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An assessment of Martin-Gruber anastomosis discrepancies between healthy individuals and patients with carpal tunnel syndrome in motor nerve conduction studies

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Abstract

Background: The Martin-Gruber anastomosis (MGA) represents a nerve innervation anomaly in the upper extremity, potentially leading to misinterpretation during standard nerve conduction studies (NCSs). This study aims to characterize the electrophysiological attributes of MGA in both healthy subjects and individuals diagnosed with carpal tunnel syndrome (CTS).

Methods: This case-control study involved the electrophysiological assessment of 506 forearms, segregated into two distinct groups: a CTS positive (+) case group and a CTS negative (-) control group. The evaluations were conducted over an average period of 8 months in the neurophysiology laboratory. The study encompassed 294 forearms from 147 healthy individuals without CTS and 212 forearms from 106 patients diagnosed with CTS, both clinically and electrodiagnostically.

Results: The relationship between the presence of type I MGA and the CTS (+) group was statistically significant (P = 0.002). Similarly, the relationship between the presence of type II MGA and the CTS (+) group was statistically significant (P = 0.013). On the other hand, the relationship between the presence of type III MGA and the CTS (+) group was not statistically significant (P = 0.208). Likewise, the relationship between the presence of type IV MGA and the CTS (+) group was not statistically significant (P = 0.807). The correlation between the side of type I MGA and the groups did not reach statistical significance (P = 0.770).

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The relationship between the side of type II MGA and the groups also did not attain statistical significance (P = 0.990). Similarly, the side of type III MGA and its association with the groups did not yield statistical significance (P = 0.402). Finally, the relationship between the side of type IV MGA and the groups was not statistically significant (P = 0.166).

Conclusion: The MGA represents a relatively frequent anatomical variation observed in the upper extremity. Notably, its presence demonstrated significance in the first dorsal interosseous (FDI) muscle (type II) and the abductor digiti minimi (ADM) muscle (type I) among patients with CTS. The present study emphasizes the importance of recognizing this variation during upper extremity NCSs for a correct diagnostic approach and treatment plan to avoid misdiagnosis of median-ulnar peripheral neuropathy.

Introduction

The median-ulnar anastomosis, also known as Martin-Gruber anastomosis (MGA), is a common anatomical variant that occurs usually in the middle forearm.[1] Within the spectrum of MGA types, anastomoses may manifest between branches extending to the deep flexor muscles of the fingers, directly between the median nerve and the ulnar nerve, or involving the anterior interosseous nerve and the ulnar nerve, encompassing various combinations of these patterns.[2,3] The prevalence of MGA ranges from 10% to 30.6% in anatomical studies, and from 5% to 40% in electrophysiological investigations.[1] Bilateral presence is observed in almost 10%-30% of the cases; however, MGA is seen usually on the right side in unilateral form. There are no significant differences between men and women in terms of gender distribution.[2,3]

As per the definitions of MGA subtypes, type I anastomosis features transverse fibers terminating in the hypothenar muscle, type II terminates in the first dorsal interosseous (FDI) muscle, and type III terminates in the thenar muscle. Type IV MGA involves the coexistence of two or more of these three types within the same forearm. Anastomosis consists almost exclusively of motor axons. The involvement of sensory axons is unusual; however, Santoro et al.[4] reported a case of such involvement. There exist many studies in the literature about the prevalence of MGA; however, there are few studies, such as those performed by Iyer and Fenichel[5] and Simonetti[6] that investigated the prevalence of this anomaly in patients with carpal tunnel syndrome (CTS). These studies have reported a prevalence of MGA in patients with CTS ranging from 26% to 54%.[7]

The MGA typically goes unnoticed but can manifest through altered motor or sensory amplitude distribution after injuries to the median and ulnar nerves.[5] In the presence of MGA, responses to median nerve stimulation at the wrist are diminished compared to the elbow due to axonal passage from the median nerve to the ulnar nerve.[8,9] The opposite is also true for ulnar nerve stimulation, where the presence of anastomotic fibers from the median nerve causes a smaller response at the elbow than at the wrist.[10,11] This anatomical peculiarity can sometimes be misinterpreted as an ulnar nerve block between the wrist and sub-elbow areas.[11,12] In addition, the coexistence of the MGA and CTS may cause erroneous interpretations during routine nerve conduction studies (NCSs).[6,8,11,13-15]

The objective of this study is to illustrate the occurrence of this anastomosis between the median and ulnar nerves, which is common in both healthy individuals and patients with CTS. The aim is to recognize abnormal innervation patterns in hand muscles, enabling an accurate diagnostic approach and treatment plan that avoids misdiagnosing entrapment neuropathy and/or median-ulnar peripheral neuropathy. Ultimately, the MGA holds significant clinical importance for comprehending specific injuries and compression syndromes involving the median and ulnar nerves.

