

Management of Cubital Tunnel Syndrome in Split-Hand/Split-Foot Malformation: A Case Report and Review of Ulnar Nerve Dependence in Cleft Hand Prehension

Seth Stidham¹, Devin Soldati², Emily K Stidham³, Kevin Thomas⁴

Learning Point of the Article:

Ulnar neuropathy in individuals with Split-Hand/Split-Foot Malformation may have more acute deficits due to certain cleft types reliance on the ulnar nerve for cleft-based prehension.

Abstract

Introduction: Split-hand/split-foot malformation (SHFM) is a congenital limb defect characterized by central ray deficiency, resulting in deep median clefts of the hands and/or feet. Due to the nature of the malformation, the outer digits – those predominantly innervated by the ulnar nerve – are typically spared. In patients who rely on central cleft adduction and abduction as a substitute for thumb-index opposition, the ulnar nerve may assume a disproportionately critical role in hand function.

Case Report: We report a case of bilateral cubital tunnel syndrome (CubTS) in a 23-year-old patient with familial SHFM. We review the variable phenotypes of SHFM, the Manske and Halikis classification system, the clinical presentation and management of CubTS, and the functional anatomy of cleft closure.

Conclusion: We argue that the prioritization of neuropathy treatment in patients with congenital hand malformations should be advanced, given their overreliance on different nerve–distributed musculature for essential hand function.

Keywords: Split-hand/split-foot malformation, cubital tunnel syndrome, ulnar neuropathy, cleft-based prehension.

Introduction

Split-hand/split-foot malformation (SHFM) is a congenital limb defect that results in deep median clefts of the hands and/or feet, and effects approximately 1 in 18,000 newborns [1]. Phenotypic variation is highly prevalent, with milder forms of aplasia solely affecting distal central phalanges, to more severe malformations that result in clefts of the metacarpal space and carpals [2]. SHFM may be non-syndromic (isolated) or may appear as part of a broader syndrome.

Surgical correction varies with severity. The Manske and Halikis

classification, based on first web space preservation, is conventionally used to evaluate hand function. In Types I, II, and III, where an index ray is present, surgical goals include creation of a stable oppositional thumb, widening of the first web space using available index and thumb structures, and cleft closure [3]. However, some authors note that the cleft itself serves as a critical pinch mechanism – a “crab-like” motion relying on intrinsic hand muscles for abduction and adduction of the bordering rays, making the second web space functionally important [4]. Regardless of cleft type or age, patients with cleft hands

Author's Photo Gallery



Mr. Seth Stidham



Mr. Devin Soldati



Miss. Emily K Stidham



Dr. Kevin Thomas

Access this article online

Website:
www.jocr.co.in

DOI:
<https://doi.org/10.13107/jocr.2026.v16.i08.7870>

¹Saint Louis University School of Medicine, St. Louis, Missouri,

²University of Iowa Carver College of Medicine, Iowa City, Iowa,

³University of Wisconsin School of Medicine and Public Health, Madison, Wisconsin,

⁴Department of Family and Community Medicine, Saint Louis University School of Medicine, SLUCare Academic Pavilion, St. Louis, MO.

Address of Correspondence:

Mr. Seth Stidham,
Saint Louis University School of Medicine, St. Louis, Missouri.
E-mail: seth.stidham@health.slu.edu; stidhamd@gmail.com

Submitted: 12/06/2026; Review: 24/06/2026; Accepted: July 2026; Published: August 2026

DOI: <https://doi.org/10.13107/jocr.2026.v16.i08.7870>

© The Author(s). 2026 Open Access. This article is distributed under the terms of the Creative Commons Attribution 4.0 International License (<https://creativecommons.org/licenses/by-nc/4.0/>), which permits unrestricted use, distribution, and non-commercial reproduction in any medium, provided you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license, and indicate if changes were made. The Creative Commons Public Domain Dedication waiver (<http://creativecommons.org/publicdomain/zero/1.0/>) applies to the data made available in this article, unless otherwise stated.

