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Case Report

Dynamic Compression of the Ulnar Nerve Associated With the Anconeus Epitrochlearis Muscle: Do We Really Know Everything?

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Ulnar nerve compression associated with the anconeus epitrochlearis muscle (AE) is an uncommon cause of peripheral nerve compression at the elbow. It is often seen in young women with a hypertrophied or severely edematous muscle. Its causes are unclear. Numerous observed features, such as a hypertrophic AE, a palpable mass on the medial side of the elbow, and the dynamic nature of symptoms, have sparked controversy in the literature. Its clinical presentation is often insidious, and occasionally symptoms only occur in prolonged positions (dynamic compression). EMG tests are usually negative, and a correct diagnosis relies on imaging. We present the case of a 21-year-old student and clarinet player who presented with dynamic compression of the ulnar nerve at the elbow associated with AE. Much remains to be elucidated about the incidence, pathophysiology, and contributing factors of this peripheral form of cubital compression. It may be time to revisit this condition.

Ulnar nerve compression at the elbow is the most common nerve compression syndrome in the upper limb after carpal tunnel syndrome. It has numerous causes. The ulnar nerve passes through a narrow channel at the level of the distal humerus in the epitrochlea, where it can be compressed by several pathologic factors. Compression can also occur proximally, in the arcade of Struthers, or distally, in the fascia of the flexor-pronator muscles in the forearm. The most common anatomic cause of ulnar nerve compression in the cubital tunnel is perhaps the anconeus epitrochlearis muscle (AE), an anomalous accessory muscle with a trapezoidal shape (Figs. 1, 2) that is inserted in the epitrochlea and contributes to the roof of the cubital tunnel in its passage above the ulnar nerve. In normal circumstances, ulnar nerve entrapment at the elbow is diagnosed by clinical examination, provocative tests,[1] and EMG. In exceptional cases, ultrasound and magnetic resonance imaging are performed to rule out compression.[2] The diagnosis of nerve compression due to AE is frequently clinical and EMG findings tend to be negative. Ulnar nerve entrapment is normally treated by surgical decompression involving the release of the nerve in the epitrochlear canal and other areas of compression. The need for submuscular and subcutaneous nerve transposition varies.[3]

According to the literature, ulnar nerve compression associated with AE is commonly observed in young women with a hypertrophied or severely edematous muscle.[4] However, cases are too few for any conclusions to be drawn. The estimated prevalence of AE in healthy individuals lies between 1% and 34%.[5,6] The muscle has an average length of 18 mm and a volume of approximately 882 mm^3.[7] The most common symptoms of ulnar nerve compression associated with AE are numbness (25% of cases) and tingling (17% of cases).[8] Patients with a hypertrophied nerve often have a palpable mass in the cubital tunnel area. However, the above symptoms can have other causes. Maslow et al[9] found no significant differences in medial elbow pain, a positive Tinel sign, or EMG findings when comparing cubital tunnel syndrome in patients with and without an AE. On the basis of the few case reports and small series in the literature, cubital tunnel syndrome attributable to AE appears to be rare, although its incidence is unknown. Dynamic compression of the ulnar nerve is even less common, with the largest series describing just 4 cases involving 2 baseball pitchers, a welder, and a vocal artist.[10] We report the case of a 21-year-old student and clarinet player who presented with dynamic compression of the

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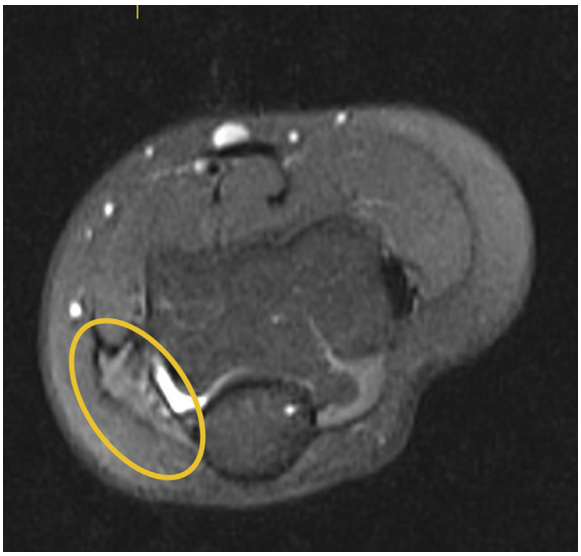

Figure 1. Magnetic resonance image showing the AE muscle inside the yellow circle.

ulnar nerve at the elbow associated with AE. Informed consent was obtained from the patient for the publication of case details and images.

