General

Anterior Interosseous Nerve Syndrome

Nathan Li[1] [a], Katherine Russo[2], Lauren Rando[2], Laura Gulotta-Parrish[2], William Sherman[3], Alan D. Kaye[4]

[1] Medical College of Wisconsin, [2] Louisiana State University Health Sciences Center - Shreveport, [3] Orthopaedic Surgery, Tulane University, [4] Anesthesiology, Louisiana State University Shreveport

Keywords: Anterior interosseous nerve syndrome, nerve entrapment, neuropathy, pain

https://doi.org/10.52965/001c.38678

Orthopedic Reviews

Vol. 14, Issue 4, 2022

Anterior interosseous nerve syndrome (AINS) is a rare form of peripheral neuropathy which involves disruption of the anterior interosseous nerve. The pathophysiology of AINS remains unclear. AINS typically initially presents with forearm pain and may gradually progress to palsy of the deep muscles of the anterior forearm. Diagnosis of AINS requires thorough patient history and physical exam. EMG is the preferred diagnostic study and classically reveals abnormal activity and prolonged latency periods within the evoked action potentials of the FPL and PQ. Due to the self-limiting nature of AINS, there is general agreement that conservative and symptomatic management should be explored for up to 6 months as first line therapy, which usually includes analgesics and nonsteroidal anti-inflammatory drugs, contracture prevention, hand therapy, and hand splinting. Surgical options such as internal neurolysis and minimally invasive endoscopic decompression may be explored if functional recovery from conservative management is limited.

INTRODUCTION

Peripheral nerves of the body are susceptible to disruption as they traverse the extremities to their final destination. The etiology and location of disruption are often predictable and produce a clinically distinct set of signs and symptoms, typically consisting of a combination of weakness, sensory disturbances, and electrodiagnostic abnormalities. Examples of common compressive lesions include carpal tunnel syndrome and cubital tunnel syndrome.[1] A rarer neuropathy involves disruption of the anterior interosseous nerve (AIN), resulting in the anterior interosseous nerve syndrome (AINS), and typically initially presents with forearm pain which gradually transitions to a palsy of the deep muscles of the anterior forearm, including the flexor digitorum profundus (FDP), flexor pollicis longus (FPL), and pronator quadratus (PQ), resulting in lack of flexion of the distal interphalangeal joints of the first and second digits, interphalangeal flexion of the thumb, and forearm pronation.[2–4] The elusive pathophysiology of AINS has been widely debated and remains unclear, although the most common theories involve either an idiopathic immune-mediated neuritis or an intrinsic compressive etiology within the forearm 1. Due to the lack of consensus regarding the condition's true etiology, an optimal treatment protocol for AINS has not been established, and further research is imperative for the improving the existing care of affected patients.[5–8] Of note, direct traumatic injury to the AIN, either due to upper extremity trauma or extrinsic mass (i.e. tumor) are rare and not considered true AINS due to differing pathophysiology.[9]

EPIDEMIOLOGY

Compared to commonly encountered peripheral nerve entrapment syndromes such as carpal tunnel syndrome and cubital tunnel syndrome, AINS is quite rare, comprising <1% of all upper limb nerve syndromes, affecting men and women, as well as dominant and non-dominent upper extremities equally.[4,9–12] Patients affected typically present in their 40s; however, the age of documented individuals have ranged widely – a study by Worner documented 69 AINS patients with ages ranging from 9 to 72.[11–13] Patients may experience bilateral palsy as well as recurrent episodes

a **Corresponding author:**
Nathan Li
Medical College of Wisconsin – Milwaukee
8701 W Watertown Plank Rd
Wauwatosa, WI, 53226
Phone: (920) 277-8327
linathan006@gmail.com

of AINS.[14] Common documented triggers often include physiologically stressful events such as trauma, surgery, pregnancy, and venipuncture.[15–19]