Materials and Methods

Study design and participants: This study was conducted at Siirt Hayat Hospital in the Southeastern Anatolia Region of Turkey, involving a total of 253 individuals aged between 17 and 75, over a span of approximately eight months. The participant pool consisted of 506 forearms, categorized into two distinct groups: the CTS (+) case group and the CTS (-) control group. The CTS (+) group encompassed 212 forearms from 106 patients who were clinically and electrodiagnostically diagnosed with CTS, while the CTS (-) group included 294 forearms from 147 healthy individuals without CTS. This analytical-descriptive case-control study was initiated after obtaining written permission from the hospital administration, informed consent from the participants, and ethical approval from the non-interventional clinical research ethics committee of Siirt University (decision number: 03.26.2021/5721). The control group's inclusion criteria demanded normal neurological

examinations of both upper extremities, accompanied by the absence of neurological symptoms. Exclusion criteria for both groups encompassed conditions like diabetes mellitus (DM), endocrine disorders, metabolic disorders, rheumatologic disorders, and neurological issues such as peripheral neuropathy, cervical radiculopathy, plexopathy, ulnar neuropathy, and CTS. Inclusion criteria for the CTS (+) group comprised clinical and para-clinical diagnosis of CTS, fulfilling electrophysiological diagnostic criteria. Patients with conditions affecting CTS, such as hypothyroidism, uremia, DM, trauma, peripheral neuropathy, hypercholesterolemia, and vasculopathy, were excluded. Both groups were evaluated electrophysiologically for MGA with electrodiagnostic tests whose working principles were stated below. A detailed neurological examination was performed for all studied subjects, and the data were gathered based on group allocation.

Electrophysiological studies: Electrodiagnostic studies included electromyography (EMG) and NCS. A two-channel evoked potential/EMG measurement system (EMG measuring system MEB-9102K, Nihon Kohden Corp., Tokyo, Japan) was utilized to assess all participants. Electrodiagnostic studies for CTS were conducted following established standardized techniques outlined by the American Association for Neuromuscular and Electrodiagnostic Medicine (AANEM) summary statement.[16] These studies exhibit a sensitivity of 56% to 85% and a specificity of 94% to 99% for diagnosing CTS.[17] For suspected CTS cases, evaluations included median sensory or mixed NCSs, median motor NCSs, needle EMG of abductor pollicis brevis (APB), ulnar and/or radial motor and sensory NCSs (to rule out peripheral neuropathy), and needle EMG of muscles innervated by C5-T1 roots (to exclude cervical radiculopathy, brachial plexopathy, and proximal median neuropathy).[18] A complete NCS protocol was applied for the evaluation of MGA to the patients who clinically and electrophysiologically met the criteria for CTS and healthy individuals.

The study was carried out at a constant room temperature of 30 °C. The ground electrode was placed proximally between the recording electrodes and the stimulation site. Precise measurements using a 1 mm precision measuring tape determined conduction distances.[19] Motor NCSs involved parameters such as a swept speed of 5 ms/division and sensitivity of 5 mV/division. The filter bandwidth ranged from 10 Hz to 10 kHz. A bipolar stimulator capable of generating 100 mA current was utilized, with a pulse duration of 0.1-0.5 ms. Rigorous care was taken during electrophysiological tests to ensure that changes in compound muscle action potential (CMAP) amplitudes were not due to technical factors such as submaximal stimulation or co-stimulation of adjacent nerves. Supramaximal stimulation was achieved by gradually increasing the stimulus current until no further increase in CMAP amplitude was observed.[20]

The study protocol used in the detection of MGA was as follows[21]

1. The procedure involved stimulating the median nerve at both the wrist and the antecubital fossa (AF), and recording from three distinct muscles - abductor pollicis brevis (APB), abductor digiti minimi (ADM), and FDI muscles - in three different stimulation scenarios. Suspicions of MGA arose if the median CMAP amplitude from the AF stimulation exceeded that of the wrist stimulation.[20,22,23] MGA was considered present if the proximal median nerve CMAP amplitude recorded from the AF was at least 2 mV higher than the distal amplitude.[21]

2. Ulnar nerve stimulation occurred at a point at least 4 cm distal to the medial epicondyle of the humerus, both at the wrist and below the elbow. Recordings were taken from APB, ADM, and FDI muscles in three distinct stimulation scenarios. In the presence of MGA, the ulnar CMAP amplitude from the below-elbow stimulation was notably less than the motor amplitude of the ulnar nerve recorded at the wrist stimulation.[20,22] Suspicion of MGA arose if the ulnar CMAP amplitude recorded from below-elbow stimulation was less than 10% of that recorded from wrist stimulation.[20]

3. During recordings from the proximal stimulation zone with the APB, the presence of an initial positive deflection or a dual component in the median nerve CMAP was noted, along with the velocity exceeding 75 m/s from the proximal stimulation site.

The statistical analysis of the data obtained in the study was performed using the SPSS software (version 25, IBM Corporation, Armonk, NY, USA). The normality assumption of the continuous variables was tested using the Kolmogorov-Smirnov and Shapiro-Wilk tests. Descriptive statistics were presented as mean ± standard deviation (SD) and frequency [n (%)]. The chi-square test, Fisher-Freeman-Halton's exact test

test, and independent samples t-test were utilized for the univariate analyses of the variables in the study. P-values less than 0.05 were regarded as statistically significant in all statistical analyses.