Case Report

A 21-year-old woman with an unremarkable history presented with pain in both hands, although it was much more intense in her dominant, right hand. She mentioned experiencing cramping and twitching in her little finger and, to a lesser extent, her ring finger when writing for long periods or playing the clarinet. She often had to modify her posture to keep playing and sometimes had to stop. The symptoms subsided on the interruption of long periods of writing or clarinet playing and did not interfere with sleep. Prior to her visit, she had received physiotherapy for several months and performed posture exercises recommended by professional music teachers. There was no evidence of muscle atrophy or other hand or forearm alterations during the physical examination. The Tinel sign was positive in the right cubital tunnel, and there was a slightly palpable mass in the same area. The results of the provocative test at the elbow were unclear, neck and shoulder disorders were ruled out on physical examination, and the Tinel and Phalen signs were negative for Guyon canal syndrome. There was no subluxation or instability of the ulnar nerve on physical examination.

The EMG findings were normal. Magnetic resonance images confirmed the presence of an AE and showed doubtful signs of an enlarged ulnar nerve in its passage through the cubital tunnel (Fig. 1).

Three months after the diagnosis, the patient underwent surgical release of the cubital tunnel with resection of the AE. The AE was small, trapezoidal in shape, and had one base in the medial epicondyle of the humerus and another in the ulna (Fig. 2). It was located in the area of the compressed underlying nerve, which appeared normal. Nerve transposition was not performed because the nerve was stable during elbow mobilization, the cubital tunnel had a normal appearance, and the patient did not have a history of nerve instability. The nerve was also released from the arcade of Struthers and the fascia of the flexor carpi ulnaris muscle. At the 6-week follow-up visit, the patient was completely asymptomatic and had achieved full clinical and functional recovery.

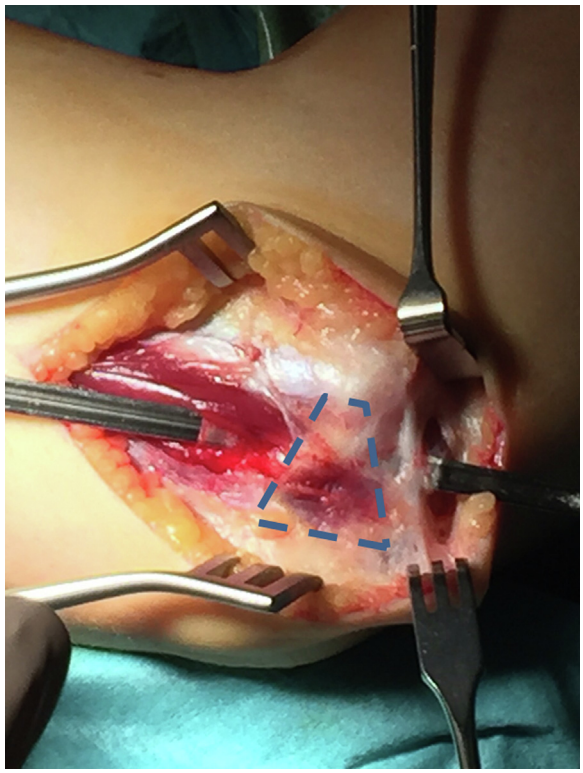

Figure 2. Intraoperative image showing the AE muscle. The dotted line shows the margin of the muscle. The grooved probe shows the cubital tunnel at the level of the epitrochlea; the probe passed through without restriction, confirming that the space was not narrow.

Discussion

We have reported the case of a patient with an AE that is possibly responsible for dynamic cubital tunnel syndrome during prolonged flexion of the elbow (writing and playing the clarinet). The causes of ulnar nerve compression in these types of patients remain unclear. Numerous features observed in our patient, such as the slightly hypotrophic AE, palpable mass, and dynamic nature of symptoms, are sources of controversy in the literature.

Pechan et al[11] reported that elbow flexion exerts greater pressure on the ulnar nerve in its passage through the cubital tunnel than either shoulder abduction or wrist extension. Although the clarinet is not a heavy instrument, prolonged playing can cause muscle tension[12] and, with time, cubital tunnel syndrome in predisposed patients. The main muscles used by clarinet players to keep the instrument stable are the abductor pollicis longus, extensor pollicis longus, and elbow flexors. Depending on the arm (left or right) and time spent playing, elbow flexion (mean angle, range 95°–105°) and internal shoulder rotation (mean angle, range 34°–44°) could increase the pressure at the cubital tunnel.