Of note, populations at risk for upper extremity fractures such as a supracondylar fracture of the humerus in children have led to symptoms similar to AINS, likely related to a combination of proximal median nerve injury, traction of the AIN, and ischemia.[20,21] Consistent external compression of the forearm has also been documented to lead to symptoms similar to AINS, including guitar players, shoulder immobilization slings, and crutches.[22] Additional iatrogenic causes of AINS include Rober-Jones-type bandage following lateral clavicle fracture, Kenny-Howard splint following acromioclavicular joint dislocation, Velpeau dressing after a modified Bristow procedure, and compression of the antecubital fossa with cotton padding prior to cast or splint placement.[23] As discussed above, these cases are not considered true AINS due to originating from an external compressive etiology, and symptoms have consistently been shown to resolve once the external compression is eliminated.[24–28]

CLINICAL PRESENTATION/DIAGNOSIS

AINS often occurs spontaneously and is often found to follow an inciting event such as trauma, pregnancy, or stress.[14,29] Diabetes, human immunodeficiency virus, and nutritional deficiencies have been associated with the development of AINS.[30] Pain is usually experienced acutely and is localized to the proximal volar forearm and worsened by repetitive action.[30] After resolution of upper extremity pain, patients begin to experience motor weakness of the FPL, FDP, and PQ.[23,31] AINS may be categorized as a complete palsy, with involvement of all three muscle groups, or partial, with involvement of one or two muscle groups.[13,14] Typically, weakness is particularly noticeable on flexion of the interphalangeal joint of the thumb or the distal interphalangeal joints of the index finger.[32] Weakness of the third finger may also be observed due to variable contribution by the AIN.[29] In a subset of individuals, the proximal interphalangeal joints may also be affected due to anomalous innervation of the flexor digitorum superficialis by the AIN.[29] Additionally, the Martin-Gruber communicating branch, a unique connection between the median or AIN and ulnar nerves, may contribute to intrinsic muscle weakness in addition to the characteristic triad of AINS.[9,29] Changes in handwriting may also be observed if the dominant hand is involved.[29] Other general manifestations of AIN syndrome may include neuropathic pain, contracture, loss of dexterity, muscle weakness or paralysis.[21,32,33] Notably, clinical symptoms are usually limited to motor palsies, and sensory deficits such as paresthesias are not commonly reported in AINS, as the AIN does not provide cutaneous sensory innervation.[1,30,31,33]

Thorough physical examination is essential in diagnosing AINS. Patients classically reveal a positive Kiloh-Nevin sign, with an inability to bring the thumb and index finger together in the shape of an "O".[1,34] Additionally, patients with AINS display increased pulp contact between the thumb and index finger with hyperflexion of the proximal interphalangeal joint of the index finger.[16] Further observation will reveal an inability to hold a piece of paper between the thumb and first finger with possible grip compensation via extension of interphalangeal joint of the thumb and distal interphalangeal joint of the first finger.[34] This compensation is also known as the key pinch maneuver and is retained in patients with AINS due to thumb adduction, which is innervated by the ulnar nerve.[1] Additional testing includes comparison of range of motion, pinch (tripod and/or key) and grip strength between affected and unaffected upper extremity.[21,32] Strength of the FPL and FDP should be assessed individually, as weakness of these muscles may be the presenting symptom in mild or incomplete cases of AINS.[33,34] Strength assessment of the PQ should be performed with the elbow flexed in order to minimize contribution from the pronator teres.[14] However, the efficacy of this maneuver is debated, and detectable weakness in pronation is often indicative of more proximal injury.[9,29] Although palpation in the distribution of the AIN may produce pain, sensory symptoms should be further explored as this may suggest an alternative cause of neuropathy such as pronator syndrome or carpal tunnel syndrome.[30,31,35] Of note, unlike other median nerve palsies, Tinel's test and Phalen's maneuver should yield negative results in AINS.[30] Additionally, calculation of disability of arm, shoulder, and hand (DASH) score is often helpful in determining the degree of upper extremity disability.[32]

