

CASE REPORT

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Anterior interosseous nerve palsy mimicking rupture of the index flexor digitorum profundus after volar locking plate fixation of a distal radius fracture

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ABSTRACT

We describe the case of a patient with distal radius fracture who became unable to flex the distal interphalangeal joint of the index finger after internal fixation using a volar locking plate. There was palpable crepitus with active thumb motion, and wrist radiographs showed prominence of the volar plate at the watershed line. Therefore, our initial diagnosis was plate-induced closed rupture of the flexor digitorum profundus tendon of the index finger. However, upon surgical removal of the plate, no tendon rupture was found. Magnetic resonance imaging after plate removal showed diffuse increased signal intensity in the index flexor digitorum profundus on T2-weighted fat-suppressed images, which indicated muscle denervation. Based on the above findings, we changed the diagnosis to anterior interosseous nerve palsy with isolated paralysis of the flexor digitorum profundus of the index finger. Finger flexion disability following volar plate fixation of distal radius fracture should always be investigated carefully..

Keywords: anterior interosseous nerve palsy, distal radius fracture; flexor digitorum profundus, index finger, volar locking plate

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INTRODUCTION

Distal radius fracture represents the most common type of upper limb fracture. Volar locking plate fixation has become an increasingly popular technique for managing distal radius fracture because it ensures fixation, provides excellent stability, and maintains anatomic reduction. However, volar locking plate fixation carries a significant risk of complications related to flexor tendon injury, with several reports describing partial and/or complete tendon rupture of the flexor pollicis longus (FPL) and flexor digitorum profundus (FDP) of the index or long finger (FDP1 and FDP2, respectively) following plate fixation of distal radius fractures.¹⁻³⁾ Another significant condition affecting the functioning of these muscles is anterior interosseous nerve (AIN) palsy, which represents weakness or motor loss of the FPL, FDP1, pronator quadratus, and, occasionally, the FDP2.⁴⁾ Therefore, it is sometimes relevant to perform a differential diagnosis between AIN palsy and flexor tendon rupture caused by plate prominence.

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We encountered a patient with unstable distal radius fracture who became unable to flex the distal interphalangeal (DIP) joint of the index finger following internal fixation using a volar locking plate system. There was palpable crepitus on the volar aspect of the wrist with active thumb motion, and radiographs of the wrist showed prominence of the volar plate at the watershed line. Therefore, it was necessary to differentiate between AIN palsy and closed rupture of the FDP1 tendon caused by plate prominence.

CASE PRESENTATION

A 33-year-old man presented to a regional hospital with an injury to his left wrist following a hang gliding accident. The initial radiographs showed intra-articular distal radius fracture (AO type C2). Five days later, the patient underwent open reduction and internal fixation with a volar locking plate system. At 27 days after surgery, the patient presented to the hospital complaining of the inability to flex the DIP joint of the left index finger. He did not experience prior upper limb pain. He was referred to our hospital 20 days after the onset of these symptoms.

On initial examination, there was palpable crepitus on the volar aspect of the wrist with active thumb motion. The patient was unable to flex the DIP joint of the left index finger but showed no dysfunction regarding active thumb flexion (Fig. 1). Although it was difficult to evaluate the strength of the pronator quadratus muscle due to operative fixation with a volar locking plate, all median nerve-innervated muscles other than FDP1 and pronator quadratus revealed normal motor function (manual muscle test (MMT) score, = 5). There was no sensory deficit. Radiographs of the left wrist showed prominence of the volar plate at the watershed line (Fig. 2). A computed tomography scan showed that the ulnar distal end of the plate was located 4 mm above the volar surface of the cortical bone (Fig. 3). We initially established the diagnosis of closed rupture of the FDP1 tendon caused by plate prominence. Since bone union was confirmed on radiograph at