The research was conducted ethically in accordance with the World Medical Association Declaration of Helsinki. The author confirms that the subjects provided their written informed consent. The study was granted approval by the non-interventional clinical research ethics committee of Siirt University (decision number: 03.26.2021/5721).

Results

A total of 253 individuals were enrolled in the study. The demographic characteristics of the individuals and the group comparisons are given in table 1. Of the individuals, 147 were in the CTS (-) control group and 106 were in the CTS (+) case group. In the CTS (-) group, there were 120 women (83.0%) and 23 men (15.6%), while two individuals (1.4%) did not disclose their gender. In the CTS (+) group, there were 74 women (69.8%), 32 men (30.2%), and 0 patients (0%) with missing gender data. The difference between groups was statistically sig-nificant in terms of gender (P = 0.007). The mean age of the patients in the case group (49.88 ± 12.97 years) was higher than that of the healthy individuals in the control group. The dif-ference between the control and case groups in terms of mean age was statistically significant (P < 0.001).

The relationship between the occurrence of MGA in both the case and control groups is displayed in table 2. As per the findings, within the control group of healthy individuals, type I MGA was absent in 142 individuals (96.6%) and present in five individuals (3.4%). Meanwhile, among patients in the CTS (+) case group, type I MGA was absent in 91 individuals (85.8%) and present in 15 individuals (14.2%). Notably, the presence of type I MGA exhibited a statistically significant correlation with the CTS (+) case group (P = 0.002). In the control group, type II MGA was absent in 114 individuals (77.6%) and present in 33 individuals (22.45%). Conversely, within the case group, absence of type II MGA was noted in 67 patients (63.2%), while it was present in 39 patients (36.8%). The connection between the presence of type II MGA and the case group was found to be statistically significant (P = 0.013). In the control group, type III MGA was absent in 134 individuals (91.2%) and present in 13 individuals (8.8%). As for the case group, type III MGA was absent in 101 patients (95.3%) and present in five patients (4.7%). The relationship between the presence of type III MGA and the case group was not statistically significant (P = 0.208). Type IV MGA was absent in 138 individuals (93.9%) and present in nine individuals (6.1%) in the control group. Type IV MGA was absent in 92 (86.8%) patients and present in 14 patients (13.2%) in the case group. The relationship between the presence of type IV MGA and the case group was not statistically significant (P = 0.087).

The results regarding the potential significant relationship between the control and case groups and the side of MGA are given in table 3. According to these results, in the CTS (-) control group, type I MGA was identified on the right side in one individual (20%), on the left side in three individuals (60%), and bilaterally in one individual (20%). In the CTS (+) case group, type I MGA was detected on the right side in six patients (40.00%), on the left side in eight patients (53.33%), and bilaterally in one patient (6.67%). However, the relationship between the lateralization of type I MGA and the groups was not statistically significant (P = 0.770).

In the control group, type II MGA was detected on the right side in 13 individuals (39.93%), on the left side in 13 individuals (39.93%), and bilaterally in seven individuals (21.21%). In the case group, type II MGA was present on the right side in 16 patients (41.03%), on the left side in 15 patients (38.46%), and on both sides in eight patients (20.51%).

Table 1. Demographic characteristics of the control and case groups

	Control (CTS -) (n = 147)	Case (CTS +) (n = 106)	P
Gender [n (%)]			
Women	122 (83.0)	74 (69.8)	0.012*
Men	23 (15.6)	32 (30.2)	
Missing	2 (1.4)	0 (0)	
Age (year) (mean ± SD)	42.94 ± 12.14	49.88 ± 12.97	< 0.001**

*Chi-square test; **Independent samples t-test
CTS: Carpal tunnel syndrome; SD: Standard deviation

Table 2. Presence of Martin-Gruber anastomosis (MGA) and its subtypes in the case and control groups

	Control (CTS -) (n = 147) [n (%)]	Case (CTS +) (n = 106) [n (%)]	P*
Type I MGA			
No	142 (96.6)	91 (85.8)	0.002
Yes	5 (3.4)	15 (14.2)	
Type II MGA			
No	114 (77.6)	67 (63.2)	0.013
Yes	33 (22.4)	39 (36.8)	
Type III MGA			
No	134 (91.2)	101 (95.3)	0.208
Yes	13 (8.8)	5 (4.7)	
Type IV MGA			
No	138 (93.9)	92 (86.8)	0.087
Yes	9 (6.1)	14 (13.2)	

*Chi-square test
MGA: Martin-Gruber anastomosis; CTS: Carpal tunnel syndrome

The relationship between the lateralization of type II MGA and the groups was not statistically significant (P = 0.990).

