

CASE REPORT



Anterior interosseous nerve neuropathy in a patient with spinal cord injury: case report and literature review

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INTRODUCTION: Entrapment neuropathies, typically carpal tunnel syndrome and ulnar neuropathy, frequently occur in patients with spinal cord injury (SCI). Upper limb impairments due to entrapment neuropathy can be particularly debilitating in this population. Anterior interosseous nerve (AIN) neuropathy has not been previously described in the SCI population.

CASE PRESENTATION: A 27-year-old left-handed man with a history of C7 ASIA Impairment Scale B spinal cord injury five years prior presented to clinic with decreased left thumb function as well as thumb flexion. Workup including nerve conduction studies, electromyogram, ultrasonographic assessment, and magnetic resonance neurography was consistent with compressive AIN neuropathy. Surgical exploration and neurolysis was performed, with improvement of symptoms.

DISCUSSION: Entrapment neuropathies should be carefully considered in the evaluation of patients with SCI with new motor deficits. We report a case of AIN neuropathy in a patient with SCI successfully treated with surgical decompression, and review the literature describing upper extremity entrapment neuropathies in this population. Surgical decompression is an effective option for treatment of AIN neuropathy in the setting of SCI, though further characterization of the optimal management strategy is needed.

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BACKGROUND

Traumatic spinal cord injury (SCI) occurs at an annual incidence of 54 per million population in the United States [1] and is a significant source of long-term disability, high healthcare utilization, and psychosocial challenges in affected patients and their caregivers [2, 3]. Entrapment neuropathy commonly occurs in patients with SCI as the upper limbs take on new roles related to mobility. The rate of carpal tunnel syndrome in this population ranges from one-half to two-thirds [4–6], while ulnar neuropathy has been reported to occur in 40% of patients with SCI [4, 7]. In contrast, carpal tunnel syndrome and ulnar neuropathy occur in less than 5% of the general population [8, 9]. It has been suggested that the high prevalence of such injuries is due to the arm motions involved in wheelchair mobility, leading to overuse and strain on wrist flexor tendons and ultimately causing nerve compression [5, 7, 10].

As most patients with SCI are particularly reliant on use of their upper extremities to perform activities of daily living, entrapment neuropathies of the upper limb can be particularly debilitating. Appropriate nerve decompression can provide long-term symptom relief; however, clinical workup can be challenging given pre-existing neurologic deficits. Anterior interosseous nerve (AIN) neuropathy is a pure motor neuropathy comprising less than 1% of all upper extremity neuropathies [11, 12] and has not been previously reported in a patient with SCI. We report a case of compressive AIN neuropathy causing loss of thumb function and threatening the functional independence of a tetraplegic patient with SCI successfully treated with surgical decompression.

CLINICAL PRESENTATION

A 27-year-old left-handed man with a history of C7 ASIA Impairment Scale (AIS) B spinal cord injury after a diving accident treated with C6-T1 anterior cervical corpectomy and fusion five years prior presented to our institution with decreased left thumb function and self-reported spontaneous thumb flexion. He reported a hyperflexion injury to the neck two weeks prior when his wheelchair came to a sudden stop, after which he stated he was only able to achieve full active thumb flexion while hyperextending his neck.

On physical exam, the patient was unable to oppose or flex the thumb. He had no new sensory deficit and reported no prodrome of pain in the forearm. Repeat cervical spine magnetic resonance (MR) imaging did not demonstrate a syrinx or worsening myelomalacia. He underwent a nerve conduction study which demonstrated markedly reduced amplitude motor amplitudes of C8-T1 innervated abductor pollicis brevis (median) and abductor digiti minimi (ulnar) of the hand, but corresponding sensory nerve action potentials from the 2nd and 5th digits respectively were within normal limits consistent with a preganglionic injury to the spinal cord grey matter [13]. Electromyography (EMG) showed a few discrete volitional motor units with reduced recruitment and activation patterns, but no fibrillation potentials or positive sharp waves to suggest active muscle denervation. Unfortunately, no recent EMG or nerve conduction velocity study (NCV) was available for comparison. This raised the possibility of AIN entrapment, but was not diagnostic. Ultrasonographic assessment

