Bilateral anomalous course of the ulnar nerve at the wrist causing ulnar and median nerve compression syndrome

Case report

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The case of a patient with a bilateral compression syndrome of the ulnar and median nerves at the wrist is described. Both ulnar nerves, which were surgically explored at different times, followed an anomalous course and passed into the canalis carpi side by side with the median nerve. This variation in the course of the ulnar nerve is extremely rare and causes a unique syndrome with characteristic electromyographic patterns.

KEY WORDS • ulnar nerve • median nerve • carpal tunnel syndrome • nerve compression

Variations in the anatomical position of the distal portion of the ulnar nerve are rare. When they occur they are usually anomalies in the origin or in the course of the distal branches of the nerve, whereas the location of the main trunk is preserved within the canal of Guyon, where it is normally found with the ulnar artery.1,2,4-6 The authors report the case of a patient with an anomalous course of both ulnar nerves, which passed into the carpal tunnel together with the median nerve. Only one case of a similar anomaly has been described previously,3 but in that case the abnormality was unilateral.

Case Report

This 43-year-old woman was admitted to the Division of Neurosurgery of our hospital in November, 1983, with complaints of paresthesias, hypesthesia, and numbness in her right hand of about 7 years' duration. Initially these symptoms had involved only the ulnar side of the hand and the fourth and fifth fingers, but subsequently the other fingers became involved also. She later experienced a progressive loss of power in the whole hand, especially when grasping. She had developed marked atrophy of the intrinsic ulnar muscles of the hand, with a forced claw-like position of the fourth and fifth fingers. Trophic disturbances of the end of these two fingers had appeared, with some atrophy of the intrinsic median musculature of the hand. This patient, a resident of a South American country, had been treated with sulfones because of an erroneous diagnosis of leprosy. During a holiday in Italy, she was examined in our outpatient department and hospitalized.

First Admission. Electrophysiological examination disclosed the absence of motor unity potentials, positive sharp waves and fibrillation potentials in the intrinsic ulnar muscles of the hand, and the absence of sensory action potentials from the fifth digit to the wrist, suggesting complete Wallerian degeneration of the ulnar nerve at the wrist. Sensory conduction velocity was reduced in the median nerve distal to the carpal tunnel, while motor latency from the wrist to the abductor pollicis brevis muscle was only slightly increased.

First Operation. With the patient under plexus block anesthesia, Guyon's canal was explored on November 18, 1983. Only a small ulnar artery was contained within the canal. The ulnar nerve was found unexpectedly in the canalis carpi, passing medially to the median nerve. Both nerves were enveloped in the same sheath but were distinctly separated, with each nerve having its own epineurium. Both nerves were pale, but only the ulnar nerve was clearly atrophic in the distal part of the carpal tunnel, where it divided into its superficial and deep branches. At this level the main trunk appeared compressed against the os pisiforme, while its distal branches, and especially the
Bilateral anomalous course of the ulnar nerve

FIG. 1. Artist’s drawing (left) and intraoperative photograph (right) showing the abnormally located ulnar nerve (UN) compressed against the radial surface of the hamate bone (HB) and passing medial to the median nerve (MN) into the canalis carpi.

superficial branch, were compressed against the radial surface of the hook of the hamate bone. After resection of the retinaculum flexorum and neurolysis, both the median and ulnar nerves appeared decompressed (Fig. 1).

Postoperatively, the painful nocturnal paresthesias disappeared. At follow-up examination 3 months after the operation, clinical and electrophysiological findings in the portion of the right hand innervated by the median nerve were normal. Hypoesthesia and muscle atrophy from the ulnar nerve lesion were unchanged. Electrophysiological examination confirmed complete degeneration of the ulnar nerve at the wrist; however, the trophic disturbances in the fourth and fifth digits had disappeared.

During this first hospitalization the patient denied symptomatology in her contralateral (left) hand and clinical examination was negative, so no electrophysiological study was performed on the left side.

Second Admission. In June, 1984, 18 months after the first operation, the patient was hospitalized again because of nocturnal pain, paresthesias, hypoesthesia, and numbness in her left hand. Examination revealed a mild hypothenar atrophy, with weakness of thumb opposition. There was hypoesthesia of the whole hand, more pronounced on the volar aspect of the fourth and fifth fingers. A soft swelling was present over the retinaculum flexorum. Percussion over the lateral side of the carpal tunnel gave rise to a positive Tinel’s sign of the distal median nerve. While percussion over Guyon’s canal was negative, percussion over the medial side of the canalis carpi evoked a positive Tinel’s sign from the distal ulnar nerve.

Nerve conduction studies were peculiar. Stimulation of the ulnar nerve at the wrist along its normal course in Guyon’s canal evoked only a volume-conduction response in the first interosseus dorsalis muscle and in the abductor muscle of the fifth digit. Recording of the orthodromic sensory responses of the ulnar nerve at the wrist along its normal course was silent. However, electrical stimulation over the median side of the carpal tunnel resulted in the recording of morphologically normal muscle action potentials, with mildly prolonged latency, in the abductor muscle of the fifth digit.

When sensory conduction velocity was recorded from the ulnar nerve with the recording electrode placed over the median side of the carpal tunnel, a markedly prolonged latency was demonstrated. Slightly decreased sensory conduction velocity and normal motor conduction velocity were disclosed in the left median nerve on recording over its normal location in the canalis carpi.

Second Operation. The patient underwent a second operation on June 29, 1984. At surgery, the same anomaly that had been found on the right was found in the course of the left ulnar nerve, which passed into the canalis carpi medially to the median nerve. After decompression, both nerves appeared congested; no hypertrophy was present in any segment of these nerves.

The patient's postoperative course was uneventful, with immediate remission of the paresthesias and pain.
and rapid recovery from the hypesthesia in the left hand. Electrophysiological examination 2 months after the operation indicated normal sensory and motor conduction velocity in both the left median and left ulnar nerves.

Discussion

In rare cases the main trunk of the ulnar nerve follows an anomalous course, passing into the carpal tunnel together with the median nerve rather than into the canal of Guyon. Even though it is possible that such an anomaly may cause no symptomatology and therefore go unnoticed, the location of both the ulnar and median nerves within the narrow space available in the canalis carpi may give rise to a peculiar syndrome that exhibits characteristic clinical and electrophysiological patterns. The case reported here is particularly interesting because the anomaly was bilateral and manifested itself clinically at different times on each side, prompting considerations of the pathogenesis and evolution of the syndrome.

The symptoms of this abnormality follow a typical sequence. Initially, pain is more severe in the sensitive superficial branch of the ulnar nerve, followed by progressive compression of the deep motor branch of that nerve. Later, symptoms of median nerve compression appear, but they are never as serious as are the symptoms of ulnar nerve compression. The particular anatomical location of the ulnar nerve and especially of its superficial branch, which are compressed against the radial surface of the pisiform and hamate bones, explains this characteristic pattern of symptom progression as well as the characteristic electrophysiological recordings, as seen in this case.

Decompressive surgery is the only possible treatment once symptoms of nerve compression have appeared. As in any peripheral nerve compression syndrome, recovery is definitely related to the time between the onset of symptoms and the operation.

References

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