In the control group, type III MGA was observed on the left side in seven individuals (53.85%), on the right side in four individuals (30.77%), and on both sides in two individuals (15.38%). In the case group, type III MGA was detected on the right side in one patient (20%), on the left side in three patients (60%), and on both sides in one patient (20%). There was no statistically significant relationship between the side of type III MGA and the groups (P = 0.402).

Type IV MGA in the control group of healthy individuals was on the right side in three (33.33%), on the left side in two (22.22%), and bilateral in four (44.44%). As for the case group, type IV MGA was on the right side in six patients (42.86%), on the left side in seven patients (50.00%), and on both sides in one patient (7.14%). There was no statistically significant relationship between the side of type IV MGA and the groups (P = 0.166).

Discussion

Among the prevalent anomalies affecting nerve innervation in the upper extremity, MGA stands out as a prominent example. It is important to remember that every electromyographer can recognize this anomaly during routine NCSs. If it is not recognized, the condition can easily be confused with a technical abnormality or, in some cases, genuine pathological conditions. The fibers originating from the median nerve, once transversed, proceed to join the distal ulnar nerve, consequently supplying various combinations of ulnar muscles.

Table 3. Side distribution of the Martin-Gruber anastomosis (MGA) subtypes among control and case groups

	Control (CTS -) (n = 147) [n (%)]	Case (CTS +) (n = 106) [n (%)]	P
Type I MGA side			
Right	1 (0.20)	6 (40.00)	0.770*
Left	3 (0.60)	8 (53.33)	
Both	1 (0.20)	1 (6.67)	
Type II MGA side			
Right	13 (39.39)	16 (41.03)	0.990**
Left	13 (39.39)	15 (38.46)	
Both	7 (21.21)	8 (20.51)	
Type III MGA side			
Right	7 (53.85)	1 (20.00)	0.402*
Left	4 (30.77)	3 (60.00)	
Both	2 (15.38)	1 (20.00)	
Type IV MGA side			
Right	3 (33.33)	6 (42.86)	0.166*
Left	2 (22.22)	7 (50.00)	
Both	4 (44.44)	1 (7.14)	

*Fisher-Freeman-Halton's exact test; **Chi-square test
MGA: Martin-Gruber anastomosis; CTS: Carpal tunnel syndrome

Various prevalences of MGA were reported by Kimura et al. (17.0%),[24] Amoiridis (32.0%),[25] Sarikcioglu et al. (3.3%),[22] Prates et al. (7.8%),[26] Lee et al. (39.0%),[10] de Almeida et al. (12.5%),[27] Felippe et al. (10.0%),[28] Kazakos et al. (10.0%),[29] Erdem et al. (27.0%),[3] Rodriguez-Niedenfuhr et al. (13.6%),[30] Shu et al. (23.6%),[31] Nakashima (21.3%),[32] Uchida and Sugioka (17%),[33] Thomson (15.5%),[34] and Gruber (15.5%).[35]

The prevalence of MGA has been reported to range from 8% to 26% in patients with CTA, whereas its occurrence rates in normal or unselected patients range from 15% to 39%.[36] In our study, the female gender was predominant in both groups, which can be explained by the higher percentage of women compared to men in the CTS (-) (83.0%) and CTS (+) (69.8%) groups. The mean age of the individuals was higher in the CTS (+) group than in the CTS (-) group.

According to the classification proposed by Oh, three electrophysiological MGA subtypes can be recognized depending on the distribution of MGA axonal fibers in the hand; each subtype has specific electrophysiological features in motor conduction studies of the median and ulnar nerves.[9] The crossed fibers in the type I anastomosis terminate in the hypothenar muscle (ADM) group. An anastomosis consists of almost exclusively motor axons. However, the ulnar nerve CMAP amplitude during ulnar motor NCSs demonstrates lower values in below-elbow stimulation compared to wrist stimulation, potentially leading to misdiagnoses if the MGA remains unrecognized with an impression of a conduction block. In this situation, to confirm the presence of MGA, the median nerve recorded from the hypothenar muscle is stimulated at the wrist level and AF. While the median nerve CMAP amplitude is received when stimulated from the AF, very low or no response is recorded when stimulated from the wrist. However, it is important not to overstimulate the median nerve in the AF; otherwise, it results in the co-stimulation of the ulnar nerve at the elbow and gives a false impression of an MGA when actually not present[20] (Figure 1).

In our study, the prevalence of type I MGA was 3.4% in the group without CTS and 14.2% in the group with CTS, with a statistically significant difference. Prior research has indicated a tendency for unilateral type I MGA to occur more frequently in the right forearm,[22,37] although another study suggested a higher occurrence in the left forearm.[28] Our study did not identify a statistically significant difference in the distribution of type I MGA among the left, right, or bilateral forearms in either group.

