

## Case Report

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# Anterior interosseous nerve palsy in the early postoperative period after open capsular release for elbow stiffness: a case report

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Surgical release of elbow contracture is associated with injury to structures traversing the elbow. To date, only one other case report has been published describing anterior interosseous nerve (AIN) palsy that developed immediately after open elbow contracture release and debridement. Here we describe the unique case of a patient that developed AIN palsy 1 week after operation, including magnetic resonance imaging and electrodiagnostic studies, to shed some light on the etiology of this rare complication.

**Keywords:** Anterior interosseous nerve palsy; Elbow; Nerve compression; Nerve injury; Basic science

Degenerative osteoarthritis of the elbow is uncommon, affecting less than 2% of the general population [1]. It can cause severe disabling symptoms such as pain, locking and stiffness [1]. Several methods of arthroscopic and open surgical treatment have been described, resulting in improvements in pain, range of motion and patient satisfaction [1]. However, while arthroscopic release is minimally invasive, there are reports of higher incidences of nerve injury compared to open capsular debridement. In a survey distributed to over 372 members of the American Society for Hand Surgery, 60% of hand surgeons described at least one peripheral nerve injury after elbow arthroscopy over a 5-year period, with nearly half requiring operative intervention [2]. In a case report, Desai et al. [2] was the first to describe a direct traumatic injury to the anterior interosseous nerve (AIN) after arthroscopic capsular debridement of an elbow.

AIN palsy is an uncommon cause of hand weakness that comprises less than 1% of all upper extremity nerve palsies [3]. AIN syndrome was first described by Parsonage-Turner and then reported as a distinctive entity by Kiloh and Nevin in 1952 [3,4].

AIN palsy after elbow contracture release is a rare occurrence compared to more common etiologies of AIN palsy including trauma, iatrogenic injury from surgery, postoperative dressings, crutches, and venipuncture [3,5]. To date, only one other case report has been published describing an AIN palsy that developed immediately after open elbow contracture release and debridement [5]. This case report describes a patient who developed delayed-onset AIN palsy approximately 1 week after open elbow release with a protracted course and partial recovery and provides a distinct injury mechanism.

Approval for this study was waived by the Institutional Review Board, and informed consent was obtained from the patient.

## CASE REPORT

### Initial Evaluation

A 42-year-old left hand-dominant male police officer presented to the office with complaints of 15 years of worsening right elbow pain and limited range of motion. His past medical history was

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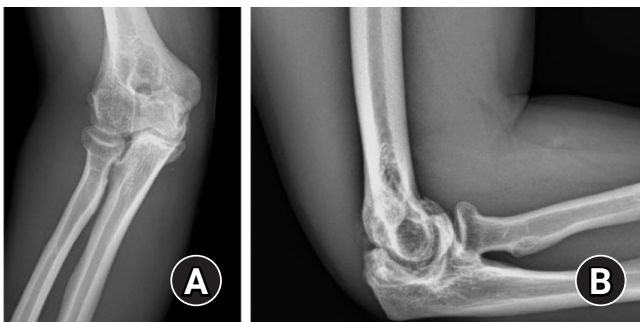
significant for herpes simplex virus type 2 (HSV-2), which was treated with daily acyclovir. His elbow range of motion included a flexion-extension (FE) arc of 40°–100° and supination-pronation arc of 50°–70° with pain present at the terminal range of motion. There were no symptoms of ulnar neuritis and no signs of ulnar nerve instability or compression neuropathy. Radiographs and magnetic resonance imaging (MRI) of his right elbow demonstrated osteoarthritic changes with osteophyte formation at the radiocapitellar and ulnohumeral joint and evidence of multiple intra-articular loose bodies (Fig. 1). After failure of extended nonsurgical treatment, persistent elbow pain at the end ranges of motion, and mechanical symptoms (locking and catching), the patient elected to proceed with open elbow contracture release, debridement and removal of loose bodies.