A variety of imaging techniques may be used to diagnose AINS. Electromyography (EMG) is the preferred test in confirming the diagnosis of AINS and classically reveals abnormal activity and prolonged latency periods within the evoked action potentials of the FPL and PQ.[36] EMG with a coaxial needle has been shown to possess superior sensitivity compared to surface recording and is favored when diagnosing AINS.[37] Serial EMG may be useful in monitoring improvement of function as well as further therapeutic planning.[34,36] Additionally, EMG has been shown to successfully exclude differential causes of neuropathy such as pronator palsy or brachial plexopathy, as well as tendon injuries.[29,31–33,38] Tendon injuries may be ruled out via direct electrical stimulation of the FDP or FPL.[30] Magnetic resonance imaging (MRI) is not commonly used in diagnosing AINS but has been described in the literature to useful in early diagnosis, with T2 weighted MRI often revealing increased signal intensity in the distribution of the AIN.[16,34] Ultrasound may be used to view the AIN despite its small size; however, the clinical utility of ultrasound in AIN syndrome is limited.[22]

PATHOPHYSIOLOGY/RISK FACTORS

The exact pathophysiology of AINS has been debated over the past five decades. Originally, compression and entrapment of the AIN was suspected to be the etiology of AINS.[4,16] However, more recent evidence has come to suggest that AINS may be a variation of neuralgic amyotrophy (NA), which is an idiopathic immune neuropathy with denervation measured on electrodiagnostic studies in upper

Table 1.

Physical Exam	Response
"OK"/Kiloh-Nevin sign	Unable to perform
Pinch paper between thumb and index finger. Then, resist removal of paper.	Unable to perform OR May compensate by extending interphalangeal joint of thumb and distal interphalangeal joint of index finger (key pinch maneuver).
Cutaneous sensory testing	Normal
Muscle stretch reflex	Normal
Tinel's test	Negative
Phalen's maneuver	Negative

extremity motor nerves.[8,9,13,39] Both NA and AINS present similarly; NA presents with an acute prodrome of upper extremity pain (i.e. shoulder, arm, elbow) followed by clinically impressive motor palsy which is also seen in a mean of 82% of patients diagnosed with AINS.[40] Electrodiagnostic studies have shown that NA and AINS are both peripheral motor nerve axonopathies with significant impairment in muscle recruitment.[37,41–43] CD8+ T-lymphocytes, fibrosis, and edema have consistently been found on histologic biopsies in AINS patients, which further suggests that AINS is due to an immune-related etiology.[43] Additionally, diagnostic studies have shown diffuse muscle degeneration and nerve enlargement of nerve branches and motor units not directly supplied by the AIN, further suggesting inflammatory cause rather than focal denervation as would be expected from compression.[13,44]

Further consideration of relevant forearm anatomy suggests that intrinsic compression of the AIN does not adequately explain the clinical symptoms observed in AINS. The AIN classically branches from the median nerve 5-8 cm distal to the lateral epicondyle and travels beneath the pronator teres.[5,6] It then continues between the FDP and FPL, giving off specific motor branches to these muscles, and proceeds along the anterior interosseous membrane adjacent to the anterior interosseous artery before terminating at the radiocarpal, midcarpal, and carpometacarpal joints.[5] Compression proximal to the branching of the AIN is unlikely to cause proper AINS as compression of the median nerve would also produce clinical sensory deficits. However, compression distal to the AIN branch point would also be an unlikely etiology for AINS, as patients often have selective denervation of one or two of the FDP, FPL, and PQ, as opposed to weakness observed in all three muscles.[40] Therefore, compressive etiologies such as the ligament of Struthers and the Gantzer muscle are unlikely to fully explain the cause of AINS.[2,12,38] Additionally, true compressive neuropathies such as carpal tunnel syndrome have symptoms which present progressively over a period of weeks to months. In comparison, AINS classically presents spontaneously with gradual resolution of symptoms, suggesting that neuritis is more likely the true cause of AINS than intrinsic compression.[45]

TREATMENT OPTIONS

CONSERVATIVE MANAGEMENT

Due to the self-limiting nature of AINS, there is general agreement that conservative and symptomatic management should be explored for up to 6 months as first line therapy.[9,41] Additionally, conservative management has been observed to have no significant difference in recovery time when compared to immediate surgical management.[17,32,41] A study performed by Nakano et al followed 2 patients with bilateral AINS who were conservatively treated on the first upper extremity and subsequently surgically treated on the opposite upper extremity. Nakano reported no significant difference in recovery of motor function between the conservative and surgical treatments, suggesting that conservative management ought to be pursued as a first line option in patients with AINS.[46]