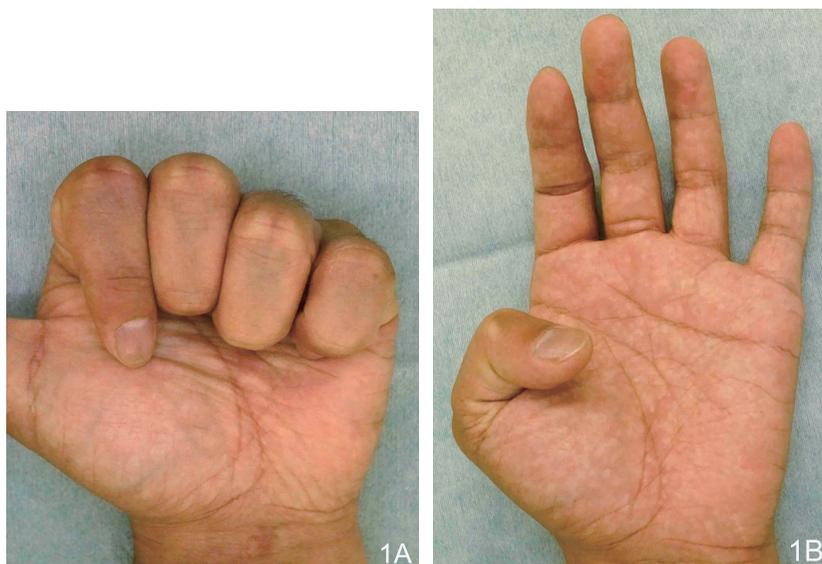


Fig. 1 The patient was unable to perform active flexion of the distal interphalangeal joint of the left index finger (A), but encountered no difficulty with active thumb flexion (B)

11 weeks after the original surgery, we removed the plate with the intention to perform tendon transfer. However, intraoperative findings revealed that the FDP1 tendon was not ruptured, despite the prominence of the distal edge of the plate and the presence of flexor tenosynovitis. Muscle



Fig. 2 Posteroanterior and lateral radiographs of the affected wrist showing prominence of the volar plate at the watershed line.

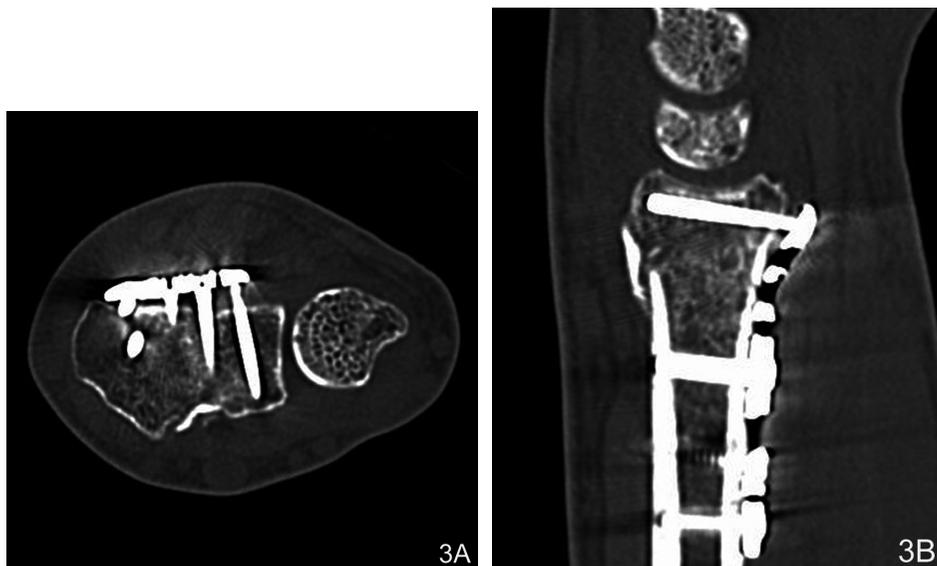


Fig. 3 Axial (A) and sagittal (B) computed tomography images showing that the ulnar distal end of the plate was located 4 mm above the volar surface of the cortical bone.

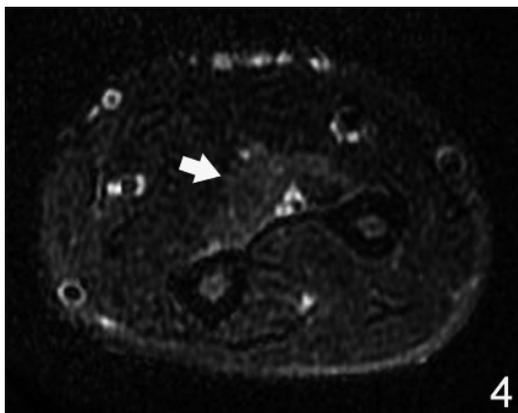


Fig. 4 Magnetic resonance imaging of the forearm showing diffuse increased signal intensity in the flexor digitorum profundus muscle (arrow) of the index finger on T2-weighted fat-suppressed images.

