Carpal tunnel syndrome can be secondary in some patients, and vascular anomalies (usually a persistent median artery), median nerve variations, or both are among the etiologic factors. High division of the median nerve proximal to the carpal tunnel (known as a bifid median nerve) is a median nerve anomaly that has an incidence rate of 2.8%. This rare entity is often associated with various abnormalities that are clinically relevant, such as vascular malformations (persistent median artery), aberrant muscles, and carpal tunnel syndrome. The bifid median nerve is one cause of carpal tunnel syndrome because of its relatively higher cross-sectional area compared with a nonbifid median nerve. Obtaining magnetic resonance imaging and ultrasounds of bifid median nerves has helped surgeons avoid potential surgical hazards.

This article describes 3 men with 4 bifid median nerves associated with a persistent median artery. Mean patient age was 38 years (range, 37-40 years). Mean follow-up was 7 years (range, 3-11 years). Patients were diagnosed with carpal tunnel syndrome and underwent open carpal tunnel release. To reveal a morphological etiology in patients in whom the possibility of having idiopathic carpal tunnel syndrome is unlikely, preoperative imaging studies should be obtained. Bifid median nerves associated with a persistent median artery in the carpal tunnel are important to understand for their clinical and surgical significance. A secondary nature should be suspected in patients with unilateral symptoms, especially those with a history of symptoms and when the symptomatic hand shows severe neurophysiologic impairment but the contralateral hand is neurophysiologically intact. Inadvertent injury to the median nerve during carpal tunnel surgery can be minimized if the variations of the median nerve are recognized.
High division of the median nerve proximal to the carpal tunnel (known as a bifid median nerve) is a median nerve anomaly with an incidence rate of 2.8%. This rare entity is often associated with various clinically relevant abnormalities, such as vascular malformations (persistent median artery),\(^1,^2\) aberrant muscles,\(^3,^4\) and carpal tunnel syndrome.\(^4,^5,^9\) A detailed description of the anatomical variations of the median nerve in the carpal tunnel, including the bifid median nerve, was previously reported.\(^1\) The bifid median nerve is a cause of secondary carpal tunnel syndrome due to its relatively higher cross-sectional area compared with a nonbifid median nerve.\(^5,^6\)

Magnetic resonance imaging (MRI) and ultrasound can be obtained to show bifid median nerves, helping surgeons avoid potential surgical hazards. This article describes 4 bifid median nerves in 3 patients with carpal tunnel syndrome. All bifid median nerves were confirmed preoperatively using MRI and intraoperatively.

**Materials and Methods**

The authors’ institution does not require institutional review board approval for retrospective clinical studies. Informed consent was obtained from all patients for publication of their data.

Three men with 4 bifid median nerves were examined. Mean patient age was 38 years (range, 37-40 years). Mean follow-up was 7 years (range, 3-11 years). Patients were diagnosed with carpal tunnel syndrome and underwent open carpal tunnel release. At presentation, 2 patients reported unilateral symptoms and 1 reported bilateral symptoms of carpal tunnel syndrome. Mean symptom duration was 10 months (range, 8-13 months). No patient had an underlying disease, such as diabetes mellitus, rheumatoid arthritis, gout, or hypothyroidism, or a history of renal dialysis, pregnancy, space-occupying lesions, previous carpal tunnel release, or previous distal radius fracture. All patients worked in office jobs. All patients had a clinical diagnosis of carpal tunnel syndrome, which was confirmed with neurophysiological tests.

The clinical diagnosis of carpal tunnel syndrome was based on a history of nocturnal pain and paraesthesiae, activity-related pain, a sensory deficit in the median nerve territory, loss of hand-grip strength, atrophy of the thenar muscles, a positive Phalen’s test, and a positive Tinel’s sign.\(^11,^12\)

Neurophysiological tests included nerve conduction studies, with measurement of the distal sensory latency, distal motor latency, and sensory nerve conduction velocity from the index finger to the wrist, and needle electromyography. All tests were conducted in normal clinical settings at room temperature. All patients underwent a physical examination and neurophysiological tests of both hands. A sensory nerve distal peak latency of less than 3 ms, an abductor pollicis brevis muscle-to-wrist distal motor latency of less than 4 ms, and an index finger-to-wrist sensory nerve conduction velocity of 50 m/s were considered normal. The lowest limit of normal amplitude was 4 mV. Pathological findings on electromyography included fibrillation activity, reduced recruitment, and abnormalities in the configuration of the motor unit action potential.\(^13\)

Carpal tunnel syndrome was classified according to neurophysiological test results as mild (prolonged sensory distal latency with or without sensory nerve action potential amplitude below the lower limit of normal), moderate (abnormal median sensory latencies and prolongation of median motor distal latency), or severe (prolonged median motor and sensory distal latencies with an absence of sensory nerve action potential, low amplitude, or absent thenar compound muscle action potential, and findings compatible with axonal injury in electromyography [ie, fibrillations, reduced recruitment, and motor unit potential changes]).\(^14\)

Magnetic resonance imaging of the symptomatic hands was obtained to reveal a morphological etiology. All 3 patients underwent open carpal tunnel release.