Conservative management generally includes analgesics and nonsteroidal anti-inflammatory drugs, contracture prevention, hand therapy, hand splinting, electrical stimulation therapy, and supplemental vitamin B^{12} when deemed appropriate.[41,47–49] Some studies have cited success in treating AINS utilizing other forms of conservative therapy. The strain-counter strain (SCS) technique is a therapy that involves locating tender trigger points and focal manipulation and has been reported to be effective in conjunction with electrical stimulation therapy, cryo massage, and neural mobilization.[50] Goyal et al reported a 37 year old female with a one month history of AINS who experienced marked improvement from 5 lbs of pincer grip strength to 8 lbs with two weeks of SCS treatment before being lost to follow up. Furthermore, long-term efficacy of this therapy has not been extensively recorded and further studies must to be done to determine its role in the conservative management of AINS.[51]

Currently, the appropriate length of time in which conservative management ought to be pursued prior to initiating surgical interventions, as well as its overall efficacy in different populations is not well understood. Seki et al examined 21 patients with AINS between the ages of 17 and 65 in a retrospective study to determine the length of time required to recover normal function without any surgical intervention. Conservative treatment consisted of vitamin B^{12} and electrical stimulation therapy and electromyography was used to monitor muscular activity and track progress. Results showed improved recovery rates in patients under 40 years old, and delayed and lack of recovery in older patients, suggesting that conservative treatment might be more suitable for younger patients.[48] However, recovery with conservative management is often incomplete and further surgical intervention and treatment is recommended in patients with insufficient or complete lack of clinical improvement.[32,47,49,52,53]

SURGICAL MANAGEMENT

Functional recovery may be insufficient when performing conservative therapy for AINS and NA. Van Alfen et al studied 246 patients with NA with conservative treatment and found that over 60% experienced residual weakness greater than or equal to three years after symptom onset 9. Smaller studies have also examined patients with diagnosed AINS and found that residual symptoms are common.[42,54,55] Related to this, more aggressive treatment options such as internal neurolysis have been explored.[8,9,43] In a study done by Kodoma et al, internal neurolysis was performed on AINS patients who did not experience any clinical improvement in symptoms after 6 months of conservative therapy. Kodoma found that 77% (10 of 13) of patients who underwent surgery at that time experienced significant improvement in strength (greater than or equal to M4 in the manual muscle test (MMT)), while only 36% (5 of 14) of patients who continued with conservative management experienced similar levels of recovery.[47] Krishnan et al reported similar success with internal neurolysis in 6 patients with chronic AINS and a mean of 14 months of conservative management; at 13.6 months follow up, 5 of 6 operative patients reported greater than M4 strength while only 1 of 6 nonoperative patients was able to recover a similar degree of strength.[56]

Minimal incision surgeries such as endoscopic decompression have also been explored as a potential treatment option for AINS. These procedures are attractive due to their minimal incision, which promotes decreased scar tissue formation, adhesions, blood loss, as well as improved recovery time and cosmetics.[57] Ochi et al compared the efficacies of combined internal and external neurolysis in the context of minimal incision surgery and wide incision surgery in patients with AINS who presented a mean of 5 months after symptom onset and reported that 82% of patients managed surgically recovered greater than or equal to M4 strength. Interestingly, they found no significant difference between minimal and wide incision surgery outcomes.[58] Additionally, Sneag et al found that 99% of nerve enlargement in AINS occurs slightly proximal to the elbow and is accessable with minimal incision surgery, further suggesting that minimal incision procedures may also be efficacious in treatment of AINS.[53,54] Limitations to minimal incision surgery includes patient anatomy; patients with increased adipose or muscle tissue require increased skill to successfully perform the procedure due to limited scope visibility.[59]

Further studies must be performed to determine the appropriate length of time spent in conservative therapy prior to considering surgical interventions. A study by Nagano et al compared 15 patients with AINS with 3 months of conservative management who underwent internal neurolysis with 11 patients who continued nonoperative management and found that all 15 (100%) of the operative patients recovered greater than M3 strength, although 9 out of 11 patients with conservative management were able to recover similar levels of strength with conservative management.[40] This study suggests that surgical consideration at 3 months after AINS onset may be premature. Studies done by Van Alfen and Yamamoto have both suggested that consideration and further workup for surgical interventions (such as high resolution imaging evaluation) may begin after 6 months of initial conservative management.[40,53] However, further studies must be performed and utilized in conjunction with appropriate clinical judgement to determine the optimal time to pursue surgical interventions in patients with AINS.