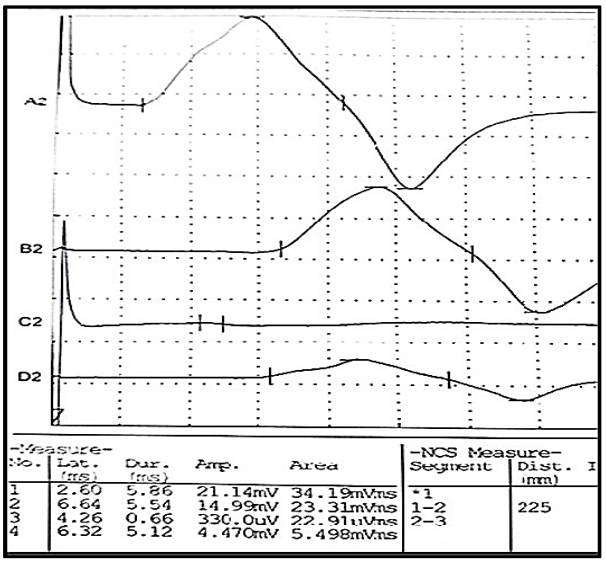

Figure 1. Type I Martin-Gruber anastomosis (MGA). The ulnar amplitude at the below-elbow stimulating site (B2) is lower than at the wrist site (A2). To confirm the presence of MGA, the median nerve is stimulated at the wrist (C2) and the AF (D2), while recording responses from the ulnar muscle. The goal is to detect a compound muscle action potential (CMAP) that is higher in amplitude when stimulated at the AF (D2) than when stimulated at the wrist (C2) (data shown are from a 35-year-old female subject in our study).

Type II anastomosis involves nerve fibers that terminate in the muscle group of the FDI. The pattern observed in NCSs of the FDI muscle, which supports the presence of MGA, mirrors the conduction pattern seen in the ADM muscle. During routine ulnar motor NCSs, there is a notable decrease in the amplitude of the CMAP between the wrist and the below-elbow stimulation sites. To demonstrate the presence of MGA recorded from the FDI muscle, stimulation of the median nerve is conducted both at the wrist and the AF. This is done to identify a higher amplitude of the median nerve CMAP during AF stimulation compared to wrist stimulation. The contribution of muscles innervated by the median nerve, along with the involvement of transverse fibers in AF stimulation, results in a CMAP with increased amplitude compared to wrist stimulation (Figure 2).

Usually, the disparity in CMAP amplitude between stimulating the median nerve at the wrist and the AF is approximately equivalent to the amplitude reduction observed between stimulating

the ulnar nerve at the wrist and the below-elbow area. Conducting an ulnar motor NCS specifically on the FDI muscle is not a standard procedure; however, it holds significance in the assessment of ulnar nerve lesions. Unfortunately, the electrophysiological examination of the deep palmar branch of the ulnar nerve adds complexity to the interpretation of MGA findings obtained from the FDI muscle, introducing an additional layer of challenge to the process.[20]

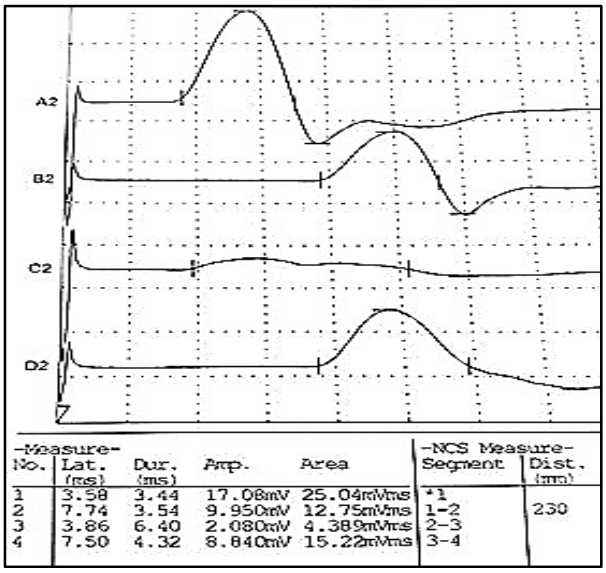

Figure 2. Type II Martin-Gruber anastomosis (MGA) [crossover of the median-to-ulnar fibers supplying the first dorsal interosseous (FDI) is the most common type of MGA]. This type of MGA manifests as a drop in the amplitude between the wrist site (A2) and below-elbow site (B2) stimulation when ulnar motor studies take recordings from the FDI. To confirm the presence of MGA, the median nerve is stimulated at the wrist (C2) and AF (D2), while recording responses from the FDI. The goal is to identify a higher median nerve compound muscle action potential (CMAP) amplitude when stimulated at the AF (D2) compared to the wrist stimulation site (C2) (data shown are from a 42-year-old female subject in our study).

In our study, the prevalence of type II MGA was 22.4% in the CTS (-) group and 36.8% in the CTS (+) group, with a statistically significant difference. Similar to type I MGA, there was no statistically significant difference in the distribution of type II MGA among the left, right, or bilateral forearms in either group.

