Isolated flexor pollicis longus nerve fascicle lesion – a rare differential diagnosis of thumb flexion deficiency

Abstract

A rare differential diagnosis of thumb flexion deficiency is an isolated flexor pollicis longus (FPL) nerve fascicle lesion. We present a 42-year-old otherwise healthy female patient who developed a weak thumb-to-index pinch and deficient right thumb flexion following the removal of osteosynthesis plates after a forearm fracture. Clinically, the flexor pollicis longus function was absent, yet index flexion and sensibility were unimpaired. Tendon rupture was excluded using a tenodesis test and the electro-physiological result of isolated interosseus nerve fascicle lesion was confirmed intraoperatively by inspection and electrostimulation. Tendon transfer using the extensor carpi radialis longus reconstruct strong thumb flexion during pinch. In summary, due to its specific location and anatomy, the FPL branch is more prone to isolated neuropathy, e.g. by injections or operations, than to other fascicles of the anterior interosseus nerve. When confronted with sudden and isolated thumb flexion deficiency, specialists should be aware of this rare phenomenon.

Keywords: anterior interosseous nerve, thumb flexion, flexor pollicis longus, isolated

Zusammenfassung


Schlüsselwörter: Nervus interosseus anterior, Daumenendgelenksbeugung, Flexor pollicis longus, isoliert
Introduction

Loss of pinch power and thumb flexion can be caused by many traumatic or atraumatic origins, most frequently by carpal tunnel syndrome, flexor tendon injuries or anterior interosseous nerve (AIN) compression. A rare, but relevant differential diagnosis is an isolated flexor pollicis longus (FPL) nerve fascicle lesion. The objective of this paper is to illustrate this phenomenon with a clinical case and provide a discussion of its anatomical, clinical background and differential diagnosis of incomplete AIN syndrome in the literature.

Clinical case

A 42-year-old otherwise healthy secretary was referred to our hand surgery department with the diagnosis of carpal tunnel syndrome for operative carpal tunnel release of the non-dominant left hand. She presented with weakened pinch and impaired control of the right dominant thumb due to deficient interphalangeal flexion. Symptoms had started after the removal of osteosynthesis plates about a year after a complicated ulna and radius fracture in 2008. Clinically, FPL muscle function was entirely lost while index finger flexion was intact (incomplete O-sign) (Figure 1) and sensibility was clinically unimpaired with a two-point discrimination of 5–6 mm.

The FPL tendon was found intact on ultrasound but an atrophy of the muscle belly was noted. A tenodesis test furtherly excluded a rupture of the FPL tendon. Neuro-electrophysiology revealed preserved interosseus nerve function, yet complete paralysis of FPL muscle which could be confirmed during intraoperative inspection and electro-stimulation of the muscle. Due to the atrophy of the FPL several years after denervation, tendon transfer was performed to restore FPL function. The extensor carpi radialis longus (ECRL) was chosen due to its proximity, favourable myo-architectural properties and synergistic action.

At follow-up after 15 months postoperatively the patient demonstrated interphalangeal flexion with fine motor skills and and pinch of 7 kg restored (Figure 2).