A subset of patients may not experience recovery of motor function following both conservative and surgical interventions. In these cases, there have been reports of performing tendon transfers in effort to restore motor function, although the overall efficacy of these procedures is unimpressive. Specifically, the brachioradialis can be used to restore function to the interphalangeal joint of the thumb, and the flexor digitorum profundus of the third or fourth digit can be transferred to the index finger, resulting in flexion of the distal phalanx.[9,32] Schantz and Nielsen have recommended pursuing conservative therapy for at least one year prior to considering tendon transfer due to possible spontaneous recovery.[55]

CONCLUSION

AINS is a variation of NA and classically presents with an acute prodrome of upper extremity pain followed by weakness in flexion of the distal interphalangeal joints of the thumb, index finger, and middle finger, leading to a positive Kiloh-Nevin sign and increased pulp contact between the thumb and index finger. Consistent compression of the forearm (external or internal i.e. mass) has also been shown to produce clinical symptoms similar to AINS, although this is not considered true AINS and resolves shortly after resolution of external compression.

Diagnosis of AINS requires thorough patient history and physical exam. EMG is the preferred diagnostic study and classically reveals abnormal activity and prolonged latency periods within the evoked action potentials of the FPL and PQ. Since AINS is commonly self-limiting, treatment usually begins with conservative and symptomatic management. Surgical interventions may be considered after patients have failed conservative therapy and include options such as internal neurolysis and endoscopic decompression. Further studies must be performed in order to determine the optimal time course of conservative therapy prior to consideration of more invasive procedures.

DISCLAIMERS

The authors did not receive any funding or financial support or potential sources of conflict of interest.

The study has been performed in accordance with the ethical standards in the 1964 Declaration of Helsinki.

REFERENCES

1. Strohl AB, Zelouf DS. Ulnar Tunnel Syndrome, Radial Tunnel Syndrome, Anterior Interosseous Nerve Syndrome, and Pronator Syndrome. *J Am Acad Orthop Surg*. 2017;25(1):e1-e10. doi:10.5435/jaaos-d-16-00010

2. Rodner CM, Tinsley BA, O'Malley MP. Pronator syndrome and anterior interosseous nerve syndrome. *J Am Acad Orthop Surg*. 2013;21(5):268-275. doi:10.5435/jaaos-21-05-268

3. Wertsch J, Melvin J. Median nerve anatomy and entrapment syndromes: a review. *Arch Phys Med Rehabil*. 1982;63(12):623-627.

4. Creighton Jr J, Strickland J, Idler R. Anterior interosseous nerve palsy. *Indiana Med*. 1991;84(3):192-194.

5. COLLINS DN, WEBER ER. Anterior Interosseous Nerve Syndrome. *South Med J*. 1983;76(12):1533-1537. doi:10.1097/00007611-198312000-00019

6. Nakano KK. Nerve entrapment syndromes. *Curr Opin Rheumatol*. 1997;9(2):165-173. doi:10.1097/00002281-199703000-00015

7. Park IJ, Roh YT, Jeong C, Kim HM. Spontaneous anterior interosseous nerve syndrome: Clinical analysis of eleven surgical cases. *J Plast Surg Hand Surg*. 2013;47(6):519-523. doi:10.3109/2000656x.2013.791624

8. Akane M, Iwatsuki K, Tatebe M, et al. Anterior interosseous nerve and posterior interosseous nerve involvement in neuralgic amyotrophy. *Clin Neurol Neurosurg*. 2016;151:108-112. doi:10.1016/j.clineuro.2016.11.001

9. Nagano A. SPONTANEOUS ANTERIOR INTEROSSEOUS NERVE PALSY. *J Bone Jt Surg [Br]*. 2003;85-B(3):313-321. doi:10.1302/0301-620x.85b3.14147

10. Winterton RIS, Farnell R. Peripheral nerve entrapment syndromes of the upper limb. *Surg*. 2013;31(4):172-176. doi:10.1016/j.mpsur.2013.01.015

11. Worner CO. The anterior interosseous nerve syndrome. *Int Orthop*. 1989;13(3):193-197. doi:10.1007/bf00268047

12. Grünert J, Beutel F, Grünert JG, ProfDrARüter R, ProfDrHReilmann A, ProfDrDNast-Kolb B. *Das N. Interosseus Anterior-Syndrom*.