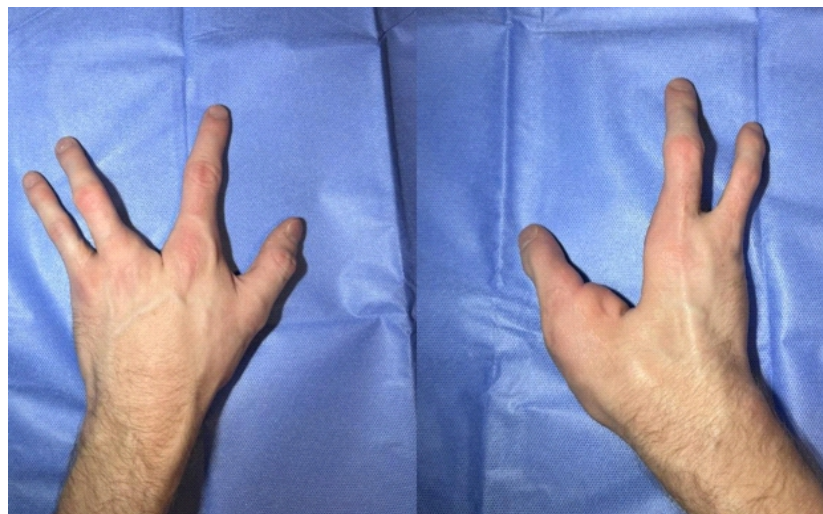


Figure 1: Extensor view of hands, demonstrating Type I/unmerged cleft (Left) and Type IV/merged cleft (Right) hands.

demonstrate impaired function and lower outcome scores compared with age-matched controls [5].

Cubital tunnel syndrome (CubTS) is one of the most common compression neuropathies, entrapping the ulnar nerve at the elbow within the medial elbow, affecting 2–6% of the population [6]. Clinical presentation of CubTS includes numbness and tingling in the ulnar fourth and fifth digits and corresponding parts of the dorsal and palmar hand. These symptoms can be worsened by activities in elbow flexion, including phone use, desk work, and sleeping [7]. Difficulty or weakness with thumb and finger abduction, pinching, grip strength, and fine motor control, such as writing with a pencil or playing an instrument, are motor deficits that may be appreciated in moderate CubTS [8]. If CubTS is left untreated, 50% of patients do not report improvement, and approximately one in five cases progressively worsens [9].

There is extremely limited literature discussing the management of compression mononeuropathies in individuals with congenital hand deformities and malformations. Specifically, there is no literature traversing how to differently manage acquired mono-neuropathies in individuals with cleft hands.

Case Report

A 23-year-old man with SHFM, having undergone corrective surgeries at ages 1, 7, and 15, for the creation of a merged cleft on his right hand, was referred to the orthopedic surgery department. He had a 4-year history of bilateral hand weakness,

paresthesia, and numbness in an ulnar nerve distribution, decreased fine manipulation, noticed in writing and typing due to fasciculations, as well as decreased sensation when using fine manipulations of tools, resulting in occasional deep tissue pressure injuries in the 4th and 5th digits. Throughout much of this time, he had not experienced any grossly decreased cleft-based prehension strength; however noted emerging prehension changes prompting re-evaluation. A prior nerve conduction study and evaluation by an orthopedic hand specialist at the time of symptom onset were grossly normal, and the patient was recommended to continue conservative management, as per guideline standards for someone with normal morphology, consisting of bracing and nerve glide exercises. This recommendation was not made by his previous hand surgeon, a congenital malformation specialist.

On physical examination, the hands demonstrated clear aplasia of the central rays consistent with ectrodactyly (Fig. 1). There was noted thenar and first web space atrophy on the left; merged first web space on the right complicated evaluation. Special testing was notable for positive Tinel's sign at the wrist and elbow, Phalen test, Wartenburg sign, and Froment sign. There was also mild bilateral ulnar clawing, more pronounced on the left than the right. electromyography / nerve conduction studies demonstrated bilateral demyelinating ulnar mononeuropathies, greater on the left, confirming bilateral CubTS, and review of previous hand X-rays demonstrated depth of cleft (Fig. 2).

