporary splint overseas for travel.

On presentation to our institution approximately one week after injury, distal perfusion was intact and routine examination documented preserved motor and sensory function in the radial, median, and ulnar nerve distributions. Passive thumb flexion and extension were well tolerated. However, active thumb IP flexion was not specifically documented, and no concern regarding isolated FPL dysfunction was recorded. An above-elbow backslab was applied, and surgery was planned for the following day.

The patient underwent open reduction and internal fixation of the radial shaft fracture as planned the following day through a standard Henry approach. Intra-operatively, the brachioradialis was mobilised, and vessels were ligated from the radial artery. The pronator teres and supinator were elevated to expose the

proximal radius. Reduction proved technically challenging because of the transverse fracture pattern, requiring provisional fixation and plate-assisted reduction. Fixation was achieved using a 3.5 mm limited contact dynamic compression plate with six cortices of fixation either side of the fracture. Final fluoroscopic imaging confirmed satisfactory alignment and implant position (Figure *2*).

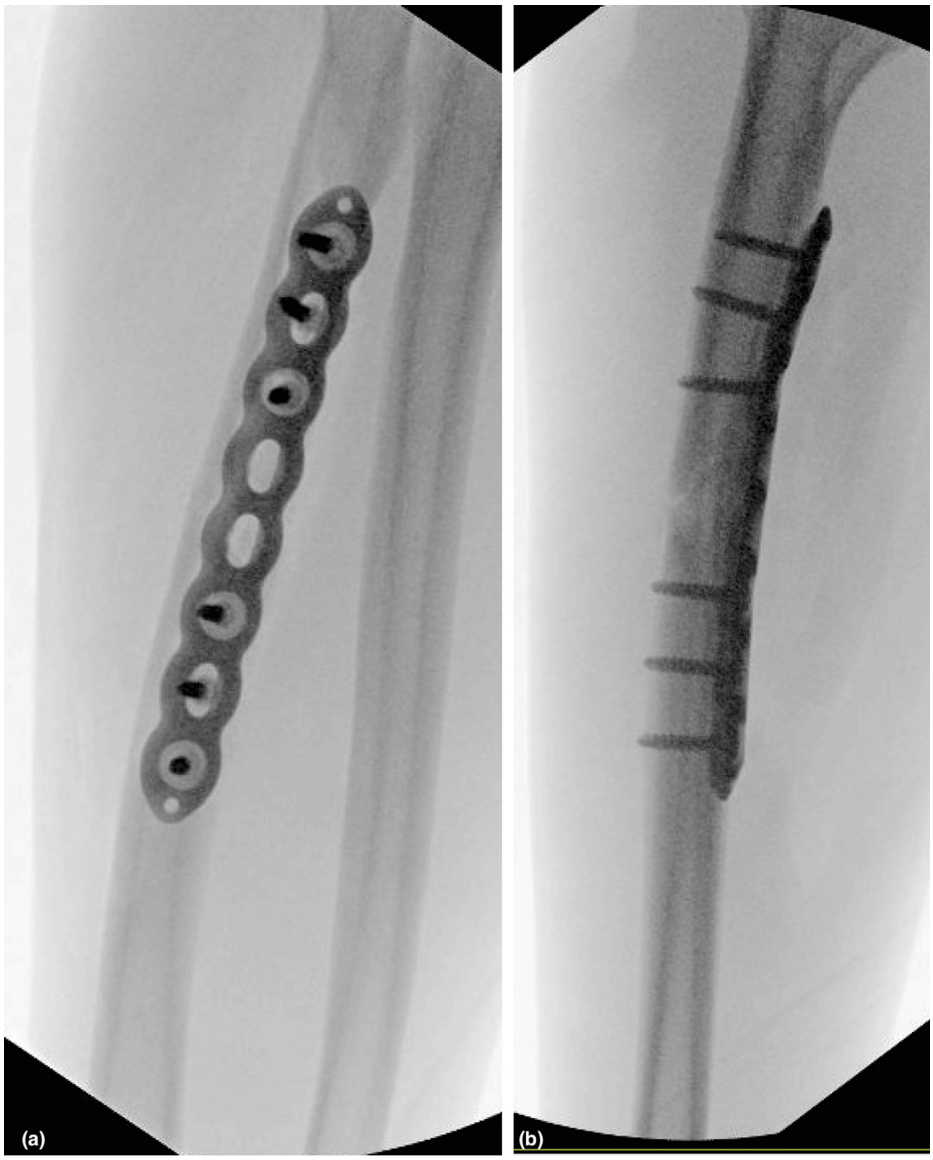


FIGURE 2: Intraoperative (a) anteroposterior and (b) lateral radiographs of radial shaft ORIF.

Satisfactory reduction and plate fixation were achieved intraoperatively.

ORIF, open reduction and internal fixation.

The patient first reported an inability to flex the thumb IP joint immediately after surgery. On retrospective questioning, he recalled that thumb flexion had been intact immediately after the injury but had become absent following application of the preoperative above-elbow plaster. His account therefore suggested onset during preoperative immobilisation, although the precise timing could not be independently confirmed. Clinical examination postoperatively demonstrated isolated absence of active thumb IP flexion, with preserved index FDP function, thenar motor function, and median nerve sensation. Mild altered sensation was present in the superficial radial nerve distribution. The loss of active thumb IP flexion persisted at early follow-up one week postoperatively.

MRI of the forearm was obtained approximately two weeks postoperatively to assess integrity of the FPL musculotendinous unit and evaluate for compressive pathology affecting the median or AIN (Figure *3*). This demonstrated a partial brachioradialis tear with an extensive intermuscular fluid collection extending

between brachioradialis and pronator teres adjacent to the neurovascular bundle. Mild oedema was present within the FPL and pronator quadratus. Metal artefact limited assessment of the proximal FPL tendon, but no convincing tendon rupture, compressive lesion, or denervation changes were identified.