### Surgery

The patient underwent open elbow contracture release under regional anesthesia. A lateral column/lateral ulnar collateral ligament sparing approach was utilized. Anterior and posterior capsulectomy, removal of loose bodies, deepening of the radial, coronoid and olecranon fossa and exostectomy of the tip of the olecranon and coronoid were performed through this surgical approach. There were no intraoperative complications and intraoperative elbow range of motion after release included FE arc of 0°–140° and supination-pronation arc of 80°–80°. Patient had a normal neurological exam after the block wore off. Supervised physical therapy was started on postoperative day (POD) 1 and consisted of active and active assisted motion exercises; edema control modalities including edema sleeve, static splinting and nighttime elevation were utilized.

### Postoperative Follow-up

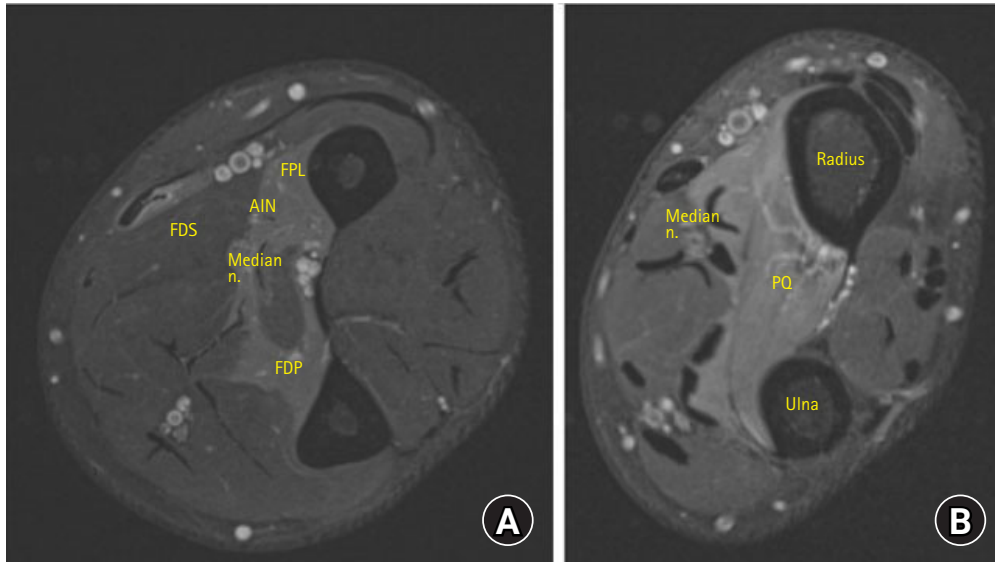
On POD 7, the patient reported a sudden inability to bend the



**Fig. 1.** Preoperative radiographs including (A) anteroposterior and (B) lateral of the right elbow demonstrating osteoarthritic changes with osteophyte formation at the radiocapitellar and ulnohumeral joint and evidence of multiple intra-articular loose bodies.

tip of his right thumb after he reflexively reached out to break a fall, causing hyperextension of his operative elbow. He reported an immediate tearing sensation along the volar aspect of his forearm, followed by tingling in his thumb. He reported considerable pain localized to his elbow, which improved with time; however, he subsequently noticed mild pain and weakness in thumb flexion. The patient was seen in the office the next day and had ecchymosis around his elbow with postoperative pain. The operative arm was sensate to light touch in the distribution of C5–T1 dermatomes but had weakness (3+/5 in thumb flexion) and tingling on the radial side of the thumb. He was able to actively flex the 3rd, 4th, and 5th digits without difficulty. On POD 12 the patient's tingling around his thumb had resolved; however, he was now experiencing tingling localized to his index finger with difficulty pinching his thumb and index finger. There were no other symptoms localized to the operative arm or in the neck.