Wilbourn and Lambert noted a higher prevalence of crossing axons that innervated the FDI muscle (95%) compared to the hypothenar (41%) and thenar (14%) muscles.[38] Uchida and Sugioka, while recording from the ADM and FDI muscles, found that the FDI muscle was more frequently innervated by the anastomotic branch than the ADM.[33] Within the realm of electrophysiological examinations, the presence of MGA is most frequently observed in the FDI muscle, followed by the ADM (hypothenar) and APB (thenar) muscles.[39,40] Additionally, a combination of these muscles showing the presence of MGA has been documented.[3]

In type III anastomosis, the transverse fibers conclude their course in the thenar muscle group. During routine median motor NCSs involving recording from the thenar muscles, a distinct pattern emerges: the CMAP amplitude acquired when stimulating the median nerve at the AF surpasses that obtained from wrist stimulation. If an MGA is present, the CMAP amplitude of the ulnar nerve during below-elbow stimulation will be noticeably lower compared to wrist stimulation (Figure 3).

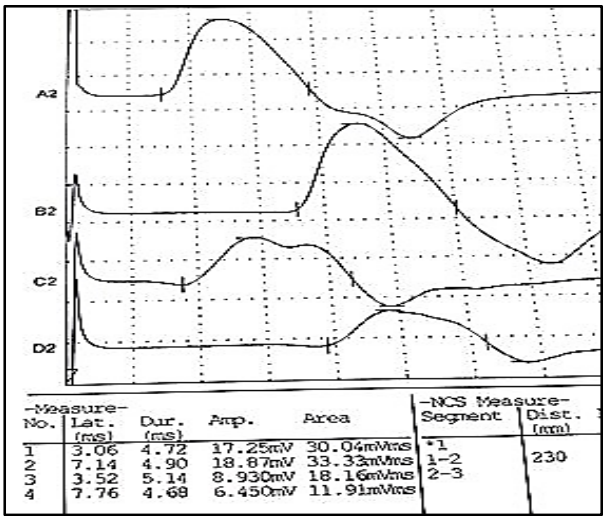

Figure 3. Type III Martin-Gruber anastomosis (MGA) (crossover of the median-to-ulnar fibers supplying the thenar muscles may occur in MGA). When recording from the abductor pollicis brevis (APB) muscle and stimulating the median nerve at the wrist (A2), the amplitude of the median compound muscle action potential (CMAP) during stimulation at the antecubital fossa (AF) (B2) surpasses that obtained from wrist stimulation. To confirm the presence of MGA, the ulnar nerve is stimulated at the wrist (C2) and below-elbow site (D2) while recording from the thenar muscles. A drop in the CMAP amplitude between the wrist and below-elbow site is sought (data shown are from a 39-year-old male subject in our study). This particular type of MGA is important in the electrophysiological evaluation of carpal tunnel syndrome (CTS). The coexistence of CTS and type III MGA exhibits distinct electrophysiological features.

Indications for identifying type III MGA along with CTS are as follows:

1) Observation of a positive deflection in the median CMAP recorded from the thenar muscles during AF stimulation (notably absent during median nerve stimulation at the wrist)

2) Notably accelerated conduction velocity of the median nerve in the forearm. Any velocity exceeding this, particularly accompanied by a positive deflection during proximal stimulation, indicates the potential presence of MGA associated with CTS.[20]

These observations can be elucidated in the following manner:

i) Upon stimulation of median motor fibers at the AF, most fibers traverse the forearm and proceed through the carpal tunnel. However, the median fibers constituting the MGA bypass the carpal tunnel, extending along the ulnar nerve to innervate the thenar muscles before the impulses traverse the median nerve, which remains entrapped within the carpal tunnel. These bypassed fibers initiate the depolarization of ulnar innervated muscles, leading to the initial deflection

ii) Building on the aforementioned explanation, when the prolonged median latency is subtracted from the typically normal median antecubital latency, a notably brief time interval emerges, resulting in an astonishingly rapid median conduction velocity.[20] Consequently, understanding the clinical significance of MGA is pivotal for comprehending CTS. In the current study, during the forearm NCS conducted on the ipsilateral hand thenar muscle, a case involving CTS/type III MGA was identified, exhibiting the previously described features in a female patient (Figure 4).

In our study, the prevalence of type III MGA was 8.8% in the CTS (-) group and 4.7% in the CTS (+) group. However, the observed difference was not found to be statistically significant. In addition, type IV MGA, encompassing combinations of three other types within a single forearm, exhibited a prevalence of 6.1% in the CTS (-) group and 13.2% in the CTS (+) group. Nevertheless, similar to type III MGA, this difference was not statistically significant. On the other hand, there were no statistically significant variations detected among left, right, or bilateral forearm involvements for both type III MGA and type IV MGA within both groups.

Conclusion

Anastomoses between the median and ulnar nerves, known as MGA, are typically asymptomatic but can complicate the interpretation of NCSs, potentially leading to misdiagnosis and inappropriate treatment. It is crucial to rule out MGA before confirming the presence of ulnar neuropathy at the elbow. Additionally, the presence of MGA can create challenges in diagnosing and managing conditions in the upper extremities, such as CTS. Hence, across various electrodiagnostic assessments of the upper extremity, it is essential to recognize that MGA holds significant clinical significance for accurately diagnosing and devising suitable treatments for peripheral lesions and compression syndromes involving the median and ulnar nerves.

