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Acute ulnar nerve compressive neuropathy at the elbow caused by an accompanying aberrant vessel: A case report

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SUMMARY

Cubital tunnel syndrome caused by a vessel anomaly is extremely rare. In our case, a patient complained of acute ulnar nerve neuropathy immediately following an operative mass excision at the contralateral elbow. There were no pathologic findings other than twisting of the accompanying vessel around the ulnar nerve. Symptoms were completely relieved following abnormal vessel ligation and ulnar nerve transposition. Hence, we conclude that aberrant vessel paths can cause acute ulnar nerve neuropathy around the elbow.

Key words: Cubital tunnel syndrome – Ulnar nerve – Accompanying vessel – Compressive neuropathy

INTRODUCTION

Cubital tunnel syndrome is the second most common compressive neuropathy of the upper extremities (Palmer and Hughes, 2010). Patients usually present with paresthesia in ulnar nerve distribution and weakness of the intrinsic muscles. In addition, patients can complain of pain, but this is usually not their predominant symptom (O'Driscoll et al., 1991). While, ulnar nerve entrapment usually occurs around the elbow, other common sites include between the two flexor carpi ulnaris heads, the Osborne's ligament, the arcade of Struthers, the deep flexor pronator aponeurosis, the medial intermuscular septum, and the medial epicondyle (Assmus et al., 2011). Secondary cubital tunnel syndrome is most often caused by fractures, osteoarthritis, and soft tissue lesions such as lipomas, and/or ganglions (Palmer and Hughes, 2010; Assmus et al., 2011). Only a few studies have reported ulnar nerve compressive neuropathy caused by accompanying vessels. Here, we report an unusual surgical outcome of cubital tunnel syndrome due to twisting of accompanying vessels.

CASE REPORT

A 19-year-old male patient was initially referred to the corresponding author's hospital for treatment of a painful palpable mass at the left elbow. The patient was military personnel with an unremarkable medical history. Plain radiographs of both elbows showed no specific abnormalities. However, contrast-enhanced magnetic resonance imaging (MRI) revealed a 0.9 × 1.0 cm2-sized T2

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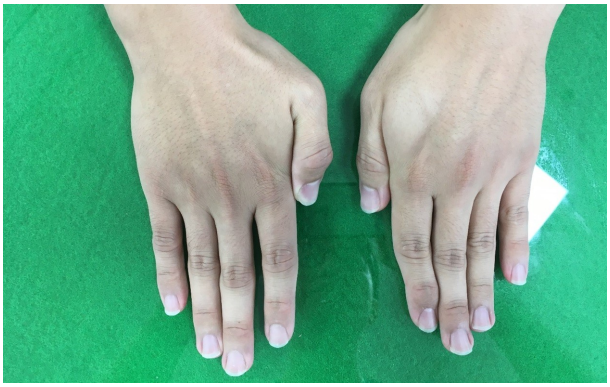

Fig 1. Atrophy of the first dorsal interosseous muscle (affected hand) is shown.

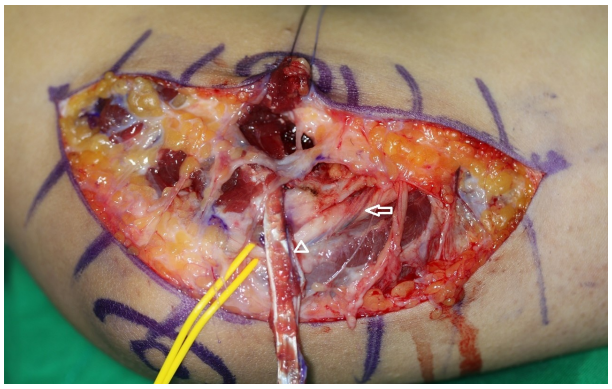

Fig 3. Operative finding: accompanying vessels twisting (arrow with tail) around the ulnar nerve (arrow without tail) was identified approximately 2 cm above the medial epicondyle.

high-signal-intensity lesion with rim enhancement in the subcutaneous layer at the radial aspect of his left elbow. Under wide-awake surgery with local anesthesia, we performed complete excision without a tourniquet. The specimen was a pilomatricoma, a hair matrix-derived benign skin tumor. The surgery took 12 minutes. The patient complained of contralateral hand paresthesia (right side) at postoperative day 1. His right hand paresthesia and grip power weakness gradually aggravated, so the patient was referred to a rehabilitation medicine specialist, who performed electrical diagnostic evaluation on postoperative week 3. We decided to observe the abrupt onset of ulnar nerve symptoms for an additional 3 weeks, and prescribed an oral steroid. Nevertheless, the symptoms exacerbated, with atrophy of the first dorsal interosseous muscle and weakness of finger adduction and abduction (Fig. 1). In addition, a follow-up electrical diagnostic test concluded that nerve conduction velocity and electrical activity had worsened compared with the previous test. The compression site was revealed to be 2 cm above from the medial epicondyle. An MRI showed a swollen and thickened ulnar nerve with increased signal intensity, which was suggestive of a neuritis (Fig. 2). We decided that a surgical intervention was needed, and the patient provided written consent. We planned to perform an anterior transmuscular transposition of the ulnar nerve, a surgical technique described by Mackinnon (Lowe et al., 2001). Further, accompanying vessel twisting around the ulnar nerve was identified approximately 2 cm above the medial epicondyle (Fig. 3). The aberrant accompanying vessel was ligated and resected in 4-cm lengths, and the ulnar nerve was transposed anteriorly. The patient's symptoms improved at postoperative week 5. Follow-up electrical diagnostic evaluation at postoperative week 6 detected partial recovery compared with preoperative results. Finally, interosseous muscle atrophy, grip power weakness, and finger motion were completely restored at postoperative month 14 (Fig. 4). Informed consent was obtained from the patient for the purpose of this case report.