The coexistence of the AE and Osborne ligament is also a source of debate. ÓDriscoll et al[13] clearly showed that most patients with cubital tunnel syndrome have a cubital retinaculum (Osborne ligament) that retains the nerve within the tunnel. However, they also reported cases in which this ligament was absent or had been replaced by an AE. Their findings suggest that the 2 structures have a common origin and that the ligament is an atrophic form of the AE. Fernandez et al,[14] in a series of patients with ulnar nerve entrapment treated with surgical division of the AE and nerve decompression, in turn confirmed the absence of residual

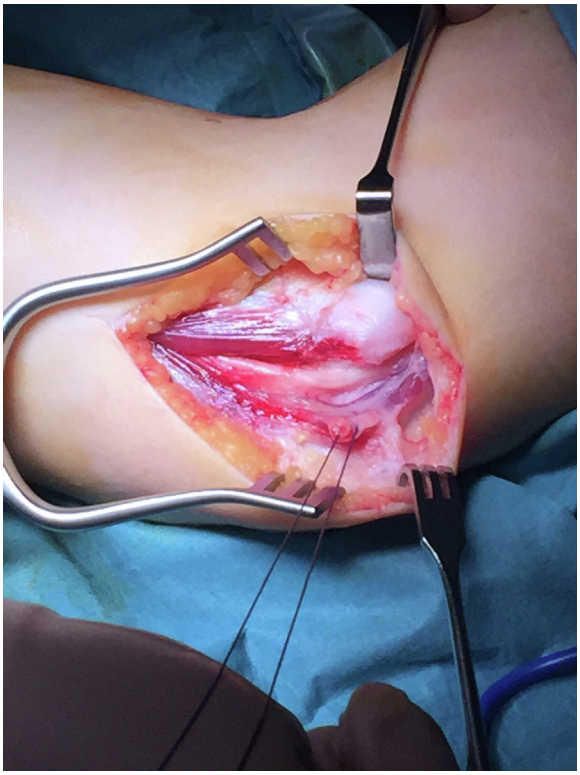

Figure 3. Intraoperative image showing the resection margins of the muscle. The muscle attachments indicate a hypotrophic muscle.

compression by opening the Osborne ligament, showing that the 2 structures can coexist.

The role of AE in ulnar nerve compression, if any, is unclear. According to Wilson et al,[4] the few cases of compression attributed to AE in the literature were due to a hypertrophied muscle, possibly explaining why this condition is more likely to affect the dominant arm. However, not all authors agree.[9] A recent meta-analysis showed that AE was rare in patients with cubital tunnel syndrome and was 3 times more common in healthy individuals.[8] The authors suggested that this muscle might protect against cubital tunnel syndrome, as the presence of muscle tissue rather than a ligament would make the entrance to the tunnel less rigid, facilitating the passage of the nerve. However, the AE tended more toward hypotrophy in our case, as indicated by the muscle attachments (Fig. 3).

The current case presents several interesting features. Of particular note is the dynamic nature of the compressive symptoms, which only appeared after prolonged periods of writing or clarinet playing. This dynamic presentation is supported by the normal EMG findings and the absence of visible nerve alterations during surgery.[15] The occurrence in a clarinet player who spends many hours practicing in the same position suggests that the condition may have been caused by a hypertrophied AE, but the insertions observed in the resection area indicated a small muscle. The presentation of ulnar nerve compression affecting the dominant arm of a young woman is also consistent with previous cases linked to AE. However, not all studies show a predilection for the female sex or the dominant arm.[7,8,14,15] Dynamic symptoms are a rare clinical presentation and have mostly been described in athletes.[10] The cases reported to date are anecdotal, and any conclusions would be premature. As in most other cases in the literature, we did not transpose the nerve,[8] although some authors believe a transposition is an effective approach.[15] In our opinion, if the presence of an AE forming the roof of the cubital tunnel is the cause of nerve compression, then the release of the nerve and confirmation of its stability within the tunnel are sufficient reasons for not performing this procedure.

In summary, dynamic ulnar nerve compression at the elbow due to AE is an uncommon, little-known disorder, with much remaining to be elucidated about its incidence, pathophysiologic mechanisms, and contributing factors. Recent findings call into question commonly accepted assumptions, as they show that AE is common in healthy individuals and even suggest a protective role. Based on current knowledge, it would be imprudent to assume that AE is a direct cause of ulnar nerve compression, although symptoms improve following resection. More clinical studies, including anatomic and imaging studies, are needed to explore the association between AE and ulnar nerve compression. Aspects such as AE incidence and the pathophysiologic role of this muscle in ulnar nerve compression and its link to symptoms must be clarified before attributing it to a causative role in ulnar nerve compression. It would be imprudent to draw conclusions based on this case involving dynamic compression of the ulnar nerve associated with AE, given the few cases in the literature.

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