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Case Report

Subacute Ulnar Nerve Compression Neuropathy Following Hand Crush Injury in the Setting of Intracanal Accessory Abductor Digiti Minimi: A Double Crush Phenomenon

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Anatomical variations within Guyon's canal such as an accessory abductor digiti minimi are described as causes of ulnar nerve compression. Here we present a unique case of delayed ulnar neuropathy following treatment of left fourth metacarpal base fracture with percutaneous pinning fixation and an uncomplicated two month postoperative course. He returned with new ulnar sensory loss and motor weakness. EMG demonstrated nerve compression with CT identifying an accessory abductor digiti minimi in Guyon's canal. Following Guyon's canal release with partial accessory muscle resection, there was immediate sensory and progressive motor recovery with resolution of clawing. Delayed compression by an accessory abductor digiti minimi following trauma has not been described, suggestive of double-crush phenomenon. The accessory muscle was an asymptomatic variable (first "crush") and with the second "crush" of post-surgical changes resulting in pathological nerve compression. With delayed onset ulnar neuropathy after trauma, surgeons should consider possible accessory structures.

Compression neuropathies, such as carpal tunnel syndrome (CTS) or ulnar nerve compression, are painful, insidious, and often debilitating for a patient. Ulnar nerve compression most commonly occurs in the cubital tunnel of the elbow, followed by Guyon canal in the wrist. Typically, patients present with numbness or paresthesia in the ulnar distribution, including the little finger and ulnar aspect of the ring finger. When compression is prolonged and severe, patients can develop motor weakness and muscle atrophy. Common causes of ulnar nerve compression at Guyon canal include repetitive hand/wrist use, masses or tumors, and trauma. More rare causes of ulnar nerve compression can be attributed to accessory muscles that cause direct compression of the nerve. Interestingly, the most common accessory muscle in Guyon canal is the accessory abductor digiti minimi (AADM), occurring in 22.4% of the wrists.[1] This muscle follows a similar path to the true abductor digiti minimi, originating from the antebrachial fascia and inserting into the ADM. In contrast to this, it can also occur as a separate muscle that inserts into the medial aspect of the proximal phalanx of the little finger.

Although uncommon, published cases of the AADM causing compression of the ulnar nerve at Guyon canal exist.[2,3] We present a unique case of a double crush phenomenon caused by trauma-induced inflammation of an anatomical muscle variant.[4] We hypothesize that the compression in our case was because of combination of inflammation and irritation resulting in compression in Guyon canal that was accentuated by the presence of AADM variant muscle in the canal by further reducing the possible space within the canal.

Case Description

The patient was a young man manual laborer who presented after a crush injury to his nondominant hand with a fracture of the fourth metacarpal base. He underwent closed reduction with percutaneous pinning of the metacarpal fracture within 10 days. The patient initially experienced an uncomplicated postoperative course. However, two months later the patient presented to clinic

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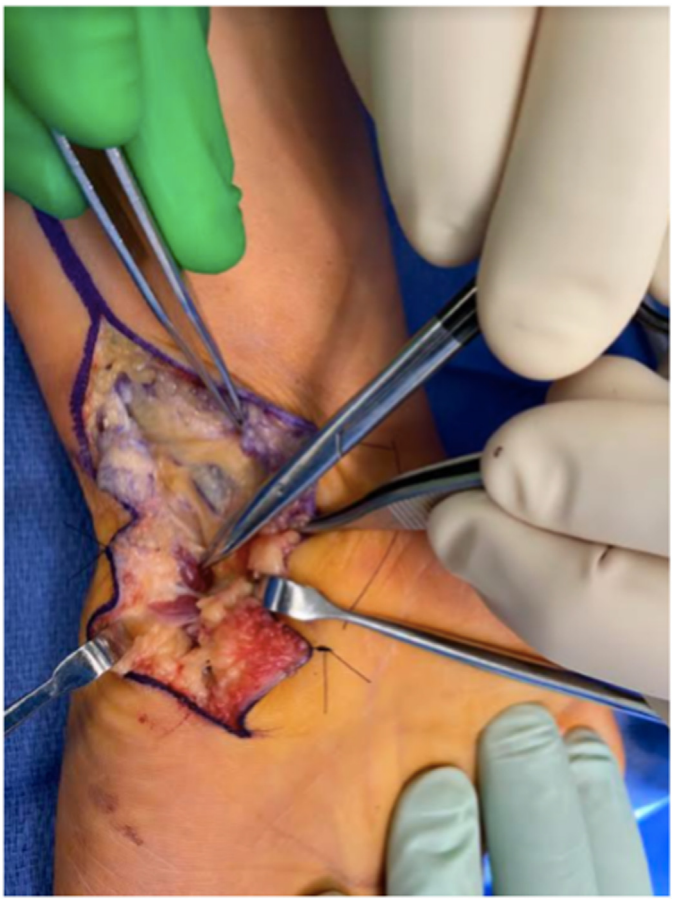

Figure 1. Accessory abductor digiti minimi in situ.

