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Anconeus Epitrochlearis-Induced Cubital Tunnel Syndrome: A Rare Case Report

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Abstract

Cubital tunnel syndrome (CTS) is the second most common compression neuropathy of the upper limb. While most cases result from compression beneath Osborne's ligament or between the heads of the flexor carpi ulnaris, rare anatomical variations such as the anconeus epitrochlearis muscle (AEM) may also cause ulnar nerve entrapment. We report the case of a 40-year-old right-hand-dominant male presenting with progressive numbness and paresthesia along the ulnar aspect of the right forearm and into the fourth and fifth digits, accompanied by medial elbow pain. Clinical examination revealed a positive Tinel's sign over the cubital tunnel and symptom reproduction with sustained elbow flexion. Electromyography demonstrated severe focal slowing of ulnar nerve conduction at the elbow. Magnetic resonance imaging identified an accessory AEM compressing the ulnar nerve. Surgical decompression with resection of the accessory muscle was performed without ulnar nerve transposition. The patient experienced immediate postoperative symptom relief and remained asymptomatic at the six-month follow-up. Although uncommon, the AEM should be considered a potential cause of CTS, particularly in patients with atypical or refractory symptoms. MRI plays a crucial role in diagnosis, and surgical decompression provides reliable symptom resolution with excellent outcomes.

Categories: Orthopedics, Anatomy, Neurosurgery
Keywords: anconeus, anconeus epitrochlearis muscle, cubital nerve, cubital tunnel surgery, nerve transposition

Introduction

Compression neuropathy of the ulnar nerve at the elbow, commonly referred to as cubital tunnel syndrome (CTS), is the second most frequent entrapment neuropathy after carpal tunnel syndrome [1]. Ulnar nerve compression most often occurs either dynamically, when the nerve subluxates or dislocates from the ulnar groove, or statically, within the cubital tunnel beneath the cubital tunnel retinaculum and Osborne's ligament, particularly between the two heads of the flexor carpi ulnaris (FCU) [2].

The ulnar nerve provides sensory innervation to the palmar and dorsal aspects of the fifth digit and the ulnar half of the fourth digit. Neuropathy may also result from proximal compression at the cervical spine or brachial plexus. One uncommon but recognized cause of CTS is the anconeus epitrochlearis muscle (AEM), a small accessory muscle first described by Gruber in 1867 [3]. The AEM represents an anatomical variant that may contribute to ulnar nerve compression, as reported in several case series [4]. The muscle originates from the medial epicondyle of the humerus, courses over the ulnar nerve, and inserts into the olecranon. Although rare in humans, it is common in several mammals, reptiles, and amphibians and is believed to be an evolutionary remnant associated with climbing behavior [4]. Interestingly, some authors have suggested that the presence of this muscle may offer a protective effect to the ulnar nerve by replacing the rigid Osborne's ligament with more compliant muscular tissue [2].

In this report, we present a case of CTS caused by compression from an AEM in an adult patient. Diagnostic evaluation, surgical management, and a favorable six-month postoperative outcome are discussed.

Case Presentation

A 40-year-old right-hand-dominant male presented to the outpatient clinic with numbness in the fingers of the right hand accompanied by ulnar-sided elbow pain. The patient described paresthesia originating at the elbow and radiating along the ulnar aspect of the forearm to the fourth and fifth digits. Symptoms had gradually worsened over a three-month period. He denied any history of trauma. His medical and surgical history were unremarkable, and he reported intermittent use of paracetamol for pain relief.

On physical examination, inspection revealed no abnormalities. Vascular examination of both upper limbs was normal. Neurological assessment demonstrated preserved motor strength, grip, and pinch function. Sensation in the elbow, forearm, and hand was intact. Froment's and Wartenberg's signs were negative.

Palpation over the cubital tunnel elicited tenderness, and Tinel's sign was positive. Provocative testing reproduced symptoms after maintaining elbow hyperflexion for 60 seconds. Carpal tunnel syndrome was excluded clinically.

Plain anteroposterior and lateral radiographs of the elbow were unremarkable. Electromyography revealed deduced motor unit recruitment, positive sharp waves in the intrinsic hand muscles, and severe focal slowing of conduction velocity across the elbow. Magnetic resonance imaging (MRI) of the right elbow demonstrated edema at the medial aspect of the elbow, with increased signal intensity and compression of the ulnar nerve caused by an accessory AEM, as indicated by the white arrows in Figures *1*, *2*.