Diagnostic and imaging studies to rule out hematoma in the elbow leading to AIN palsy versus a spontaneous rupture of flexor pollicis longus (FPL) were conducted. MRI of the elbow showed postoperative changes around the elbow without any considerable hematoma. MRI and ultrasound of the wrist demonstrated asymmetric fatty atrophy with volume loss of the right pronator quadratus (PQ) muscle compared to the left, suggestive of an anterior interosseous palsy but no lesions along the course of the AIN (Fig. 2). On MRI neurogram, there was intramuscular edema within the flexor digitorum profundus (FDP), FPL, and PQ. The AIN, posterior interosseous nerve, ulnar, radial and median nerve were all in continuity and there was no evidence of nerve thickening or neuroma. Electrodiagnostic studies (electromyography and nerve conduction studies) demonstrated abnormal sharp waves, abnormal latency, and abnormal compound motor action potentials at both the FPL and opponens pollicis (Fig. 3). Six months postoperative both his FPL and FDP to the index finger were 3/5, at which time nerve exploration of the AIN was discussed. Despite what he reported as “clumsiness” of his fingers he continued to work with both occupational and physical therapy and declined to have exploratory surgery. One-year postoperative our patient still had some weakness (4/5 FPL and FDP) but was now able to perform pinching and grasping function with index finger and thumb. On the final 2-year follow-up, the patient's elbow range of motion was from 10°–130° for FE arc and 60°–70° for supination-pronation arc with a Quick Disabilities of the Arm, Shoulder and Hand (QuickDASH) questionnaire score of 11.4. Patient had similar strength at 2 years as he did at 1 year 4/5 for FPL and FDP.



**Fig. 2.** Magnetic resonance imaging neurogram: axial cuts of the forearm and wrist demonstrating (A) intramuscular edema within the flexor digitorum profundus (FDP) and flexor pollicis longus (FPL), with both the median nerve and anterior interosseous nerve (AIN) labeled. (B) Intramuscular edema and mild fatty atrophy of the pronator quadratus (PQ). FDS: flexor digitorum superficialis.

## DISCUSSION

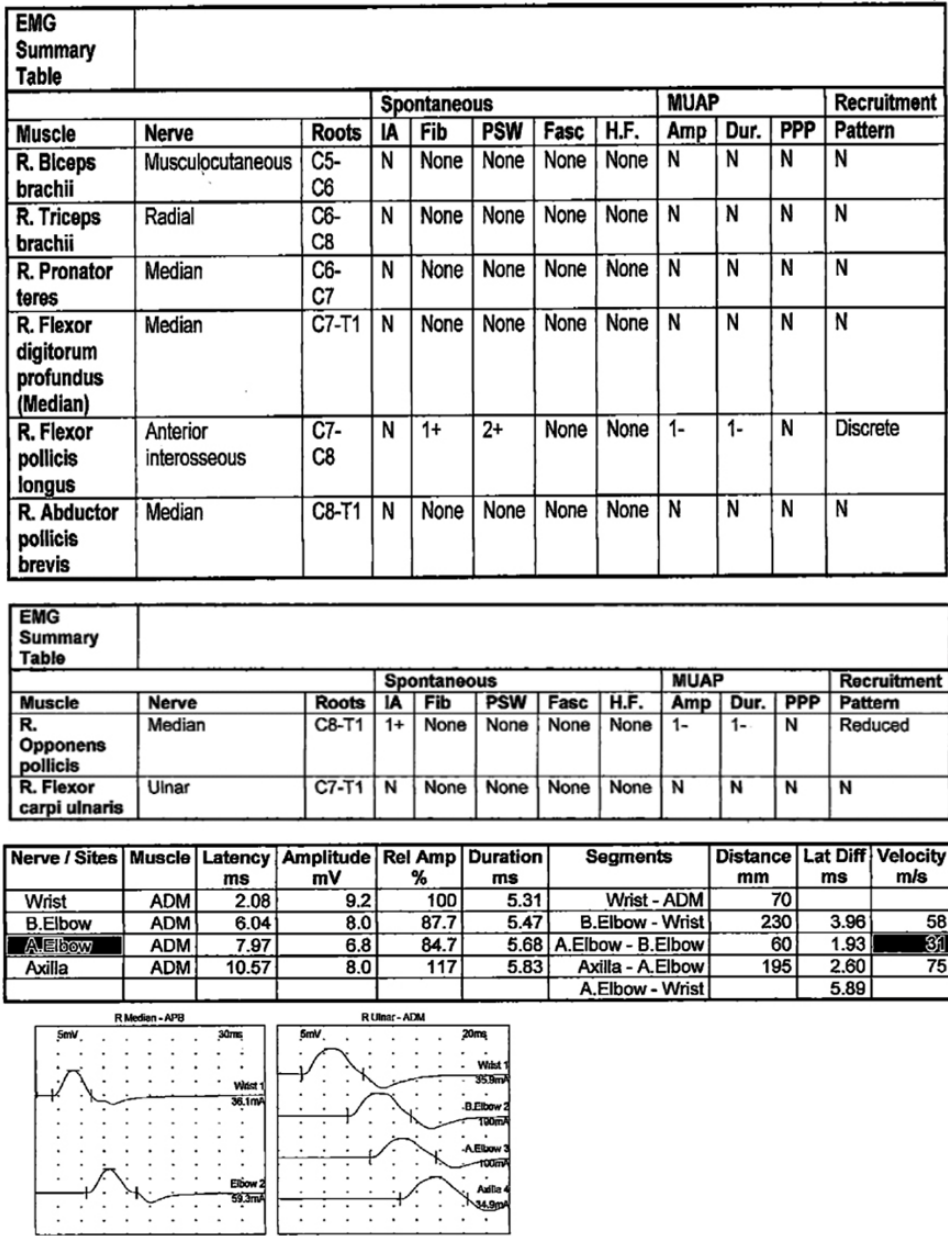
In this case report we present a case of AIN palsy that occurred seven days after open elbow contracture release. Although AIN palsy is rare, especially after open elbow contracture release, this case is unique in that the presentation of AIN palsy was delayed and our patient continues to have residual deficits 24 months out from the initial event. Furthermore, the utilization of advanced imaging and electrodiagnostic studies proved to be useful in identifying the location of our patient's deficit.

