Complete Anaesthesia in the Cutaneous Distribution of the Ulnar Nerve following Submuscular Anterior Transposition

A Case Report

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Abstract

Complete anaesthesia in the cutaneous distribution of the ulnar nerve following submuscular anterior transposition of the ulnar nerve has not been previously reported. We postulate vascular insult as the etiology of this condition and suggest there may be clinical importance to preserving the ulnar nerve blood supply during submuscular anterior transposition.

Compression of the ulnar nerve at the cubital tunnel is common. Among upper extremity compressive neuropathies, it is second in incidence to carpal tunnel syndrome. Surgical options range from in situ decompression to medial epicondylectomy to various anterior transposition techniques. Proponents advocate submuscular transposition as the definitive procedure for ulnar nerve compression, as well as the procedure of choice for previously failed cubital tunnel surgery. We present a case of complete anesthesia in the cutaneous distribution of the ulnar nerve following submuscular anterior transposition. There has been no such previous report in the English literature.

Case Report

A 48-year-old, right-hand dominant female presented with a history of paraesthesia and numbness of 2 years duration that involved all of the right hand digits, as well as pain on the medial aspect of her right forearm. Physical examination elicited a positive Tinel over the cubital tunnel; elbow flexion resulted in increased paraesthesia of the ring and little digits. The patient had signs of carpal tunnel syndrome, a positive Tinel and Phalen’s maneuver, as well as a positive Durkin’s compression test. Sensation was diminished in the distribution of the right median nerve but was normal in the ulnar one-and-a-half digits (two-point discrimination, 5 mm). There was no muscle atrophy. Abductor pollicis and first dorsal interosseous muscle strength was 5/5. Electromyographic (EMG) and nerve conduction studies showed right-sided mild to moderate carpal tunnel syndrome and mild to moderate ulnar nerve neuropathy on the elbow.

Following unsuccessful nonoperative treatment of therapy and splinting, the patient underwent an uneventful submuscular anterior right ulnar nerve transposition and carpal tunnel release. The ulnar nerve was gently retracted and not subjected to traction. There was no residual compression or kinking of the ulnar nerve post transposition. Immediately postoperatively, the patient complained of complete anaesthesia of the medial one-and-a-half digits. Two-point discrimination could not be performed. There was no evidence for external compression or hematoma, which would have necessitated evacuation. Based on the operative procedure, we believe that there was no kinking of the nerve causing compression. Four weeks postoperatively, the examination showed clawing of the ring and little digits, with 3/5 weakness of the ulnar intrinsic muscles of the right hand and 4/5 weakness of the right flexor carpi ulnaris. On sensory evaluation, there was complete anaesthesia of the medial aspect of the right hand, involving both palmar and dorsal aspects. At this stage, the patient was offered a reexploration to definitively rule out a mechanical cause for compression. The patient refused further surgery.

Two months postoperatively, electromyelography (EMG)

yielded a significant amount of active denervation involving the first dorsal interosseus and flexor carpi ulnaris muscles. There was electrophysiological evidence of a moderately severe right ulnar neuropathy. At 15-month follow-up, the patient’s condition had improved, but she continued to report decreased sensation in the ulnar nerve distribution. Her two-point discrimination at last follow-up was 7 mm. The first dorsal interosseous and the remaining ulnar intrinsics had a muscle strength of 5/5. The clawing had resolved and the point of maximal Tinel had progressed from the proximal forearm to just distal to the distal wrist crease.

Discussion
One of the advantages cited by advocates of anterior transposition of the ulnar nerve at the cubital tunnel is that the nerve, in its new position, is less subject to stretch on elbow flexion. From a mechanical point of view, this is thought to reduce possible stretch-induced ischemia of the nerve. However, surgical mobilization of the nerve during anterior transposition, iatrogenically impairs the blood supply to the nerve and places it at risk for ischemic necrosis. On a microscopic level, the ulnar nerve has both an extrinsic and intrinsic blood supply. The extrinsic vascular system is composed of epineural segmentally arranged vessels that originate at various points from the superior ulnar collateral, inferior ulnar collateral, and posterior ulnar recurrent arteries. The inferior ulnar collateral artery provides direct vascularization to the nerve, with no anastomosis in the region just proximal to the cubital tunnel. The intrinsic supply is comprised of perineural and endoneural plexuses with stepladder communicators, resulting in a rich network of vessels. Sunderland and Lundborg, have shown that it is the intrinsic supply that is critical to the delivery of adequate oxygen supply to the individual nerve fascicles and axons.

When the elbow is flexed, the ulnar nerve is stretched and narrowed, thereby constricting the vessel lumens of the microcirculation. Clinical dysfunction can result from a transient, slowed blood flow through the narrowed lumens of the stretched vessels of the stretched nerve. Microscopically, there will be reduced oxygenation to those nerve fascicles supplied by the affected vessels. Anterior transposition significantly decreases the potential for nerve compression/tension during elbow flexion; however, the procedure itself may devascularize the nerve by interfering with the intra-neural circulation namely, elimination of existing multiple mesentery-like vessels, especially those originating from the inferior ulnar collateral artery.

To date, there has been no report in the literature documenting complete anaesthesia in the cutaneous distribution of the ulnar nerve, following its submuscular anterior transposition. This is probably related to the observation that even if it is necessary to sacrifice the inferior ulnar collateral vessel to allow anterior transposition, this has not been shown to result in either permanent ischemia or functional compromise. Ogata and colleagues, in a study of primates, showed transient loss of blood supply for 72 hours after anterior transposition. It is postulated that the rich, intrinsic anastomotic microcirculation protects the nerve’s functioning.

In summation, risk of ischemic necrosis to the ulnar nerve is inherent to the technique of anterior transposition, should a segment of the nerve be completely separated from its nourishing blood supply. It is our postulation that this was the precipitating event in our patient. We, therefore, conclude there may be clinical importance in maintaining the extrinsic blood supply during the procedure of anterior transposition.

Disclosure Statement
None of the authors have a financial or proprietary interest in the subject matter or materials discussed, including, but not limited to, employment, consultancies, stock ownership, honoraria, and paid expert testimony.

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