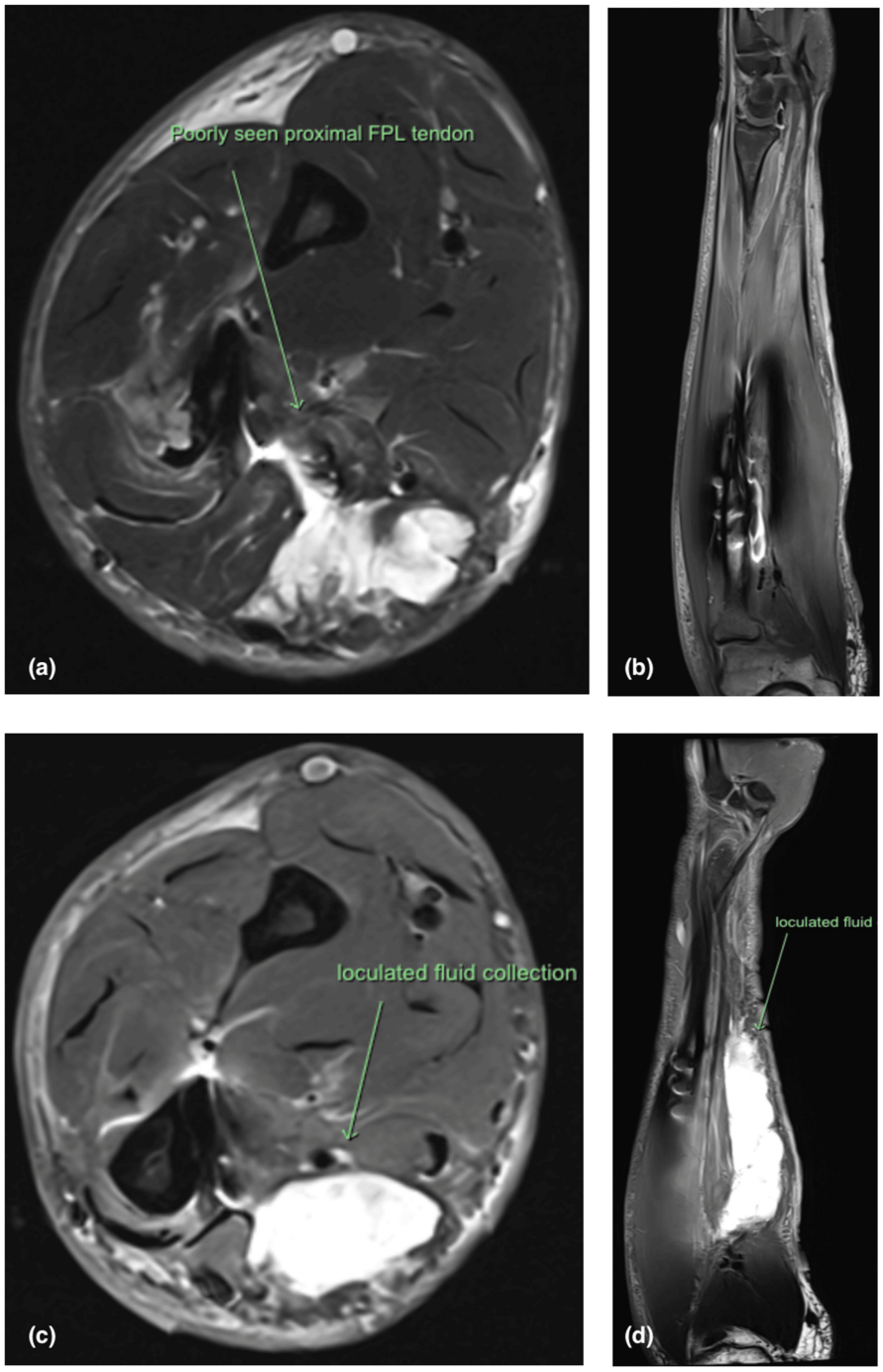


FIGURE 3: Postoperative MRI imaging of the left forearm.

Representative MRI images demonstrating poor visualisation of the proximal flexor pollicis longus tendon on axial (a) and sagittal (b) views, and a postoperative loculated fluid collection between the brachioradialis and pronator teres on axial (c) and sagittal (d) views.

FPL, flexor pollicis longus; MRI, magnetic resonance imaging.

Electrodiagnostic studies, performed approximately six weeks postoperatively, demonstrated markedly reduced left superficial radial sensory amplitude, consistent with severe superficial radial sensory neuropathy (Table *1*). Recorded limb temperatures ranged from 33.1°C to 34.7°C where documented. Needle

electromyography (EMG) was limited by the recent surgical scar and bandaging/splinting. No additional technical limitations affecting nerve conduction testing were documented, and the report did not state whether such factors were considered in interpretation.

Nerve/study	Stimulation site	Recording site	Latency (ms)	Peak amplitude (μV)	Distance (cm)	Velocity (m/s)	Temp. (°C)
R median – digit II	Wrist	Digit II	3.31	29.5	13	47.6	33.5
L median – digit II	Wrist	Digit II	2.83	32.5	13	58.9	34.1
R ulnar – digit V	Wrist	Digit V	2.65	25.7	11	58.0	33.3
L ulnar – digit V	Wrist	Digit V	2.38	22.6	11	61.4	33.5
R radial – thumb	Forearm	Thumb	1.96	40.9	10	69.6	33.9
L radial – thumb	Forearm	Thumb	3.29	1.5	10	37.2	34.7

TABLE 1: Selected sensory nerve conduction findings.

The left radial sensory response showed a markedly lower amplitude (1.5 μV) than the right (40.9 μV). This finding relates to the associated sensory disturbance and does not establish the cause of the FPL motor deficit. Laboratory-specific reference ranges were not provided in the original neurophysiology report and were therefore unavailable for inclusion. Latencies and conduction velocities are reproduced as reported. The source report labels the sensory latency column 'Lat. 2' without specifying whether this represents onset or peak latency.