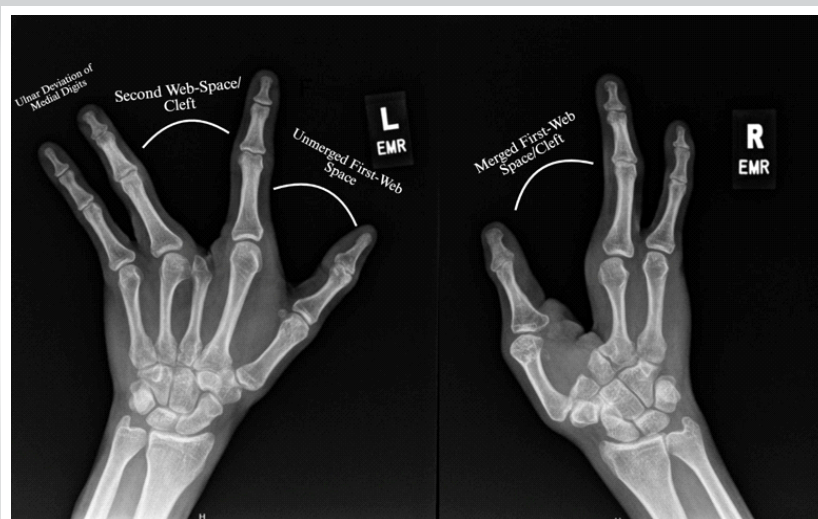


Figure 2: Bilateral merged hand X-rays demonstrating different cleft depth and difference in unmerged and merged phenotype.

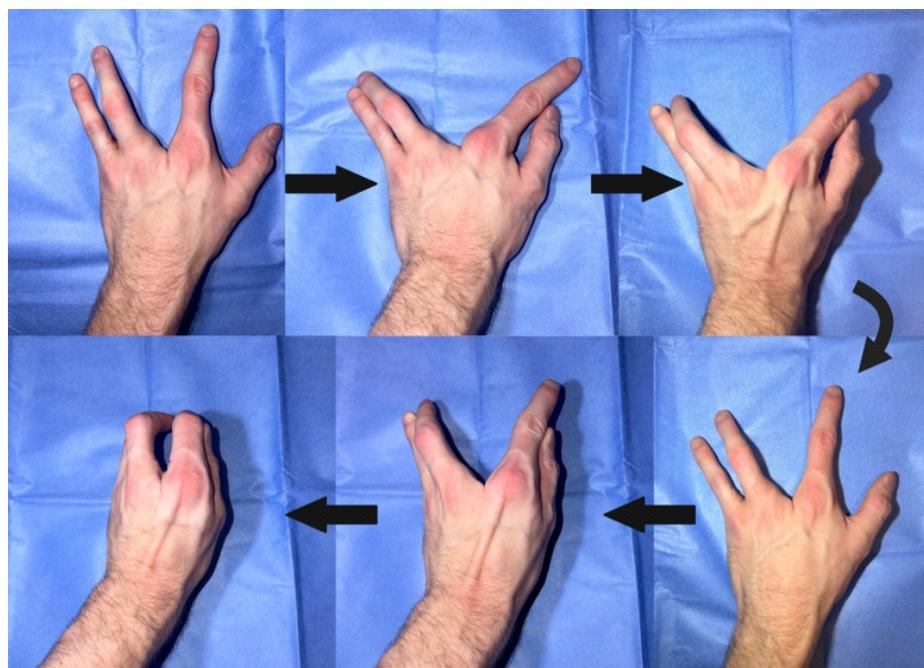


Figure 3: Abduction of the left unmerged central cleft into adduction functioning as lateral (key) prehension.

Manske and Halikis classification system stratifies cleft hands based on the integrity of the first web space. In Types I-II, the presence of an index ray allows for the persistence of a merged or unmerged cleft-based pinch. In our patient, whose left hand was a Type I, the mild cleft results in the formation of a functional cleft, with a preserved 1st web space, thus allowing for the use of the cleft as an alternative and preferred lateral prehension mechanism. This is furthered by the structural deviation of the retained 5th and 4th digits, which have ulnar deviation, allowing for large-degrees of cleft abduction and adduction (Fig. 3). In patients who use the central cleft as a substitute for the pinching mechanism normally performed by thumb opposition, the ulnar nerve assumes a disproportionately critical role. The

Left cubital tunnel release

Surgery was scheduled and performed 4 months after the pre-operative evaluation. A left ulnar nerve in situ decompression was performed under general anesthesia with a sterile arm tourniquet. Through a 5 cm longitudinal incision between the medial epicondyle and anteromedial olecranon, the ulnar nerve was identified. The fascial roof overlying the nerve was released 8 cm proximal to the elbow, and the Osborne ligament and flexor carpi ulnaris fascia were released 8 cm distally, constituting the site of compression. The preservation of traversing cutaneous nerves was ensured throughout. The ulnar nerve remained stable with elbow flexion and extension; transposition was not required. The wound was irrigated and closed in layers. The patient tolerated the procedure without complications.