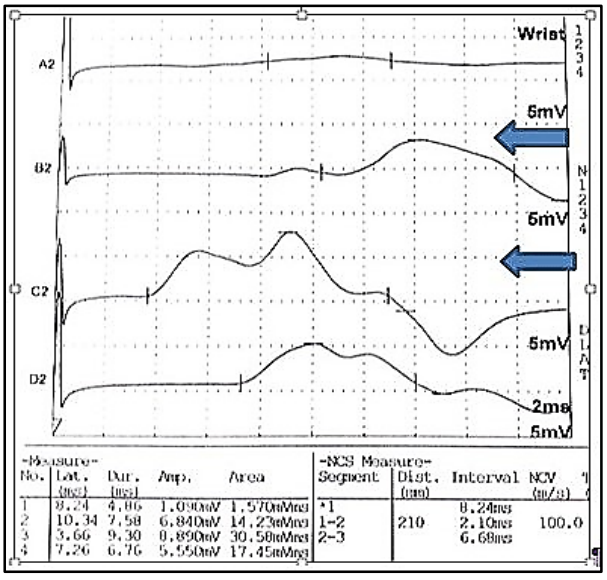

Figure 4. Type III Martin-Gruber anastomosis (MGA) + severe carpal tunnel syndrome (CTS) [there is a prolonged distal latency at the wrist stimulation site (A2)]. At the antecubital fossa (AF) site (B2), a positive dip and a remarkably fast conduction velocity (100.0 m/s) are evident (top arrow). Notably, a higher amplitude is observed at the proximal stimulation site (B2). The anastomosis is demonstrated by stimulating the ulnar nerve at the wrist (C2) and below-elbow site (D2), while recordings are made from the thenar muscles. A larger potential is noted when stimulating at the wrist compared to the below-elbow site (bottom arrow) (data shown are from a 38-year-old female subject in our study).

Conflict of Interests

The authors declare no conflict of interest in this study.

Acknowledgments

None.