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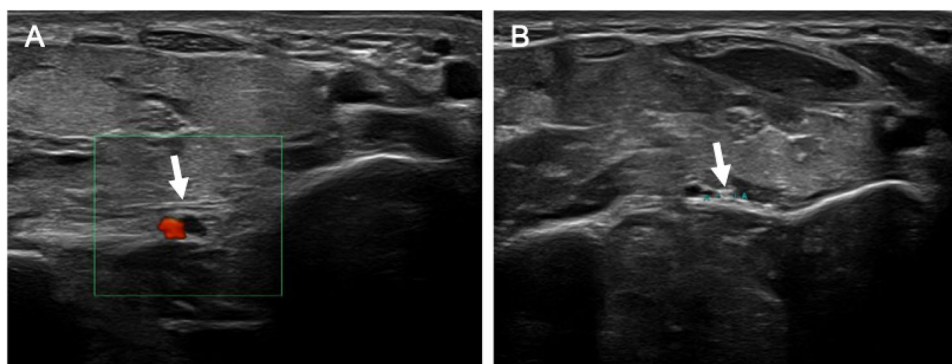


Fig. 1 **Ultrasound assessment at initial encounter.** **A** Transverse ultrasound image of the mid left forearm demonstrating an abnormally thickened anterior interosseous nerve (arrow) adjacent to its accompanying vessel (red color). **B** Comparison of the unaffected right forearm with normal AIN and barely perceptible hypoechoic fascicles adjacent to its accompanying vessel (black color).

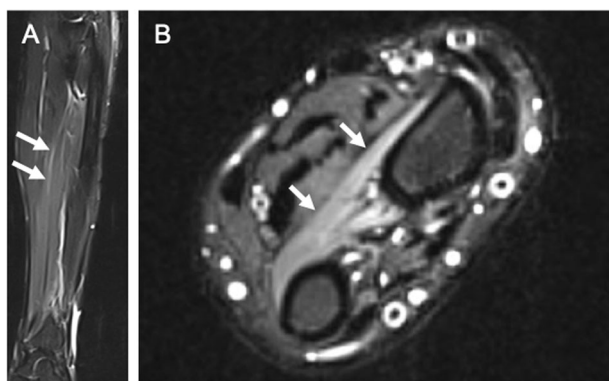


Fig. 2 **Preoperative MRI of the left forearm.** **A** T2 weighted fat suppressed MRI of the left forearm demonstrates thickening and hyperintensity of the median nerve focally in the mid forearm. **B** Downstream diffuse muscle denervation edema of the pronator quadratus, which is innervated by the AIN.

of the AIN revealed fascicular enlargement and hypoechoogenicity of the left AIN within the distal forearm (Fig. 1). MR neurography of the left forearm was notable for diffuse edema of the forearm flexor muscles and median nerve edema distal to the pronator teres (Figs. 2 and 3) consistent with subacute or chronic denervation. Comparison with the contralateral side was not performed.

After his clinical exam deteriorated, the patient continued with ongoing physical therapy as needed for his functional baseline. No other interventional strategies were taken. Imaging and EMG studies were obtained over the course of three months, during which the patient had no symptom improvement. At this point, the patient underwent surgical exploration and neurolysis of the AIN (Fig. 4). Compression was noted at the intramuscular septum and from the deep head of the pronator teres, which was noted to be very hypertrophied. There was additionally a vascular leash crossing the AIN and a fibrous band from the flexor digitorum superficialis along the course of the AIN, which were also sharply divided.

The patient recovered well postoperatively and was discharged later that day. He did not have any specific activity restrictions and had a compressive dressing over the incision for two days. He had initial improvement of his symptoms noted at a two-week postoperative visit which continued to near baseline until three months postoperatively, at which time he had recurrent weakness with 1/5 thumb flexion. This was evaluated with repeat EMG, nerve conduction velocity studies, and MRI, which were unrevealing and demonstrated continued improvement. However, this weakness had resolved by the time he returned to clinic four

months postoperatively, at which time his thumb flexion was at his baseline strength of 4/5. EMG was again performed, demonstrating dramatic improvement in the volitional activation pattern of the left flexor pollicis longus consistent with resolution of peripheral demyelinating/neuropathic lesion. MR neurography was performed in the interim without evidence of compression or abnormality.

DISCUSSION

AIN neuropathy is an uncommon pure motor neuropathy, the diagnosis of which is complicated in patients with SCI due to existing neurologic deficits. The etiology and pathophysiology of AIN neuropathy remain uncertain, and the condition has been characterized as a neuritis as well as a compression neuropathy [14, 15]. In the present case, preoperative and intraoperative evidence of compressive neuropathy were demonstrated, with resolution of symptoms following neurolysis. Additionally, an element of compression due to a hypertrophied pronator teres was noted intraoperatively, though the patient did not exhibit any new sensory deficit which would be expected of pronator teres syndrome [11].