COMMENTS

A comprehensive diagnosis of compressive neuropathy can be achieved using a combination of

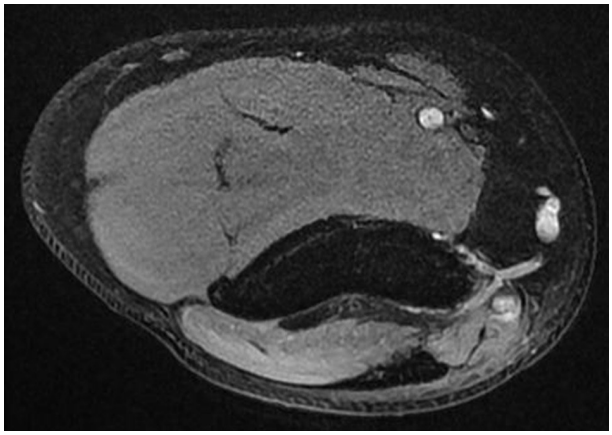

Fig 2. MRI finding: at 2 cm above the medial epicondyle, the ulnar nerve was swollen and thickened with increased signal intensity.

Fig 4. Interosseous muscle atrophy and grip power weakness were completely recovered at postoperative month 14.

clinical assessments, electrophysiological examinations, and imaging studies (Palmer and Hughes, 2010). In the present case, the patient presented with rapidly progressing weakness of his contralateral hand's intrinsic muscles and paresthesia of his ring and small fingers. The ulnar nerve exhibited swelling and thickness, as depicted by increased MRI signal intensity. An electrical diagnostic test indicated that the potential compression site of the patient's ulnar nerve was 2 cm above the medial epicondyle.

Compression at common sites was not evident during surgery, and abnormalities such as swelling were not observed in the ulnar nerve. The only pathological finding was the accompanying vessel at the same site. After ligation of the accompanying vessel and anterior transposition of the ulnar nerve in a trans-muscular fashion, intrinsic muscle weakness was fully resolved at the time of the last follow-up. All of these findings suggest that the aberrant accompanying vessel was the cause of ulnar neuropathy. In addition, the observed increase in the MRI signal intensity was possibly due to the accompanying vessel.

Several studies have addressed the anatomy of the ulnar nerve blood supply (Prevel et al., 1993). The ulnar nerve at the elbow is supplied by three arteries, namely, the superior ulnar collateral artery, the inferior ulnar collateral artery, and the posterior ulnar recurrent artery. These arteries pass through the ulnar nerve posterolaterally, and anastomosis of the terminal branches at the surface of the ulnar nerve forms the epineurium vessel (Li et al., 2015). Twisting of the accompanying vessel around the ulnar nerve points towards vessel malformation, whereby the associated symptoms worsened after contralateral elbow surgery. A blood pressure cuff and an intravenous catheter were placed on the patient's right upper arm during the first (left) elbow surgery. The cuff and IV catheter may have affected the venous flow or created a thrombus, which possibly aggravated the malformation of the accompanying vessel.

Treatment strategy in this case could be controversial. However, the patient's symptoms, including intrinsic muscle weakness, worsened rapidly. Treatment with oral steroids for 3 weeks did not result in any significant improvement in the patient's condition. Considering the severity of the symptoms and the failure of conservative management, it was our opinion that surgery was inevitable. Following second surgery, muscle weakness disappeared and sensation was fully restored. Blonna et al. (2014) also reported excellent operative outcome for rapidly progressive neuropathy.

Peripheral neuropathy caused by vessels is very rare. Some studies have reported nerve entrapment caused by vessel anomalies such as arteriovenous malformations. However, there have been no reports showing that cubital tunnel syndrome is caused by vessel anomalies (Kim et al., 2015; Van Gompel et al., 2010). In the present case, there is insufficient evidence that the accompanying vessel was the major cause of ulnar neuropathy, although we could not identify any other pathologic finding except twisting of the aberrant vessel around the nerve. Further investigation is required to address this aspect.

Our observations suggest that vessel anomalies, such as twisting of the accompanying vessel, can result in acute onset cubital tunnel syndrome. Careful identification of abnormal anatomies during ulnar neuropathy is essential, especially for the diagnosis of acute cubital tunnel syndrome. In addition, we recommend surgery as a viable option for the treatment of acute cubital tunnel syndrome.

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