FPL, flexor pollicis longus; L, left; R, right.

Routine median, ulnar, and radial motor conduction parameters were preserved (Table *2*).

Nerve/muscle	Stimulation site	Latency (ms)	Amplitude (mV)	Distance (cm)	Velocity (m/s)	Temp. (°C)
R median – APB	Wrist	3.92	6.3	—	—	—
R median – APB	Elbow	8.23	5.3	22	51.0	33.3
L median – APB	Wrist	3.15	4.5	—	—	—
L median – APB	Elbow	7.52	4.1	23	52.6	34.0
R ulnar – ADM	Wrist	2.44	10.8	—	—	—
R ulnar – ADM	Below elbow	6.56	10.6	23	55.8	—
R ulnar – ADM	Above elbow	8.56	10.3	11.5	57.5	33.1
L ulnar – ADM	Wrist	2.06	9.8	—	—	—
L ulnar – ADM	Below elbow	5.92	8.7	22	57.1	—
L ulnar – ADM	Above elbow	8.08	8.8	12	55.4	—
L radial – EDC	Elbow	1.98	6.3	—	—	—
L radial – EDC	Upper arm	3.81	6.7	12	65.5	—

TABLE 2: Motor nerve conduction studies.

Routine motor conduction studies recorded responses from APB, ADM, and EDC. These recordings did not directly assess the FPL motor branch or establish the integrity of the AIN. The radial motor study recording from the EDC did not assess FPL function. Laboratory-specific reference ranges were not provided in the original neurophysiology report and were therefore unavailable for inclusion.

Em dashes indicate fields left blank in the original report.

ADM, abductor digiti minimi; AIN, anterior interosseous nerve; APB, abductor pollicis brevis; EDC, extensor digitorum communis; FPL, flexor pollicis longus; L, left; R, right.

Needle EMG showed no active denervation in the sampled muscles (Table *3*). The examination was limited by the recent surgical scar and bandaging/splinting. Neither FPL nor other AIN-innervated muscles were sampled; therefore, these findings did not exclude an isolated FPL branch neuropathy or anterior interosseous neuropathy.

Muscle	Nerve	Roots	IA	Fib.	PSW	Fasc.	H.F.	Amp.	Dur.	PPP	Recruitment
L deltoid	Axillary	C5-C6	N	None	None	None	None	N	N	N	N
L triceps brachii	Radial	C6-C8	N	None	None	None	None	1+	N	N	N
L biceps brachii	Musculocutaneous	C5-C6	N	None	None	None	None	N	N	N	N
L pronator teres	Median	C6-C7	N	None	None	None	None	N	N	N	N
L extensor digitorum communis	Radial	C7-C8	N	None	None	None	None	N	N	N	N
L first dorsal interosseous	Ulnar	C8-T1	N	None	None	None	None	N	N	N	N

TABLE 3: Needle electromyography.

No fibrillation potentials or positive sharp waves were recorded in the sampled muscles. FPL, index FDP, and pronator quadratus were not sampled; therefore, these findings could not exclude selective FPL motor branch or AIN neuropathy.

The '1+' entry for left triceps brachii motor unit amplitude is reproduced as recorded in the original report, which did not define the grading convention.

AIN, anterior interosseous nerve; Amp., amplitude; Dur., duration; Fasc., fasciculations; FDP, flexor digitorum profundus; Fib., fibrillation potentials; FPL, flexor pollicis longus; H.F., high-frequency discharges; IA, insertional activity; L, left; N, normal; PPP, polyphasic potentials; PSW, positive sharp waves.

The patient commenced active physiotherapy and hand therapy four weeks postoperatively to aid functional recovery. At approximately six weeks postoperatively, following consultant review after the electrodiagnostic assessment, continued observation of the FPL deficit was planned because the investigations had not identified an obvious cause.

Ultrasound performed approximately two months postoperatively demonstrated FPL tenosynovitis with A1 pulley thickening and triggering, together with focal tendon thickening at the level of the thumb metacarpophalangeal joint. The relationship of these findings to the initial acute loss of thumb IP flexion remained uncertain.