Notably, the patient stated that he noticed his symptoms after a relatively minor trauma. However, findings of edema and muscle atrophy seen on the MRI and US point instead to chronic compression. Given the extensive denervation changes seen on his MRI, this was felt to be a coincidental trauma that prompted the patient to seek evaluation, which revealed his underlying chronic pathology.

The patient's remaining thumb function was critical for him to operate his wheelchair and cell phone. After consideration of the imaging findings, as well as the patient's wishes to improve as quickly as possible, the decision was made to proceed with an operation rather than watchful waiting, and he was able to return to better than baseline function at his next follow up visit. Conservative management strategies, which include physical therapy to maintain passive range of motion, nerve gliding exercises, and neuromuscular electrical stimulation protocols, have been described in the management of AIN neuropathy [16, 17], though the timeframe of spontaneous recovery ranges from 3 to 24 months [14], which may be untenable for patients with significant mobility limitations at baseline.

The patient experienced a transient recurrence of thumb weakness at 3 months postoperatively. Repeat imaging, EMG, and NCV were unrevealing. While the precise cause of the current patient's weakness could not be determined, treatment failure following surgery to correct compressive neuropathy is not uncommon and may be due to extraneural scarring, inflammation, or fibrous proliferation, contributing to recurrent nerve

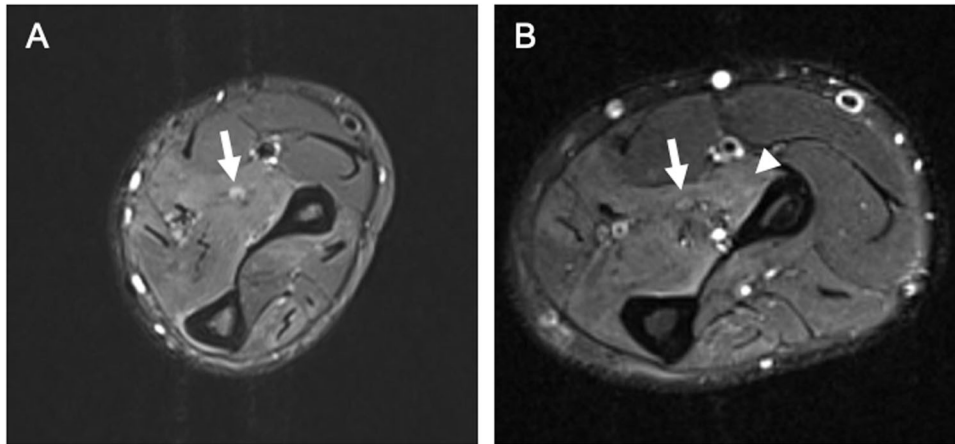


Fig. 3 Preoperative MRI of the left forearm. **A** Axial T2 weighted fat suppressed images of the left forearm preoperatively and **B** three months following decompression. The median nerve (white arrow) is abnormally T2 hyperintense on the preoperative image, indicating neural edema/neuropathy. The nerve signal normalizes on the postoperative image. The AIN was not well seen. Note the geographic A9 muscle denervation edema in some of the flexor musculature (arrowhead).