References

1. Kaur N, Singla RK, Kullar JS. Martin-Gruber anastomosis- a cadaveric study in North Indian population. J Clin Diagn Res 2016; 10(2): AC09-AC11.
2. Simon R, Aminoff M, Greenberg D. Clinical Neurology. 4th ed. Stanford, CA: Appleton and Lange; 1999. p. 170-3.
3. Erdem HR, Ergun S, Erturk C, Ozel S. Electrophysiological evaluation of the incidence of martin-gruber anastomosis in healthy subjects. Yonsei Med J 2002; 43(3): 291-5.
4. Santoro L, Rosato R, Caruso G. Median-ulnar nerve communications: electrophysiological demonstration of motor and sensory fibre cross-over. J Neurol 1983; 229(4): 227-35.
5. Iyer V, Fenichel GM. Normal median nerve proximal latency in carpal tunnel syndrome: A clue to coexisting Martin-Gruber anastomosis. J Neurol Neurosurg Psychiatry 1976; 39(5): 449-52.
6. Simonetti S. Electrophysiological study of forearm sensory fiber crossover in Martin-Gruber anastomosis. Muscle Nerve 2001; 24(3): 380-6.
7. Oh SJ. Electrophysiology. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2003. p. 345-52.
8. Simonetti S, Krarup C. Unusual ulnar sensory innervation and Martin-Gruber anastomosis in a patient with a carpal tunnel syndrome. J Neurol 2000; 247(2): 141-2.
9. Oh SJ. Clinical electromyography: Nerve conduction studies. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2003. p. 848.
10. Lee KS, Oh CS, Chung IH, Sunwoo IN. An anatomic study of the Martin-Gruber anastomosis: Electrodiagnostic implications. Muscle Nerve 2005; 31(1): 95-7.
11. Cho NS, Kim DH, Kim MY, Park BK. Electrophysiological and ultrasonographic findings in ulnar neuropathy with Martin-Gruber anastomosis. Muscle Nerve 2013; 47(4): 604-7.
12. Burakgazi AZ, Russo M, Bayat E, Richardson PK. Underrecognized anomaly: proximal martin-gruber anastomosis at the elbow. J Clin Neurophysiol 2014; 31(1): 31-4.
13. Gutmann L. Median--ulnar nerve communications and carpal tunnel syndrome. J Neurol Neurosurg Psychiatry 1977; 40(10): 982-6.
14. Rubin DI, Dimberg EL. Martin-Gruber anastomosis and carpal tunnel syndrome: [corrected] morphologic clues to identification. Muscle Nerve 2010; 42(3): 457-8.
15. Kayamori R. Electrodiagnosis in Martin-Gruber anastomosis. Nihon Seikeigeka Gakkai Zasshi 1987; 61(12): 1367-72. [In Japanese].
16. American Association of Electrodiagnostic Medicine, American Academy of Neurology, and American Academy of Physical Medicine and Rehabilitation. Practice parameter for electrodiagnostic studies in carpal tunnel syndrome: Summary statement. Muscle Nerve 2002; 25(6): 918-22.
17. Jablecki CK, Andary MT, Floeter MK, Miller RG, Quartly CA, Vennix MJ, et al. Practice parameter: Electrodiagnostic studies in carpal tunnel syndrome. Report of the American Association of Electrodiagnostic Medicine, American Academy of Neurology, and the American Academy of Physical Medicine and Rehabilitation. Neurology 2002; 58(11): 1589-92.
18. American Association of Electrodiagnostic Medicine. Guidelines in electrodiagnostic medicine. Muscle Nerve 1992; 15(2): 229-53.
19. Saba EK. Median versus ulnar medial thenar motor recording in diagnosis of carpal tunnel syndrome. Egypt. Rheumatol 2015; 37(3): 139-46.
20. Preston DC, Shapiro BE. Electromyography and Neuromuscular Disorders: Clinical-electrophysiologic correlations. 3rd ed. London, UK: W.B. Saunders; 2013.
21. Di Stefano, V, Gagliardo A, Barbone F, Vitale M, Ferri L, Lupica A, et al. Median-to-Ulnar nerve communication in carpal tunnel syndrome: An electrophysiological study. Neurol Int 2021; 13(3): 304-14.
22. Sarikcioglu L, Sindel M, Ozkaynak S, Aydin H. Median and ulnar nerve communication in the forearm: An anatomical and electrophysiological study. Med Sci Monit 2003; 9(9): BR351-56.
23. Hasegawa O, Matsumoto S, Iino M, Kirigaya N, Mimura E, Wada N, et al. Prevalence of Martin-Gruber anastomosis on motor nerve conduction studies. No To Shinkei 2001; 53(2): 161-4. [In Japanese].
24. Kimura J, Murphy MJ, Varda DJ. Electrophysiological study of anomalous innervation of intrinsic hand muscles. Arch Neurol 1976; 33(12): 842-4.
25. Amoiridis G. Frequency of ulnar-to-median nerve anastomosis. Electromyogr Clin Neurophysiol 1992; 32(4-5): 255-6.
26. Prates LC, de Carvalho VC, Prates JC, Langone F, Esquisatto MAM. The Martin-Gruber anastomosis in Brazilians: An anatomical study. Braz J Morphol Sci 2017; 20(3): 177-80.
27. de Almeida JA, Vitti M, Garbino JA. Anatomical study of the Martin-Gruber anastomosis. Hansen Int 1999; 24(1): 15-20. [In Portuguese].
28. Felippe MM, Telles FL, Soares ACL, Felippe FM. Anastomosis between median nerve and ulnar nerve in the forearm. J. Morphol Sci 2012; 29(1): 23-6.
29. Kazakos KJ, Smyrnis A, Xarchas KC, Dimitrakopoulou A, Verettas DA. Anastomosis between the median and ulnar nerve in the forearm. An anatomic study and literature review. Acta Orthop Belg 2005; 71(1): 29-35.
30. Rodriguez-Niedenfuhr M, Vazquez T, Parkin I, Logan B, Sanudo JR. Martin-Gruber anastomosis revisited. Clin Anat 2002; 15(2): 129-34.
31. Shu HS, Chantelot C, Oberlin C, Alnot JY, Shao H. Martin-Gruber communicating branch: anatomical and histological study. Surg Radiol Anat 1999; 21(2): 115-8.
32. Nakashima T. An anatomic study on the Martin-Gruber anastomosis. Surg Radiol Anat 1993; 15(3): 193-5.
33. Uchida Y, Sugioka Y. Electrodiagnosis of Martin-Gruber connection and its clinical importance in peripheral nerve surgery. J Hand Surg Am 1992; 17(1): 54-9.
34. Thomson A. Third Annual Report of Committee of Collective Investigation of Anatomical Society of Great Britain and Ireland for the Year 1891-92. J Anat Physiol 1893; 27(Pt 2): 183-94.
35. Gruber W. Ueber die verbindung des nervus medianus mit dem nervus ulnaris am unterarme des menshen und der saugethiere. Arch Anat Phisiol 1870; 37: 501-22. [In German].
36. Oh SJ. Clinical electromyography: Nerve conduction studies. 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 1993. p. 314-33.
37. Taams KO. Martin-Gruber connections in South Africa. An anatomical study. J Hand Surg Br 1997; 22(3): 328-30.
38. Wilbourn A, Lambert E. The forearm median-to-ulnar nerve communication: electrodiagnostic aspects. Neurology 1976; 26: 368.
39. Khosrawi S, Kianimehr L, Andalib S. The prevalence of Martin-Gruber anastomosis in Iranian subjects by electrodiagnostic criteria. Iran J Neurol 2015; 14(4): 231-2.
40. Roy J, Henry BM, PEkala PA, Vikse J, Saganiak K, Walocha JA, et al. Median and ulnar nerve anastomoses in the upper limb: A meta-analysis. Muscle Nerve 2016; 54(1): 36-47.