Discussion

This case report illustrates an exceptional, but anatomically interesting and clinically relevant cause of sudden loss of thumb interphalangeal flexion. Thumb to index pinch may become deficient due to a great variety of causes, most obviously by tendon ruptures due to acute trauma, inflammation, e.g. rheumatoid arthritis, degeneration caused by exostosis with pseudoarthrosis of the scaphoid bone, necrosis of the lunate bone, stenosing tendovaginitis or trigger finger and tenodesis sequelae [1]. Lesions of the anterior interosseus nerve (AIN) can result in various clinical manifestations, depending on location and degree of axonal damage. In 1918, Tinel [2] first described paralysis of the AIN and in 1957, Turner and Parsonage [3] mentioned six cases of the syndrome in a review of 136 patients with neuralgic amyotrophy. In 1952, Kiloh and Nevin [4] published two cases of the syndrome as isolated neuritis which until today bears their names (Kiloh-Nevin syndrome). The main clinical sign is an awkward pinch grip and inability to form a circle (or O), by flexing the interphalangeal joints of the thumb and the distal interphalangeal joint of the index finger. Especially in incomplete AIN paralysis where this pathognomonic sign may be partly absent, it has been described as difficult to distinguish between flexor tendon ruptures interosseus anterior neuropathy [5]. Misinterpretation of the clinical picture, especially in post-traumatic situations, may even lead to unnecessary operations [6], as illustrated by two cases presented by Joist et al. [7]: while in the first case of suspected interosseous nerve compression, a spontaneous rupture of flexor pollicis longus was detected intraoperatively, in the second patient, surgical exploration of flexor pollicis longus tendon and profundus tendon to index finger was performed due to suspected rupture, but revealed intact tendons and only a second operation with neurolysis of the interosseus anterior nerve achieved full recovery of thumb-index
pinch. Notably, simple clinical tests regarding the tenodesis effect at the interphalangeal joint were described by Mody [8] and Melton et al. [9] and can rule out tendon discontinuity, as we could in our patient. Diagnosis and decision-making may still be challenging, above all in incomplete anterior interosseous nerve syndrome. Seror [10] reported on 17 cases of abnormal pinch due to anterior interosseous nerve (AIN) palsy. The AIN lesion was complete with FPL and flexor digitorum profundus of the index (FDP 2) in nine, while isolated lesion of either the FPL or FDP 2 was observed in four cases each. Three of those eight cases were initially considered to be tendon ruptures. Electro-diagnosis assessed AIN lesion and respect of the main median trunk in all cases. Pronator quadratus examination provided the diagnosis in 14 of 17 cases and FPL or FDP 2 testing revealed the diagnosis in the remaining three cases. Additional nerve lesions were documented in four cases. Pinch grip spontaneously recovered in nine of ten cases. The AIN lesion was due to compression in three cases and to mononeuities such as Parsonage Turner neuralgic amyotrophy in 14 cases. As two out of three cases of compression resolved spontaneously, the author discouraged surgical exploration before 12 to 16 months when the lesion is not clearly traumatic or with evidence of compression. This conservative approach is in line with other studies with recovery rates of 70 percent without operations [11]. Seki et al. [12] studied 21 patients aged between 17 and 65 (mean, 39) years with spontaneous onset of AIN palsy. Pain around the elbow or another region (forearm, shoulder, upper arm, systemic arthralgia) was documented in 17 patients, typically lasting for two to three and always ending within six weeks. In ten cases the palsy began after the pain went away. Complete palsy occurred in 13 cases, isolated palsy of FPL in five. All patients were treated non-operatively using antiinflammatory agents and physiotherapy. The mean time to initial muscle recovery was nine months in FDP 2 palsy and ten months for FPL palsy. Grade 4 muscle strength or better was seen in 15 patients with a FDP 2 palsy and in 16 of 18 with palsy of the FPL. Patient age was strongly correlated with recovery, as recovery occurred within 12 months in all patients under 40 years who achieved a final British Medical Research Council grade of 4 or better. Consequently, surgical decompression seemed unnecessary in young individuals with anterior interosseous syndrome. In our patient with onset of isolated FPL palsy immediately postoperatively, we assumed a traumatic origin. Dolderer et al. [13] have investigated the anatomical reason for isolated neuropathy of the FPL branch. As they could demonstrate by dissection and immune-histochemical staining, the FPL fascicle runs within the anterior interosseous nerve in a common epineurium, but is located on the outer aspect without interneural cross-links from the main trunk of the median nerve. This explains why it is more vulnerable to injury and solitary paralysis, even with minor trauma, e.g. vein punctions or endoscopic elbow procedures, than other muscles with unremarkable motor activity (flexor digitorum profundus and pronator quadratus) that are also innervated by the AIN. If neuritis seems unlikely, they also favour an initial conservative approach. In our case, as more than two years had elapsed since the beginning of FPL denervation, we directly chose a tendon transfer to restore strong thumb flexion function as fast as possible.

Conclusions

AIN syndrome is uncommon and incomplete manifestations with isolated palsy of the flexor FPL, FDP 2 or pronator teres are even more seldom. Tendon ruptures have to be excluded by clinical tests preoperatively. Conservative approaches may lead to functional recovery in 70 percent if there is no clear evidence of compression or injury beyond neuropraxia. Solitary lesion of the FPL motor branch is a rare, but clinically important differential diagnosis in case of sudden thumb flexion loss. Due to its specific topographical location on the outer aspect over a long distance without interneural crosslinks from the main trunk of the median nerve, the FPL nerve fascicle is more prone to isolated traumatic lesion and palsy and clinicians should be aware of this pathology when facing with unclear paralysis of FPL function.

Notes

Competing interests

The authors declare that they have no competing interests.

References


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