**Results**

All patients had a history of nocturnal pain and paraesthesiae and activity-related pain. On clinical examination, a mild thenar atrophy and a sensory deficit in the median nerve territory existed in all patients. Phalen’s test and Tinel’s sign were positive on the affected sides in all patients, and hand-grip strength was significantly reduced compared with the contralateral side, except in the patient with bilateral symptoms.

Patient data and neurophysiological results are shown in the Table. The contralateral hand was neurophysiologically intact in the 2 patients with unilateral symptoms. Clinical symptoms and neurophysiological tests showed severe carpal tunnel syndrome in 1 hand, whereas the contralateral hand was healthy in the 2 patients with unilateral symptoms, which implied a secondary disease. The patient with bilateral carpal tunnel syndrome was a 40-year-old businessman, so it was unlikely he had idiopathic carpal tunnel syndrome. Magnetic resonance imaging showed bifid median nerves associated with persistent median arteries with high division proximal the carpal tunnel in all patients (Figures 1-4).

Patients underwent intravenous regional anesthesia and open carpal tunnel release through a palmar incision. Intraoperatively after carpal tunnel release, the bifid median nerves and associated median vessels were apparent (Figures 5, 6). The radial trunk was thicker than the ulnar trunk in all patients. All patients reported relief of all symptoms postoperatively, and no recurrence had last occurred at follow-up (range, 2-10 years).

**Discussion**

Although carpal tunnel syndrome is usually idiopathic, a certain etiology can
be detected in some patients. Any process that causes an increase in the pressure in the carpal tunnel, such as space-occupying lesions (eg, lipoma and ganglion), tenosynovitis due to connective tissue diseases, gouty tophus, vascular anomalies (persistent median artery with or without an aneurysm), or malunited distal radial fractures, may lead to carpal tunnel syndrome. Benign neoplasms of the nerve, such as lipofibromatous hamartoma of the nerve and schwannoma, may also cause carpal tunnel syndrome when they occur in the carpal tunnel. Likewise, a bifid median nerve occurs relatively frequently in patients with carpal tunnel syndrome. The bifid nature may facilitate compression in the carpal tunnel because of its higher cross-sectional area compared with a non-bifid median nerve.

Lanz reported various median nerve anomalies in the carpal tunnel and classified them into 4 groups: I, variation in the course of the thenar branch; II, accessory branches at the distal portion of the carpal tunnel; III, bifid median nerves; and IV, accessory branches proximal to the carpal tunnel. The incidence of bifid median nerves was 2.8%. Persistent median arteries usually accompany bifid median nerves. The 2 parts of the nerve may be equal in size, or a predominance may exist of either the radial or ulnar part. Larger radial trunks approximately equal in size to a normal median nerve and larger ulnar trunks were reported. When a pathology of the median vessels exists, the bifidity may be missed if the small ulnar trunk is surrounded by the abnormal median vessels. Accessory lumbrical muscles passing through the bifid nerve and bifid median nerves in which the radial part passes through a separate compartment of the carpal tunnel were also reported.

The persistent median artery of the forearm is an accessory artery arising from the ulnar artery at the proximal forearm, which courses alongside the median nerve to the carpal tunnel. Along with the interosseous artery, the median artery is the main route of blood supply to the hand in embryos. After the eighth week of gestation, it regresses and is replaced by the ulnar and the radial arteries. The median artery may persist into adult life as 2 dif-
Different patterns: palmar and antebrachial. In the palmar type, the artery reaches the palm of the hand; in the antebrachial type, it terminates before reaching the wrist. It may supply the superficial palmar arch or may be the main blood supply to the radial digits. In a normal nerve, the artery, when present, runs on its ulnar side. When associated with a bifid median nerve, the artery courses between the 2 nerve trunks (Figures 4, 5). Anatomical studies have reported a prevalence of the median artery in 10% to 20% of cadaveric dissections and the palmar type in 12% of cases.

In the current patients, the persistent median arteries were all palmar type. The bifid median nerve and the persistent median artery are nonindependent variables. Both variations are an embryological malformation, and their coexistence in the same carpal tunnel may be higher than previously thought. Although a persistent median artery can cause carpal tunnel syndrome, it is not necessary to ligate or resect the artery when it is patent; simple carpal tunnel release would relieve the symptoms.