The AIN innervates the deep muscles of the forearm, including the FPL, the FDP to the index and middle fingers, and the PQ muscle. AIN syndrome is typically characterized by forearm pain and partial or complete dysfunction of AIN-innervated muscles [2]. AIN palsies can manifest clinically as weakness in flexion of the interphalangeal joint of the thumb, distal interphalangeal joints of the index and middle fingers, and possibly with weakness of pronation with the elbow in flexion [3]. Characteristic physical exam findings include the “O” sign (Kiloh-Nevin sign) due to inability to actively flex the interphalangeal joint of the thumb with the distal interphalangeal joint of the index finger [3]—all presenting symptoms in our patient.

The pathophysiology and etiology of AIN syndrome remain unclear and include elbow and forearm trauma, immune-mediated inflammatory neuritis, and spontaneous idiopathic cases [2, 3,6,7]. Several mechanisms have been proposed to explain postoperative anterior interosseous syndrome after upper extremity

surgery [5]. Peripheral neuropathy as a complication after regional anesthesia, external compression, and Parsonage Turner syndrome (PTS) [3,8] have been described as potential etiologies [3]. Our patient had two risk factors for an acute episode of PTS postoperative, which included acyclovir therapy for HSV-2 as well as suffering from an upper respiratory viral infection prior to his elbow procedure; however, an isolated AIN palsy as solitary presentation is very uncommon [4]. The part of the AIN that descends under the flexor digitorum superficialis (FDS) arch is prone to traction injury and, has limited capacity to tolerate stretch. It is likely that our patient's long standing elbow contracture, increased postoperative range of motion and the sudden eccentric stretching episode of our patient when he braced himself while falling led to a traction injury of the AIN in the transition zone. This mechanism could also potentially explain the etiology in the prior case report by Katolik and Cohen [5], where the authors proposed that intraoperative passive stretching of the elbow in extension during surgery was the most likely etiology of immediate postoperative AIN palsy.