Serial radiographs at three and five months postoperatively demonstrated stable fixation and delayed union progressing towards healing (Figure *4*).



FIGURE 4: Postoperative forearm radiographs at three and five months.

Anteroposterior (a) and lateral (b) radiographs obtained at three months postoperatively, and anteroposterior (c) and lateral (d) radiographs obtained at five months postoperatively.

At approximately six months postoperatively, follow-up findings suggested a return of active FPL function, with resolving thumb triggering documented clinically. Formal Medical Research Council (MRC) grading of FPL strength and goniometric measurement of active thumb IP joint range of motion were not recorded, limiting assessment of the extent of recovery. In view of the clinical improvement, repeat targeted electrodiagnostic testing was not pursued. The underlying cause of the initial loss of flexion remained uncertain.

Discussion

AIN syndrome is rare, accounting for less than 1% of upper limb compression neuropathies [1-2,7]. The AIN typically arises from the median nerve 5-8 cm distal to the lateral epicondyle and supplies the FPL, the radial portion of FDP, and pronator quadratus [1,2]. The motor branch to FPL usually arises approximately 4 cm distal to the origin of the AIN, promptly dividing into several branches that enter the proximal muscle along its medial border, close to the radius [4]. This anatomy may render the FPL motor branches particularly vulnerable to local trauma, traction, or compression.

Complete AIN palsy classically produces weakness of thumb IP joint flexion, index finger distal IP joint flexion, and forearm pronation with the elbow flexed [2,7]. Incomplete palsies caused by selective fascicular

or motor branch involvement are less common, with isolated FPL dysfunction representing a particularly unusual presentation [1,3,4]. Anatomical variation in FDP innervation may also partially account for preservation of index FDP function in some patients with AIN lesions [4]. Therefore, preserved index finger flexion does not exclude a selective fascicular lesion within the median nerve or an FPL motor branch neuropathy.

The pathophysiology of AIN palsy remains debated. Proposed mechanisms include inflammatory neuritis, traction, focal compression, fracture-related soft tissue injury, postoperative oedema or haematoma, operative retraction, and direct nerve trauma [2,5,7]. In the present case, the deficit was first reported immediately after surgery, although the patient retrospectively recalled its onset during preoperative immobilisation. His account raises the possibility of evolving neurapraxia related to fracture displacement, traction, haematoma, or progressive soft tissue swelling. However, uncertainty regarding the timing and reliability of retrospective recall prevents confident attribution to a traumatic or perioperative mechanism.

Several traumatic and postoperative cases provide useful comparisons. Haflah et al. described isolated FPL palsy following a closed proximal radial fracture, with electrodiagnostic evidence of acute AIN neuropathy and full recovery after two months [4]. Salkinder and Liebenberg reported isolated FPL dysfunction following plating of a both-bone forearm fracture, attributed to traction neurapraxia, with spontaneous recovery after 10 weeks [5]. More recently, Qadi et al. reported isolated FPL weakness following proximal radial shaft fixation, supported by EMG and nerve conduction studies, with recovery by approximately 16 weeks [6]. Campbell et al. similarly described isolated FPL palsy following a radial shaft fracture, with electrodiagnostic evidence of an isolated FPL neuropathy and near-complete recovery at six months [3]. In our patient, the deficit persisted at five months, with follow-up findings suggesting returning FPL function at six months. However, its precise onset and anatomical basis remained uncertain.

Electrodiagnostic studies demonstrated superficial radial sensory neuropathy but did not establish the cause of the isolated FPL dysfunction. Routine median motor conduction was normal; however, this assessed responses from abductor pollicis brevis and did not directly evaluate the AIN or its branch to FPL. Needle EMG was limited by the recent surgical scar and splinting and did not include FPL, pronator quadratus, or index FDP. The absence of active denervation in the sampled muscles therefore could not exclude a selective FPL motor branch neuropathy or a selective fascicular lesion within the median nerve. Routine nerve conduction studies may be falsely reassuring when an abnormality is confined to a single distal motor branch [5]. Aljawder et al. reported electrodiagnostically confirmed AIN syndrome despite the absence of a compressive lesion on MRI, illustrating the complementary roles of structural imaging and neurophysiology [8]. In our patient, electrodiagnostic sampling was incomplete and MRI assessment was limited by metal artefact; consequently, a neurological mechanism remained possible but unconfirmed.