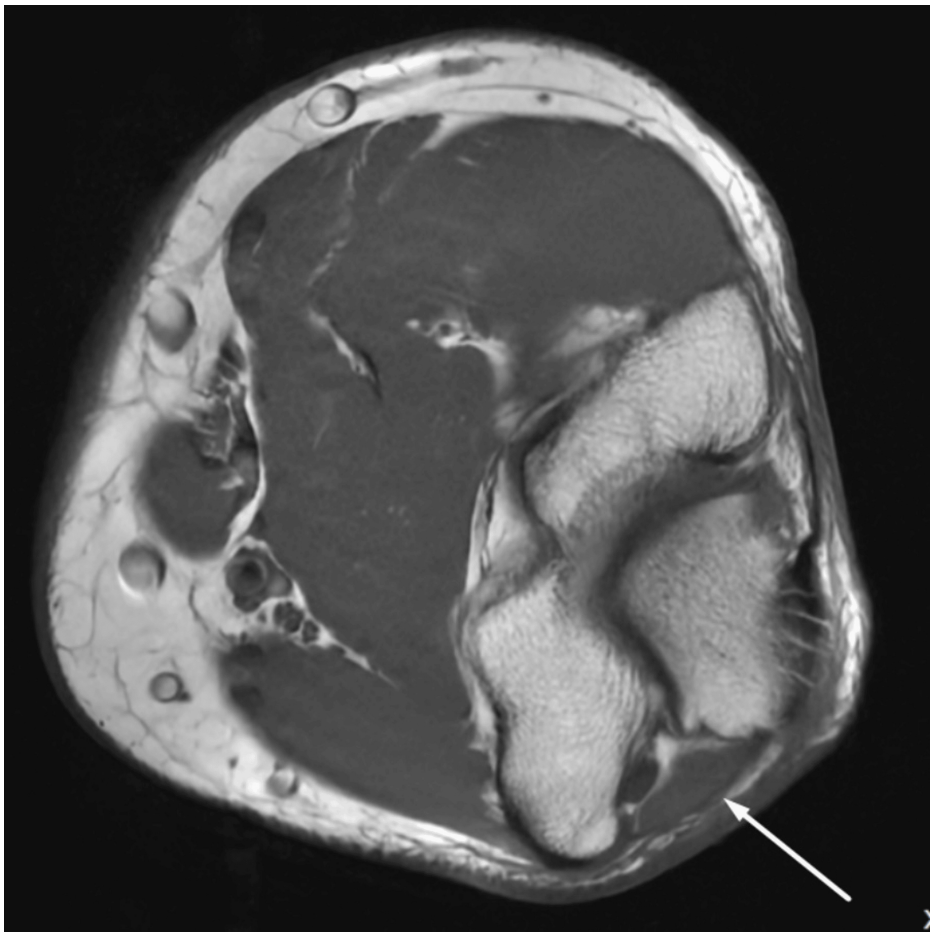


FIGURE 1: MRI axial plane slice revealing the presence of AEM.

White arrow points to AEM belly.

AEM: anconeus epitrochlearis muscle.



FIGURE 2: MRI sagittal plane slice revealing the presence of AEM.

White arrow points to AEM belly in sagittal plane.

MRI: magnetic resonance imaging; AEM: anconeus epitrochlearis muscle.

Given the severity and persistence of symptoms despite physiotherapy, surgical decompression was indicated. With the patient in the supine position under general anesthesia and tourniquet control, a curvilinear posterior elbow incision approximately 5 cm in length was made. Dissection proceeded through the subcutaneous tissue to the fascia, where the AEM was identified, as indicated by the black arrow in Figure *3*. The muscle was dissected, and the ulnar nerve was released from beneath its belly (Figure *4*). Macroscopic signs of nerve compression were evident. Proximal exploration revealed no additional sites of compression, as indicated by the black arrow in Figure *5*. Dynamic assessment during elbow flexion and extension confirmed the absence of ulnar nerve subluxation. The wound was closed with nylon sutures, and a soft dressing with arm sling support was applied. Perioperatively, the patient received intravenous ceforanide.