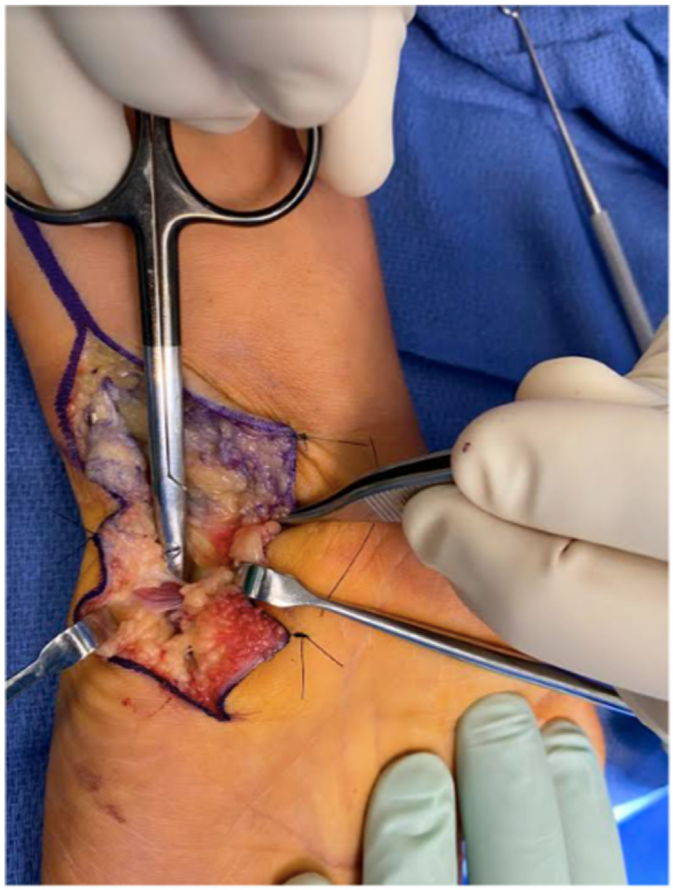

Figure 2. Guyon canal below AADM.

complaining of new-onset, progressive sensory loss in the ulnar distribution as well as a positive Wartneberg sign with mild resting ulnar claw deformity indicative of motor weakness. EMG was suggestive of ulnar nerve compression at the wrist. Computed tomography scan demonstrated an AADM muscle in Guyon canal. The patient subsequently underwent Guyon canal release with partial resection of the accessory muscle, resulting in near-immediate sensory recovery and early progressive motor recovery with resolution of clawing and return to work. Intrasurgical photographs of the AADM and Guyon canal are included in Figures 1 and 2, respectively. Written informed consent was obtained from the patient for the publication of this case report and accompanying images.

Discussion

Multiple case reports documenting ulnar nerve compression at Guyon canal by an AADM muscle exist. Dimitriou and Natsis[5] reported ulnar nerve compression by an AADM in a 60-year-old Greek housewife; they attributed compression to muscular hypertrophy secondary to extensive manual labor without known trauma. James et al[6] presented a case series of two patients who experienced ulnar compression secondary to an AADM. The first patient was a 37-year-old housewife with a known trauma who presented with loss of sensation; she had full sensory recovery at 6 months. The second patient was a 50-year-old man who worked as a hospital porter and presented with both pain, numbness, and early clawing of his ring and little fingers. Additionally, he recently experienced a fifth metacarpal chip fracture. After Guyon canal release, this patient had a full sensory recovery but incomplete motor recovery at one year.[6]

Our patient has similarities to each of these three—a young, manual laborer with a known fracture. However, his ulnar nerve compression presented differently, and only after having surgical repair of his fracture. He denied any numbness or paresthesia at the time of injury as well as immediately after surgery. His first report of ulnar compression was not until 2 months after surgery. Although considering possible causes of this delayed presentation, a double crush syndrome (DCS) was proposed secondary to the inflammation and healing cascade causing ulnar nerve compression that was only accentuated by the presence of the AADM.

The concept of a DCS was initially introduced in 1973 by Upton and McComas[7] after a comprehensive EMG study identified that approximately 70% (81 of the 115 cases) of patients with peripheral nerve compression (CTS or ulnar compression at the elbow) had associated cervical root lesions in the neck. Their theory states that a proximal lesion along a peripheral nerve increases the nerve's vulnerability to a second lesion. Thus, compression at one location may be asymptomatic, as with this patient's AADM, but when coupled with further compression, as in the trauma-induced inflammation, the initial compression becomes apparent.[7] Further studies have shown similar associations, with Hurst et al[8] revealing a statistically significant higher incidence of bilateral CTS in patients with osteoarthritis of the cervical spine. Additionally, Wood et al[9] demonstrated that 44% of patients with thoracic outlet syndrome had EMG evidence of distal compression. Although the exact pathophysiology of DCS is unknown, one theory focuses on impaired axonal transport of nutrients bidirectionally, increasing

nerve susceptibility to ischemic changes.[4] However, debates persist regarding the legitimacy of DCS as other studies produce contradictory results. Morgan et al[10] conducted a retrospective review that showed that only 0.8% of patients with peripheral compression neuropathy had an acceptable cervical root lesion that could fit DCS.

Although an AADM muscle has been described as a potential cause for ulnar nerve compression, uniquely, a description of a delayed onset of compression suggestive of the double crush phenomenon does not exist. The patient was asymptomatic before injury and in the acute postoperative period; hence, it is safe to say that the presence of the accessory muscle alone did not produce symptoms nor was any direct injury to the nerve responsible for this. We suspect that the presence of the AADM muscle was an asymptomatic variable (or first "crush") that when combined with the second "crush" of posttraumatic edema and postsurgical changes (ie, bony callous formation, muscle fibrosis, and subacute edema) resulted in pathologic nerve compression. Given the proximity of the healing fourth metacarpal base to the path of the ulnar nerve, we hypothesize that the expected timeline of the proliferative phase of healing at and around the fracture site is responsible for the delayed clinical presentation of both motor and sensory neuropathy 2 months after the initial trauma and surgery. In cases of delayed-onset ulnar neuropathy in the setting of trauma, surgeons should consider the potential presence of accessory structures as well. In addition, concurrent release of Guyon canal when these are known to be present even in cases of minor trauma to prevent detrimental loss of function should also be considered.

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