The principal differential diagnoses were a selective AIN fascicular or FPL motor branch palsy, occult FPL musculotendinous injury, tendon tethering, and postoperative adhesion. Preserved passive thumb movement excluded joint contracture but did not distinguish neurological dysfunction from musculotendinous disruption. MRI demonstrated no definite tendon discontinuity or compressive lesion; however, metal susceptibility artefact limited assessment of the proximal FPL musculotendinous junction. Consequently, neither a selective motor neuropathy nor an occult musculotendinous injury could be conclusively excluded. The associated partial brachioradialis tear, extensive forearm haematoma, and superficial radial sensory neuropathy were consistent with a substantial soft tissue injury sustained at the time of the initial trauma.

Persistent distal radioulnar joint pain and restricted forearm rotation also raised concern for an occult Galeazzi-type injury. Although this concern did not directly explain the isolated FPL dysfunction, it highlighted the need to consider associated distal radioulnar joint injury. Subsequent ultrasound demonstrated FPL tenosynovitis and A1 pulley thickening and triggering. These distal abnormalities may have contributed to pain and mechanical restriction during rehabilitation, although their relationship to the initial loss of active thumb flexion remained uncertain.

Management of incomplete AIN palsy remains controversial. In a retrospective series of 14 patients, Ulrich et al. reported spontaneous recovery between 3 and 12 months in all eight patients managed conservatively, with no difference in final functional outcome between operative and nonoperative groups [7]. Published traumatic cases have similarly demonstrated recovery over periods ranging from approximately two to six months [3-6]. Initial observation is therefore reasonable when tendon continuity is preserved, and there is no evidence of nerve transection or a surgically remediable compressive lesion. However, persistent paralysis warrants serial clinical and electrodiagnostic assessment, particularly when there is no evidence of reinnervation.

Isolated FPL dysfunction following radial shaft fracture has previously been described by Campbell, Haflah, Salkinder, and Qadi [3-6]. The contribution of the present case is its illustration of diagnostic uncertainty rather than confirmation of a new mechanism. The deficit was first reported postoperatively, but the patient retrospectively recalled onset during preoperative immobilisation. Preserved routine median motor recordings did not directly assess FPL function, targeted needle EMG sampling was incomplete, and MRI

assessment was limited by metal artefact. Subsequent ultrasound identified tendon and pulley abnormalities whose relationship to the initial deficit remained uncertain. As a teaching case, it highlights the importance of documenting active thumb IP flexion before and after immobilisation and fixation, distinguishing routine median nerve testing from targeted assessment of AIN-innervated muscles, and acknowledging uncertainty when neurological and mechanical explanations remain unresolved.

Conclusions

Isolated loss of FPL function following a radial shaft fracture presents a diagnostic challenge. In this case, the patient retrospectively reported onset during preoperative immobilisation, although the precise timing remained uncertain. Selective motor neuropathy remained unconfirmed, and occult musculotendinous injury or mechanical restriction could not be excluded. This case highlights the importance of documenting thumb IP flexion before and after immobilisation and fixation, performing serial clinical examinations, and recognising the limitations of imaging and electrodiagnostic investigations that do not adequately assess the affected structures.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Ampili E. Mathews

Acquisition, analysis, or interpretation of data: Ampili E. Mathews, Sanka Bambarawana

Drafting of the manuscript: Ampili E. Mathews

Critical review of the manuscript for important intellectual content: Ampili E. Mathews, Sanka Bambarawana

Supervision: Ampili E. Mathews, Sanka Bambarawana

Disclosures

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