At 8 months post-operative, the patient reports less frequent symptomology, including paresthesia and fasciculations; however, notes that these symptoms still occur. Ulnar clawing, thenar atrophy, and webspace atrophy are approximately the same as before the operation.

Discussion

Functional anatomy in SHFM

This case illustrates the variable phenotypes of SHFM and highlights the critical importance of the ulnar nerve in patients who rely on cleft-based hand function. The

“crab-like” cleft closure motion – bringing the bordering rays together across the cleft – depends primarily on the palmar interossei for adduction and the dorsal interossei for abduction. These structures are normally innervated by the deep branch of the ulnar nerve (Tables 1 and 2) [10].

In total, the ulnar nerve innervates all seven interossei, the medial two lumbricals, the three hypothenar muscles, the adductor pollicis, and partially the flexor pollicis brevis – constituting the majority of intrinsic hand musculature [10]. In a normal hand, the functional consequences of ulnar

Table 1: Muscles involved in cleft closure: Adduction toward the cleft

Muscle	Action	Nerve	Root level
1 st Palmar interosseous	Adducts index finger toward middle finger (toward cleft)	Ulnar nerve (deep branch)	C8–T1
2 nd Palmar interosseous	Adducts ring finger toward the middle finger (toward cleft)	Ulnar nerve (deep branch)	C8–T1

Table 2: Muscles involved in cleft opening: Abduction away from the cleft

Muscle	Action	Nerve	Root level
2 nd Dorsal interosseous	Index finger abduction (away from cleft)	Ulnar nerve (deep branch)	C8–T1
3 rd Dorsal interosseous	Ring finger abduction (away from cleft)	Ulnar nerve (deep branch)	C8–T1

neuropathy are distributed across a full complement of digits and can be partially compensated by median nerve-innervated muscles.

Functional consequences of ulnar neuropathy in SHFM

Studies of ulnar nerve injury in anatomically normal hands demonstrate that grasping strength decreases by approximately 59–62%, with normal index-thumb-based key pinch by 51–61%, and pinch-to-zoom strength by 75–76% compared with the unaffected hand [11]. Grip strength receives greater contributions from the ulnar nerve than the median nerve, and ulnar nerve palsy results in reductions across all pinch and grip strength measures. The primary intrinsic muscles responsible for lateral and tip pinch – the adductor pollicis and first dorsal interosseous – are both ulnar-innervated [12].

In the context of SHFM, these deficits are amplified. Loss of interosseous function due to CubTS would effectively eliminate the cleft-based pinch mechanism, rendering the hand functionally inert for fine manipulation. This stands in contrast to the anatomically normal hand, where ulnar neuropathy impairs but does not eliminate prehension, as thumb opposition (primarily median-innervated) remains intact.

Furthermore, peripheral nerve anatomy in cleft hand syndrome has been shown to reflect the presence and absence of corresponding muscle and skin innervation areas. In a case of severe bilateral clefts, high-resolution ultrasound demonstrated severe bilateral reduction in median nerve size, with relative sparing of the ulnar nerve, consistent with the preservation of ulnar-innervated structures [13]. This finding underscores that in SHFM, the ulnar nerve may be the dominant – and in some cases, the sole – functional motor nerve to the remaining hand musculature.

Implications for neuropathy management

The disproportionate reliance on ulnar nerve-innervated musculature in SHFM has direct implications for the

prioritization of neuropathy treatment. In the general population, CubTS is managed along a stepwise algorithm: conservative measures for mild disease, with surgical intervention reserved for severe or refractory cases [14]. In patients with SHFM—or others reliant on cleft-based pinch, even mild ulnar neuropathy may produce functionally significant deficits. Conservative management should be initiated promptly and monitored closely, and surgical evaluation should be considered earlier in the disease course than would be typical for the general population.