Typically, imaging is not helpful for diagnosing AIN syndrome because imaging is usually unremarkable [2,3,9]. However, Dunn et al. [9] and Grainger et al. reported that the most reliable indicator of neuritic AIN palsy was edema within the PQ. These signal changes reflect a shift of muscle water from the intracellular to the extracellular space in the absence of a net increase in total muscle water content occurring within the first 24 hours after muscle denervation [9]. This correlates closely with our patient;



**Fig. 3.** Electrodiagnostic studies (electromyography [EMG] and nerve conduction) demonstrating abnormal sharp waves, abnormal latency, and abnormal compound motor action potentials at both the flexor pollicis longus and opponens pollicis. MUAP: motor unit action potential, IA: insertion activity, Fib: fibrillations, PSW: positive sharp waves, Fasc: fasciculations, H.F: high frequency, Amp: amplitude, Dur: durations, PPP: polyphasic potential, R: right, N: normal, B: below, A: above, ADM: abductor digiti minimi, APB: abductor pollicis brevis.

both ultrasound and MRI neurogram demonstrated intramuscular edema within the FDP, FPL, and PQ. In our patient, electromyography demonstrated abnormal sharp waves, abnormal latency, and abnormal compound motor action potentials at both the FPL and opponens pollicis allowing us to conclude that the pathology was within the transition zone, as the nerve dips below the aponeurotic arch of the FDS.

The literature about treatment of AIN palsies of all causes does

not support a clear advantage to surgical exploration and decompression [5]. Furthermore, the timing of surgical exploration is controversial. In a case series of 20 patients with acute AIN syndrome, Schantz and Riegels-Nielsen [10] demonstrated satisfactory results in 11 of 15 patients (73%) treated surgically versus 2 of 5 of those treated with observation. The authors ultimately concluded that AIN exploration is the treatment of choice. However, this interpretation may be misleading, because many of the

patients treated surgically were treated before the mean recovery time of those patients who were successfully treated non-surgically. We offered the option of surgical exploration of the AIN to our patient after 6 months of nonsurgical management. Although, the prognosis for spontaneous recovery is relatively good for AIN palsy, our patient did not have any signs of recovery until 9 months after the inciting event. At the last telehealth visit at 24 months he continued to have some residual weakness of his thumb with flexion.

In a case report of two patients who underwent open elbow capsular debridement, Katolik and Cohen [5] described immediate postoperative AIN palsy in both patients. The authors attributed their patient's AIN palsy to passive stretching of the elbow in extension after surgical release to correct residual elbow extension deficit [5]. Similar to the two cases described by Katolik and Cohen [5], our patient also developed an AIN palsy after open capsular release via lateral exposure. However, what makes our case unique is the patient developed an AIN palsy 1-week postoperative after reflexively extending his right elbow in an attempt to break a fall. We believe that trauma played a role in our patient's delayed presentation, but this extent of trauma in a non-surgical setting would not typically be sufficient to cause AIN palsy. Additionally, the two cases described by Katolik and Cohen [5] had complete resolution of their symptoms by 12 months postoperatively; however, our patient did not recover despite being close to 24 months out from surgery, thereby demonstrating that not all AIN palsies after open elbow release happen immediately after surgery or recover spontaneously. Additionally, we report findings of advanced imaging (MRI) and electrodiagnostic studies in this case report, shedding some light on the etiology of this rare complication.

In conclusion, we describe a case of delayed-onset AIN palsy developing one week after open elbow contracture release. We believe this was most likely a result of a stretch injury to the AIN nerve in the transition zone in the immediate postoperative period. Advanced imaging and electrodiagnostic studies demonstrated complete nerve continuity with no extrinsic compression. Although the overall prognosis for AIN palsy is generally good, AIN injury after elbow surgery can take a long time to recover. It is unclear, based on this single case report, if recovery would have been more rapid with surgical nerve exploration and decompression.

## NOTES

### Author contributions

Writing – original draft: CAC. Writing – review & editing: CAC, MB, JH, MSV.

### Conflict of interest

None.

### Funding

None.

### Data availability

None.

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