tonus of the FDP1 decreased in comparison with the tonus of the FDP in the other fingers. Magnetic resonance imaging of the forearm after plate removal showed diffuse increased signal intensity in the FDP1 on T2-weighted and T2-weighted fat-suppressed images, which indicated muscle denervation (Fig. 4). Based on the above findings, we changed the initial diagnosis to AIN palsy. Because the patient did not improve, we performed surgical exploration of the median nerve and AIN at 3.5 months after the onset of symptoms, which indicated no obvious extrinsic compression of the nerve but revealed a 2-cm fibrotic region on the median nerve at the level of the medial epicondyle. We dissected the thick and hard epineurium and performed interfascicular neurolysis. We noted no hourglass-like fascicular constriction. Recovery from motor paralysis began at 2 months after neurolysis, and was completed 6 months later.

DISCUSSION

AIN palsy is a relatively uncommon condition and is manifested as partial or complete paralysis of the FPL, FDP1, pronator quadratus, and often the FDP2.⁵⁾ Patients usually experience pain in the region of the elbow before the onset of the palsy.⁴⁾ Paralysis of the FPL and FDP1 is not always simultaneous; while this phenomenon is relatively rare, there are reports of isolated FDP1 paralysis. Werner⁶⁾ revealed concomitant paralysis in 34 patients, isolated FPL paralysis in 25 patients, and isolated FDP1 paralysis in 10 patients. Furthermore, Seror⁷⁾ indicated that, while complete lesions of the AIN involving both the thumb and index finger are well known by specialists, incomplete AIN lesions with isolated involvement of a single finger are often misdiagnosed.

Complications of volar plate fixation for distal radius fractures have been reported, often including flexor tendon injury.¹⁻³⁾ Valbuena *et al.*¹⁾ described 5 cases of flexor tendon rupture after volar plate fixation for distal radius fracture, noting that only the FPL tendon was ruptured in three patients, both the FDP1 and FDP2 tendons were ruptured in one patient, and only the FDP1 tendon was ruptured in one patient. Soong *et al.*²⁾ noted that implant prominence at the watershed line may increase the risk of flexor tendon injury. Tada *et al.*⁸⁾ reported that subdermal crepitus with active thumb motion was considered as the risk factor for tendon attrition. Yamazaki *et al.*⁹⁾ conducted a prospective clinical cohort study assessing the risk of tendon attrition following

treatment of distal radius fractures with volar locking plates, and demonstrated that the findings of crepitus and volar plate positioning were independent risk factors for flexor tendon attrition. In the case we described here, there was palpable crepitus on the volar aspect of the wrist with active thumb motion, and radiographs of the wrist demonstrated prominence of the volar plate at the watershed line. Additionally, there was no prior upper limb pain and the patient was unable to flex only the DIP joint of the left index finger. Therefore, we initially set the diagnosis of closed rupture of the FDP1 tendon, caused by plate prominence. However, this diagnosis was not consistent with the early onset of symptoms at 27 days after surgery. Most flexor tendon ruptures following volar plate fixation of distal radius fractures develop at ≥ 4 months after surgery.³⁾ Our patient experience strongly suggests that, in order to distinguish between flexor tendon rupture and neuropathy, it is important to investigate the tenodesis effect of the flexor tendons, which involves spontaneous finger flexion during wrist extension.

Staff *et al.*¹⁰⁾ described 33 patients with post-surgical inflammatory neuropathies caused by inflammatory mechanisms. Post-surgical neuropathy is typically attributed to the mechanical forces of stretch, compression, contusion, or transection of nerves during surgery. However, post-surgical inflammatory neuropathy is caused by the immune system attacking the nerves, and it is difficult to explain based on mechanical factors because the phenomenon is either spatially or temporally segregated from the surgery. All post-surgical inflammatory neuropathies develop within 30 days of a surgical procedure and are further categorized into focal, multifocal, or diffuse patterns. In the case described here, paralysis developed 27 days after surgery and there was no prior upper limb pain, whereas most patients experience upper limb pain as an early symptom. This type of paralysis may originate from focal post-surgical inflammatory neuropathy. Based on our experience, we recommend that, when encountering thumb or finger flexion disability following volar plate fixation of distal radius fractures, it is necessary to consider the possibility of neuropathy.

Informed consent for publication of clinical data and photographs has been obtained from the patient.

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