FIGURE 3: Intraoperative picture showing the AEM belly and its course.

The black arrow points to the AEM belly as showed over the instrument.

AEM: anconeus epitrochlearis muscle.



FIGURE 4: Intraoperative picture showing dissection of the muscle belly.

The ulnar nerve laid underneath. Extreme care was taken in this step to avoid traumatizing the nerve.



FIGURE 5: Intraoperative picture showing the ulnar nerve released.

The black arrow points to the ulnar nerve, as shown over the instrument.

The patient reported immediate postoperative relief of numbness and paresthesia. He was discharged the following day after wound inspection. No oral antibiotics were prescribed. At the 14-day follow-up, the wound was well healed, sutures were removed, and the patient reported complete symptom resolution. At six months, he remained asymptomatic and satisfied with the outcome.

Discussion

The AEM, first described by Gruber in 1867, originates from the medial epicondyle of the humerus and inserts onto the medial olecranon, crossing superficially over the ulnar nerve. Considered an atavistic structure, it is commonly observed in amphibians, reptiles, and mammals. The muscle is innervated by the ulnar nerve and is thought to assist in elbow extension, adduction, and forearm supination, while potentially offering protection to the ulnar nerve.

The AEM has been described under various names, including epitrochleoanconeus, subanconeus, and accessory anconeus. Most authors agree that when present, it replaces Osborne's ligament. Although typically single-headed, rare multi-headed variants have been described [2]. The muscle frequently extends from the medial olecranon to the medial epicondyle and may be associated with hypertrophy of the medial head of the triceps, which can exacerbate ulnar nerve compression during elbow flexion [1,5]. This association was not observed in this case.

Ulnar nerve compression is commonly encountered in clinical practice and significantly impacts quality of life. While the elbow is the most frequent site of compression, anatomical variations, such as the presence of an AEM, must be considered in atypical or refractory cases [6-8]. Conversely, some authors suggest that replacement of Osborne’s ligament with muscular tissue may reduce compressive forces on the nerve, offering a protective effect [6].

CTS appears to affect both limbs equally [9,10], with bilateral involvement reported in approximately 12.8% of cases. Dellon’s cadaveric study of 64 specimens identified the AEM in 11% of elbows and noted an association with a prominent medial triceps head in 24% of cases, which may contribute to ulnar neuritis during elbow flexion [11].

Ultrasonography is a safe, noninvasive modality for evaluating the ulnar nerve and its anatomical variants. High-resolution sonography allows dynamic assessment of nerve morphology, position, and tension during elbow movement [12]. MRI remains the most effective imaging modality for identifying the AEM, typically visualized as an oval or ellipsoid structure extending between the medial epicondyle and olecranon. In symptomatic patients, MRI may demonstrate muscle edema and signal changes within the ulnar nerve [13].

Several studies report excellent outcomes following surgical decompression. Wachsmuth and Wilhelm reported symptom resolution in 16 patients after myotomy and anterior ulnar nerve transposition [14]. Hirasawa et al. described a weightlifter with bilateral AEM hypertrophy who achieved complete symptom resolution following bilateral myotomy and unilateral transposition [15]. Other authors have reported favorable outcomes with muscle division alone, without routine nerve transposition [7,9,16,17]. Masear et al. described a ligamentous variant of the AEM and identified it in 5 of 42 surgical explorations for ulnar nerve compression [10].

Conclusions

When evaluating a patient with ulnar neuropathy at the elbow (CTS), the clinician should recognize that an AEM is an uncommon but documented cause of compression. In most cases, CTS is due to more typical mechanisms (e.g., compression under Osborne's ligament, between the (FCU) heads, or dynamic subluxation); AEM should be considered when the presentation is atypical or when common causes have been excluded. When clinical and electrodiagnostic findings support ulnar neuropathy at the elbow but the etiology remains unclear, MRI of the elbow is the imaging modality of choice to identify structural causes, including accessory muscles such as AEM, ganglia, osteophytes, or dynamic instability. MRI can demonstrate an anomalous muscle belly superficial to the cubital tunnel and clarify its relationship to the ulnar nerve. For patients with persistent or severe CTS, especially with motor deficit or failure of conservative care, surgical decompression of the ulnar nerve is a reliable treatment to relieve pressure and improve symptoms and function.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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