13. Pham M, Baumer P, Meinck HM, et al. Anterior interosseous nerve syndrome: Fascicular motor lesions of median nerve trunk. *Neurology*. 2014;82(7):598-606. doi:10.1212/wnl.0000000000000128

14. Worner CO. The anterior interosseous nerve syndrome. *International Orthopaedics*. 1989;13(3):193-197. doi:10.1007/bf00268047

15. Pope D, Wottowa C. Mixed neuropathy presenting clinically as an anterior interosseous nerve palsy following shoulder arthroscopy: a report of four cases. *J Shoulder Elb Surg*. 2016;25(10):1699-1703. doi:10.1016/j.jse.2016.04.037

16. Jain M, Nayak C, Samal BP, Tirpude A. Kiloh-Nevin syndrome associated with humeral shaft fracture. *BMJ Case Reports*. Published online September 28, 2018. doi:10.1136/bcr-2018-226506

17. SOOD MK, BURKE FD. Anterior Interosseous Nerve Palsy: A review of 16 cases. *Journal of Hand Surgery*. 1997;22(1):64-68. doi:10.1016/s0266-7681(97)80020-4

18. Wong L, Dellon AL. Brachial neuritis presenting as anterior interosseous nerve compression—Implications for diagnosis and treatment: A case report. *J Hand Surg Am*. 1997;22(3):536-539. doi:10.1016/s0363-5023(97)80025-1

19. Van Alfen N, Van Engelen BGM. The clinical spectrum of neuralgic amyotrophy in 246 cases. *Brain*. 2006;129(2):438-450. doi:10.1093/brain/awh722

20. Joist A, Joosten U, Wetterkamp D, Neuber M, Probst A, Rieger H. Anterior interosseous nerve compression after supracondylar fracture of the humerus: a metaanalysis. *J Neurosurg*. 1999;90(6):1053-1056. doi:10.3171/jns.1999.90.6.1053

21. Gainor BJ, Olson S. Combined Entrapment of the Median and Anterior Interosseous Nerves in a Pediatric Both-Bone Forearm Fracture. *J Orthop Trauma*. 1990;4(2):197-199. doi:10.1097/00005131-199004020-00018

22. Helfenstein Júnior M. Uncommon compressive neuropathies of upper limbs. *Best Pract Res Clin Rheumatol*. 2020;34(3):101516. doi:10.1016/j.berh.2020.101516

23. Casey PJ, Moed BR. Fractures of the forearm complicated by palsy of the anterior interosseous nerve caused by a constrictive dressing. A report of four cases. *J Bone Jt Surg - Ser A*. 1997;79(1):122-124. doi:10.2106/00004623-199701000-00014

24. Rieck B. Inkomplettes Nervus interosseus anterior-Syndrom bei einem Gitarristen. *Handchir Mikrochir Plast Chir*. 2005;37(6):418-422. doi:10.1055/s-2005-837702

25. Murphy EP, Fenelon C, Alexander M, Quinlan J. Anterior interosseous nerve palsy as a result of prolonged shoulder immobilisation. *BMJ Case Rep*. 2019;12(5):e229010. doi:10.1136/bcr-2018-229010

26. Macneal P, Crome CR, McNally S. Persistent anterior interosseous nerve palsy following forearm crutch use. *BMJ Case Rep*. Published online January 11, 2017:bcr2016218240. doi:10.1136/bcr-2016-218240

27. Vailas JC. Compression of the anterior interosseous nerve after use of a sling for dislocation of the acromioclavicular joint. A report of two cases. *J Bone Jt Surg Am*. 1991;73(6):948-949. doi:10.2106/00004623-199173060-00022