Conclusion

This case highlights the intersection of congenital hand malformation and acquired compression neuropathy. This is the only reported case discussing the management of CuTS in SHFM. In patients with SHFM who do not have a merged cleft-and-first webspace (Types I-II), the cleft may still act, and may even be used more favorably, than the first webspace. In these individuals, the ulnar nerve is the primary motor supply to the muscles that enable cleft closure—the dorsal and palmar interossei. CubTS in this population threatens to eliminate the primary grasping mechanism of the hand. Clinicians should recognize that the functional impact of ulnar neuropathy may be amplified in patients with congenital hand anomalies and should prioritize early diagnosis and treatment accordingly. Further study is warranted to characterize the prevalence of compression neuropathies in patients with congenital limb differences and to develop tailored management algorithms for this population.

Clinical Message

Those with Split-hand/split-foot malformation should be monitored closely if compression neuropathies are of concern due to the possibility of more devastating deficits if they progress.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given the consent for his/ her images and other clinical information to be reported in the journal. The patient understands that his/ her names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Conflict of interest: Nil **Source of support:** None

References

1. Sowińska-Seidler A, Socha M, Jamsheer A. Split-hand/foot malformation - molecular cause and implications in genetic counseling. *J Appl Genet* 2014;55:105-15.
2. Gurrieri F, Everman DB. Clinical, genetic, and molecular aspects of split-hand/foot malformation: An update. *Am J Med Genet Part A* 2013;161A:2860-72.
3. Manske PR, Halikis MN. Surgical classification of central deficiency according to the thumb web. *J Hand Surg Am*



1995;20:687-97.

4. Aleem AW, Wall LB, Manske MC, Calhoun V, Goldfarb CA. The transverse bone in cleft hand: A case cohort analysis of outcome after surgical reconstruction. *J Hand Surg Am* 2014;39:226-36.
5. Rosa MF, Do Monte TM, Raposo-Amaral CE, Raposo-Amaral CA, De Abreu MF. Functionality assessment of patients with cleft hands. *J Craniofac Surg* 2022;33:104-7.
6. An TW, Evanoff BA, Boyer MI, Osei DA. The prevalence of cubital tunnel syndrome: A cross-sectional study in a U.S. metropolitan cohort. *J Bone Joint Surg Am* 2017;99:408-16.
7. Burahee AS, Sanders AD, Shirley C, Power DM. Cubital tunnel syndrome. *EFORT Open Rev* 2021;6:743-50.
8. Dawson DM. Entrapment neuropathies of the upper extremities. *N Engl J Med* 1993;329:2013-8.
9. Padua L, Aprile I, Caliandro P, Foschini M, Mazza S, Tonali P. Natural history of ulnar entrapment at elbow. *Clin Neurophysiol* 2002;113:1980-4.
10. Stewart JD. Peripheral nerve fascicles: Anatomy and clinical relevance. *Muscle Nerve* 2003;28:525-41.
11. Bertelli JA. Prior to repair functional deficits in above- and below-elbow ulnar nerve injury. *J Hand Surg Am* 2020;45:552.e1-10.
12. Misirlioglu TO, Palamar D, Taskiran OO, Terlemez R, Akgun K. Relationship between ultrasonographic hand muscle thickness measurements and muscle strength following median or ulnar nerve reconstruction. *Muscle Nerve* 2021;63:351-6.
13. Falco P, Hovius S, Van Alfen N. Peripheral nerve innervation in bilateral cleft hand syndrome elucidated by ultrasound. *Front Neurol* 2022;13:857363.
14. Wade RG, Griffiths TT, Flather R, Burr NE, Teo M, Bourke G. Safety and outcomes of different surgical techniques for cubital tunnel decompression: A systematic review and network meta-analysis. *JAMA Netw Open* 2020;3:e2024352.

Conflict of Interest: Nil
Source of Support: Nil

Consent: The authors confirm that informed consent was obtained from the patient for publication of this article

How to Cite this Article

Stidham S, Soldati D, Stidham EK, Thomas K. Management of Cubital Tunnel Syndrome in Split-Hand/Split-Foot Malformation: A Case Report and Review of Ulnar Nerve Dependence in Cleft Hand Prehension. *Journal of Orthopaedic Case Reports* 2026 August;16(08):279-283.