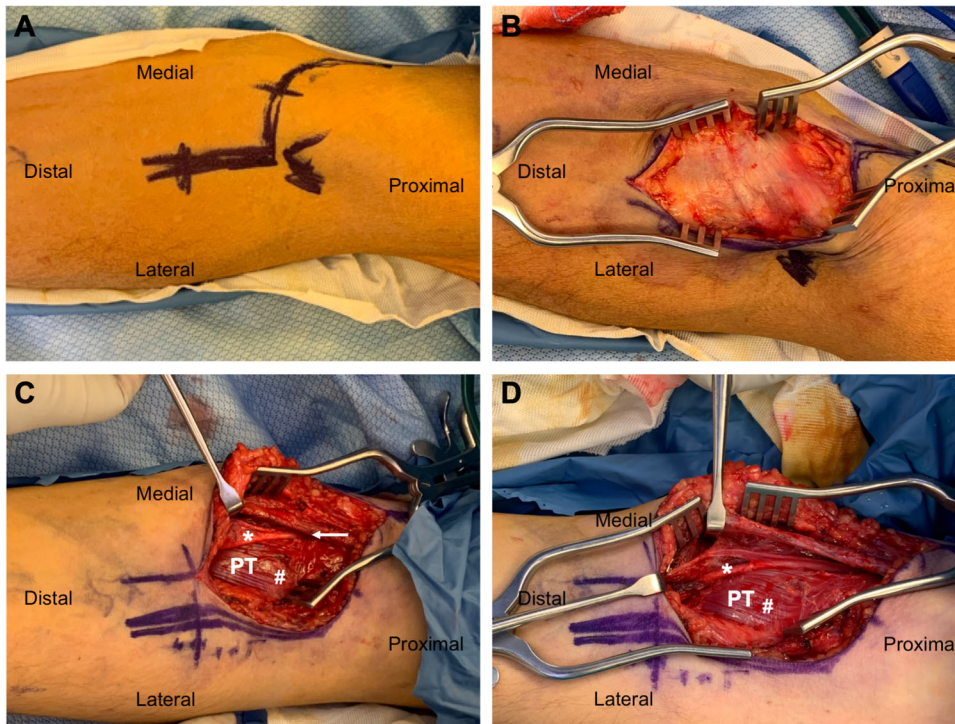


Fig. 4 Intraoperative images of AIN neurolysis. **A** The marked incision follows the antecubital creases and distally traces the intermuscular septum to allow for proximal decompression if needed. The biceps tendon is marked. **B** Initial dissection demonstrating the superficial fascia. **C** Median nerve (*) with compression at the intermuscular septum (arrow). Hypertrophied deep pronator teres (PT) is seen within the operative field (#) after decompression. **D** Decompression of the median nerve. A vascular leash (not pictured) crossing the median nerve was divided.

compression [18]. In this case, the patient's spontaneous recovery and continued improvement were reassuring and highlight the need for continued postoperative monitoring for symptom recurrence.

The workup for new deficits in patients with SCI should include a careful consideration of entrapment neuropathy, which may occur secondary to new patterns of activity required for mobility and daily tasks. As even minor changes to upper extremity function have a large impact on quality of life in this population [19, 20], timely diagnosis and treatment of entrapment

neuropathies is crucial. After progression of existing spinal cord injury is ruled out, electrophysiologic testing is a crucial tool in the evaluation of peripheral neuropathies and may be of particular value in patients with SCI [14, 21–23]. However, since an acquired mononeuropathy is often superimposed on chronic changes from grey matter degeneration after SCI [24], further evaluation of the peripheral nerves via ultrasound and MR neurography may also be essential for localization [25].

Upper extremity entrapment neuropathies, namely carpal tunnel syndrome and ulnar neuropathy, frequently occur in

patients with SCI. Carpal tunnel release and ulnar nerve release may be readily performed in such patients to treat severe symptoms [26]. Less commonly, entrapment neuropathies may occur elsewhere in the upper extremity. Shoulder pain and functional limitation has been described in patients with SCI with axillary nerve and radial nerve entrapment treated successfully with surgical decompression [27], as well as suprascapular nerve entrapment treated successfully with glucocorticoid injection [28].

We demonstrate a case of successful surgical treatment of AIN neuropathy in a patient with SCI. In addition to non-operative strategies, another surgical approach is brachialis-to-AIN nerve transfer, which has been previously described to restore hand function in patients with SCI and tetraplegia [29, 30] and may be a feasible alternative depending on symptom severity. Though patients stand to benefit greatly from operative restoration of upper extremity function, it should be noted that patients with SCI have a higher risk of developing perioperative and postoperative complications from elective surgery [31], necessitating careful consideration of the risks and benefits before pursuing surgical intervention. Given the potentially challenging diagnosis and debilitating nature of entrapment neuropathies in patients with SCI, greater awareness of their evaluation and management is desirable.

CONCLUSIONS

AIN neuropathy is rare and has not been previously described in patients with SCI. Entrapment neuropathies, ranging from AIN neuropathy to the more common carpal tunnel syndrome and ulnar nerve neuropathy, should be carefully considered in the evaluation of patients with SCI who present with new motor deficits. New weakness of the flexor pollicis longus and flexor digitorum profundus to the second and third digits should prompt evaluation for AIN neuropathy, which may be challenging to diagnose in patients with SCI and significant motor impairment at baseline. Surgical decompression is an effective option for treatment of AIN neuropathy in the setting of SCI, though further characterization of the optimal management strategy is needed.

DATA AVAILABILITY

Data relating to this case are available upon reasonable request to the authors.

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AUTHOR CONTRIBUTIONS

All authors contributed equally and substantially to this case report.

COMPETING INTERESTS

The authors declare no competing interests.

ADDITIONAL INFORMATION

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