28. ALBANESE S, BUTERBAUGH G, PALMER AK, LUBICKY JP, YUAN HA. Incomplete Anterior Interosseous Nerve Palsy Following Spinal Surgery. *Spine*. 1986;11(10):1037-1038. doi:10.1097/00007632-198612000-00015

29. Chin DHCL, Meals RA. Anterior interosseous nerve syndrome. *J Am Soc Surg Hand*. 2001;1(4):249-257. doi:10.1053/jssh.2001.28806

30. Lee MJ, Lastayo PC. Pronator Syndrome and Other Nerve Compressions That Mimic Carpal Tunnel Syndrome. *J Orthop Sports Phys Ther*. Published online October 2004. doi:10.2519/jospt.2004.1359

31. Lubahn J, Cermak MB. Uncommon Nerve Compression Syndromes of the Upper Extremity. *J Am Acad Orthop Surg*. 1998;6(1):378-386. doi:10.1001/archsurg.1965.01320070070015

32. Ulrich D, Piatkowski A, Pallua N. Anterior interosseous nerve syndrome: retrospective analysis of 14 patients. *Arch Orthop Trauma Surg*. 2011;131(11):1561-1565. doi:10.1007/s00402-011-1322-5

33. Wertsch JJ. AAEM Case Report #25: Anterior Interosseous Nerve Syndrome. *Muscle Nerve*. 1992;15(9):977-983. doi:10.1002/mus.880150902

34. Dang AC, Rodner CM. Unusual Compression Neuropathies of the Forearm, Part II: Median Nerve. *J Hand Surg Am*. 2009;34(10):1915-1920. doi:10.1016/j.jhsa.2009.10.017

35. Genova A, Dix O, Saefan A, Thakur M, Hassan A. Carpal Tunnel Syndrome: A Review of Literature. *Cureus*. Published online March 19, 2020. doi:10.7759/cureus.7333

36. Nakano K, Lundergan C, Okihiro M. Anterior Interosseous Nerve Syndromes. *Arch Neurol*. 1977;34(9):477-480. doi:10.2746/095777307x240829

37. Seror P. Selective anterior interosseous nerve conduction study: Coaxial needle versus surface electrode recordings. *Neurophysiol Clin*. 2017;47(4):315-321. doi:10.1016/j.neucli.2017.06.001

38. Pai MM, Nayak SR, Krishnamurthy A, et al. The Accessory Heads of Flexor Pollicis Longus and Flexor Digitorum Profundus: Incidence and Morphology. *Clin Anat*. 2008;21(3):252-258. doi:10.1002/ca.20612

39. Murlimanju B, Rao YL, Pai M, Tonse M, Prabhu L, Krishnaprasad P r. Exploring the morphology of anterior interosseous nerve and relating it, to its clinical conditions. *Turk Neurosurg*. 2021;31(1):107-111. doi:10.5137/1019-5149.jtn.29917-20.2

40. Krishnan KR, Sneag DB, Feinberg JH, Wolfe SW. Anterior Interosseous Nerve Syndrome Reconsidered: A Critical Analysis Review. *JBJS Rev*. 2020;8(9):e20.00011. doi:10.2106/jbjs.rvw.20.00011

41. Miller-Breslow A, Terrono A, Millender LH. Nonoperative treatment of anterior interosseous nerve paralysis. *J Hand Surg Am*. 1990;15(3):493-496. doi:10.1016/0363-5023(90)90069-4

42. Feinberg JH, Nguyen ET, Boachie-Adjei K, et al. The electrodiagnostic natural history of parsonage–turner syndrome. *Muscle Nerve*. 2017;56(4):737-743. doi:10.1002/mus.25558

43. Pan Y, Wang S, Zheng D, et al. Hourglass-like constrictions of peripheral nerve in the upper extremity: A clinical review and pathological study. *Neurosurgery*. 2014;75(1):10-22. doi:10.1227/neu.0000000000000350