Al-Qattan et al subclassified the bifid median nerve anomaly according to its associated abnormalities. According to this subclassification, bifid median nerves with persistent median vessels but no pathology of the vessels or other abnormalities are asymptomatic; only bifid median nerves with a pathology of the persistent median vessels (ie, arteriovenous malformation, aneurysm, thrombosis, or venous malformation) are symptomatic. The current authors do not share this belief because sufficient evidence exists that bifid median nerves with concomitant persistent median arteries that do not have a pathology may cause carpal tunnel syndrome.

Although imaging studies for the diagnosis of carpal tunnel syndrome are becoming increasingly popular, most surgeons condemn these expensive and time-consuming studies as unnecessary. However, some authors report that ultrasound is an accurate and useful diagnostic tool in patients with carpal tunnel syndrome, with a sensitivity of 99% and specificity of 100%, that can be used as the initial test in patients presenting with clinical symptoms of carpal tunnel syndrome because it is equivalent to neurophysiological studies and provides additional valuable anatomical information.

The current authors prefer using neurophysiological tests instead of imaging studies for the diagnosis of carpal tunnel syndrome when a diagnostic tool is necessary. However, imaging provides additional information compared with that obtained from clinical tests and neurophysiological studies. Although neurophysiological studies depict physiologic median nerve malfunctions, imaging studies depict structural abnormalities of carpal tunnel syndrome. By allowing direct visualization of the compressed median nerve and the carpal tunnel content, imaging studies can reveal the causes of secondary carpal tunnel syndrome and show anatomical variants, such as a bifid median nerve, persistent median artery, or space-occupying lesions. Bifidity may
predispose the median nerve to compression because of the relatively higher cross-sectional area of the 2 nerve bundles. The cross-sectional area threshold for carpal tunnel syndrome in patients with a bifid median nerve is also higher than usual.9,10

Although the current authors do not routinely use MRI or other imaging modalities for the diagnosis of carpal tunnel syndrome, MRI was obtained in the current patients because of their relative young age, male sex,24 and unilateral occurrence in 2 patients, which implied a secondary disease. The MRIs revealed bifid median nerves.

Carpal tunnel syndrome presents bilaterally in 59% to 87% of patients,25-27 and approximately half of patients with unilateral symptoms have positive neurophysiological test results in the asymptomatic, contralateral hand.25-27 During follow-up of these patients with unilateral symptoms, bilateral neurophysiological impairment showed that contralateral symptoms developed in most cases.25 The incidence of space-occupying lesions in unilateral carpal tunnel syndrome is also higher than that of bilateral carpal tunnel syndrome. Nakamichi and Tachibana28 reported an increased incidence of space-occupying lesions in unilateral vs bilateral carpal tunnel syndrome and concluded that a space-occupying lesion should be suspected when the condition is unilateral and the etiology is unclear. A secondary nature should be suspected in patients with unilateral symptoms, especially those with a long symptom history and when the symptomatic hand shows severe neurophysiologic impairment but the contralateral hand is neurophysiologically intact. Median nerve anomalies are a cause of secondary carpal tunnel syndrome. In patients with severe unilateral carpal tunnel syndrome, especially in the nondominant hand, physicians should consider the possible presence of a median nerve variation. Likewise, Bagatur and Zorer,23 Padua et al,22 and Bodofsky et al29 reported that bilateral median nerve impairment is the rule in patients with carpal tunnel syndrome.

CONCLUSION

Imaging studies, which are usually not obtained for carpal tunnel syndrome, should be obtained for patients in whom the possibility is unlikely of having carpal tunnel syndrome (ie, severe unilateral symptoms, young age, and male sex). Singer and Ashworth7 reported the surgical findings of 147 hands that underwent carpal tunnel release and reported 47 variations in 60 hands. They reported that the odds of observing an anatomical variation was 3.2 times greater in patients aged 40 years or younger when compared with patients older than 40 years. Median nerve variations should be considered when performing open or endoscopic carpal tunnel release to prevent iatrogenic injuries. Inadvertent injury to the median nerve during carpal tunnel surgery can be minimized if the variations are recognized. Although it has no electrophysiologic or clinical differential diagnosis, physicians should consider the possibility of median nerve variation in patients with unilateral severe carpal tunnel syndrome, especially in the nondominant hand. One should also be aware of this anomaly during wrist laceration repairs.

Many studies on the anatomical variations of the median nerve and the persistent median artery have been conducted surgically,1,21 sonographically,8,10 or with MRI.4,9 The current authors preferred MRI instead of ultrasound because MRIs display the carpal tunnel and its contents, including the median nerve, with conspicuity. Similarly, MRI facilitates the detection of anatomical variations in the tunnel.

REFERENCES