44. Maldonado AA, Amrami KK, Mauermann ML, Spinner RJ. Reinterpretation of Electrodiagnostic Studies and Magnetic Resonance Imaging Scans in Patients with Nontraumatic "Isolated" Anterior Interosseous Nerve Palsy. *Plast Reconstr Surg*. 2016;138(5):1033-1039. doi:10.1097/prs.0000000000002689

45. Na K tae, Jang D hyun, Lee Y min, Park I jung, Lee H woo, Lee S uk. Anterior Interosseous Nerve Syndrome: Is it a Compressive Neuropathy? *Indian J Orthop*. 2020;54(S1):193-198. doi:10.1007/s43465-020-00099-2

46. Nakano KK, Lundergan C, Okihiro MM, Clinic S, Bent Brigham Hospital P. *Anterior Interosseous Nerve Syndromes Diagnostic Methods and Alternative Treatments From the Department of Neurology (Drs*.

47. Kodama N, Ando K, Takemura Y, Imai S. Treatment of spontaneous anterior interosseous nerve palsy. *J Neurosurg*. 2020;132(4):1243-1248. doi:10.3171/2018.11.jns181609

48. Seki M, Nakamura H, Kono H, Director Kono DH, Director C. Neurolysis is not required for young patients with a spontaneous palsy of the anterior interosseous nerve RETROSPECTIVE ANALYSIS OF CASES MANAGED NON-OPERATIVELY. doi:10.1302/0301

49. Aljawder A, Faqi MK, Mohamed A, Alkhalifa F. Anterior interosseous nerve syndrome diagnosis and intraoperative findings: A case report. *Int J Surg Case Rep*. 2016;21:44-47. doi:10.1016/j.ijscr.2016.02.021

50. Chaitow L. *Positional Release Techniques E-Book*. Elsevier health sciences; 2007.

51. Goyal M, Goyal K, Narkeesh K, Samuel AJ, Sharma S, Chatterjee S. The strain – Counter strain technique in the management of anterior interosseous nerve syndrome: A case report. *J Taibah Univ Med Sci*. 2017;12(1):70-74. doi:10.1016/j.jtumed.2016.05.003

52. Wang Y, Liu T, Song L, et al. Spontaneous peripheral nerve palsy with hourglass-like fascicular constriction in the upper extremity. *J Neurosurg*. 2019;131(6):1876-1886. doi:10.3171/2018.8.jns18419

53. Alfen N. Trapped or twisted? Teasing out anterior interosseous neuropathy. *Muscle Nerve*. 2020;61(3):268-270. doi:10.1002/mus.26811

54. Sneag DB, Arányi Z, Zusstone EM, et al. Fascicular constrictions above elbow typify anterior interosseous nerve syndrome. *Muscle Nerve*. 2020;61(3):301-310. doi:10.1002/mus.26768

55. Schantz K, Riegels-Nielsen P. The Anterior Interosseous Nerve Syndrome. *Journal of Hand Surgery*. 1992;17(5):510-512. doi:10.1016/s0266-7681(05)80232-3

56. Krishnan KR, Sneag DB, Feinberg JH, et al. Outcomes of Microneurolysis of Hourglass Constrictions in Chronic Neuralgic Amyotrophy. *J Hand Surg Am*. 2021;46(1):43-53. doi:10.1016/j.jhsa.2020.07.015

57. Damert HG, Hoffmann R, Kraus A, Stowell RL, Lubahn J. Minimally Invasive Endoscopic Decompression for Anterior Interosseous Nerve Syndrome: Technical Notes. *J Hand Surg Am*. 2013;38(10):2016-2024. doi:10.1016/j.jhsa.2013.07.026

58. Ochi K, Horiuchi Y, Tazaki K, Takayama S, Matsumura T. Surgical treatment of spontaneous anterior interosseous nerve palsy: A comparison between minimal incision surgery and wide incision surgery. *Journal of Plastic Surgery and Hand Surgery*. 2013;47(3):213-218. doi:10.3109/2000656x.2012.753079

59. Keiner D, Tschabitscher M, Welschehold S, Oertel J. Anterior interosseous nerve compression syndrome: Is there a role for endoscopy? *Acta Neurochir*. 2011;153(11):2225-2229. doi:10.1007/s00701-011-1091-5